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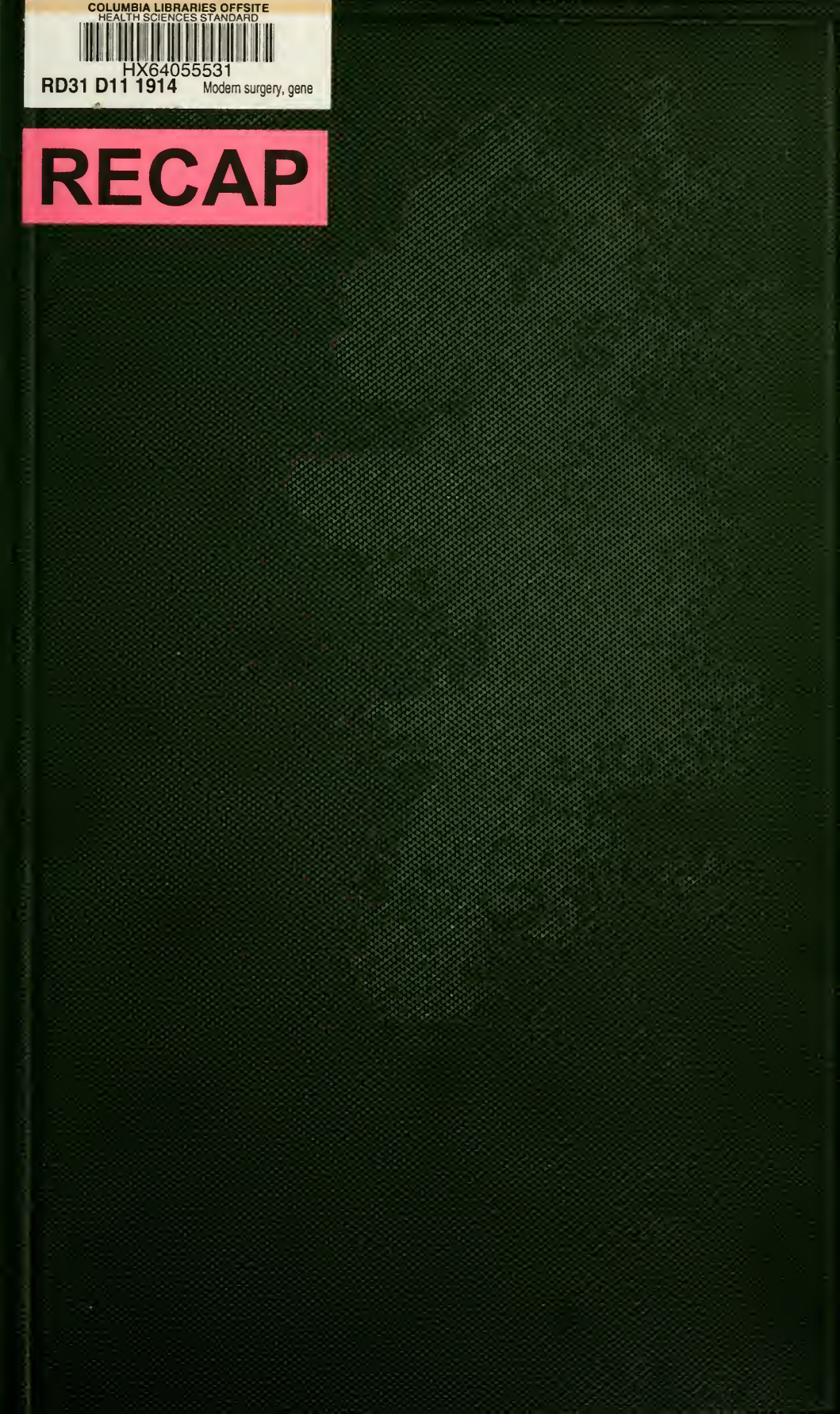

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# MODERN SURGERY

*GENERAL AND OPERATIVE*

BY

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*Seventh Edition, Revised, Enlarged, and Reset*
*With 1085 Illustrations, Some of Them in Colors*

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

1915

Copyright, 1894, by W. B. Saunders. Reprinted March, 1895, and August, 1896. Revised, entirely reset, reprinted, and recopyrighted June, 1898. Reprinted October, 1898, and June, 1899. Revised, reprinted, and recopyrighted August, 1900. Reprinted August, 1901; August, 1902, and November, 1902. Revised, entirely reset, reprinted, and recopyrighted August, 1903. Reprinted July, 1904; October, 1905; September, 1906, and October 1906. Revised, reprinted, and recopyrighted January, 1907. Reprinted April, 1907; March, 1908, and July, 1909. Revised, reprinted, and recopyrighted January, 1910. Reprinted September, 1910; May, 1911; August, 1912, and August 1913. Revised, entirely reset, reprinted, and recopyrighted April, 1914.

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Reprinted January, 1915, and September, 1915

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PRINTED IN AMERICA

PRESS OF
W. B. SAUNDERS COMPANY
PHILADELPHIA

"Yet each man, following his sympathies,
Unto himself assimilating all,
Using men's thoughts and forms as steps to rise,
Who speaks at last his individual word,
The free result of all things seen and heard,
Is in the noblest sense original.
Each to himself must be his final rule,
Supreme dictator, to reject or use,
Employing what he takes but as his tool.
But he who, self-sufficient, dares refuse
All aid of men, must be a god or fool."

W. W. Story ("A Contemporary Criticism").

THIS BOOK IS DEDICATED TO THE CHIEF SURGEON AND INSPIRATION OF ONE OF THE GREATEST, MOST PROGRESSIVE, AND MOST INFLUENTIAL SURGICAL CLINICS IN THE WORLD. A CLINIC FROM WHICH COME IMPORTANT FACTS, REAL IDEAS, AND BRILLIANT MEN.

TO THE OPERATOR, THE TEACHER, THE INVESTIGATOR, AND THE SURGICAL PHILOSOPHER. TO

DR. WILLIAM STEWART HALSTED,

THE DISTINGUISHED PROFESSOR OF SURGERY IN

JOHNS HOPKINS UNIVERSITY.

# PREFACE TO SEVENTH EDITION

It is the custom to have a preface in a medical book. So this book, too, shall have its preface. The natural tendency is to do the customary thing in the conventional way. Were I to do this I would tell how many volumes of previous editions have been sold, would claim appreciation as the due of the seventh edition, give a list of the attractive novelties it contains, and express the conviction that the latest edition (like the newest baby) is superior to all of its predecessors. I have done this sort of thing in the past, but I shall do it no more, because such a preliminary proclamation has in it an unpleasant suggestion of the shopkeeper advertising his stock or the peddler vending his wares.

A man who has worked long and earnestly in the completion of a book has upon him a sense of disenchantment and has within him a host of misgivings. Hope is seldom a constant companion of the solitary worker. No one can be more conscious than the author that there are defects in this book. I have done my best to correct them and have corrected many of them, but others are uncorrectable without writing an entirely new book.

In the making of this, as of previous editions, I have again and again been in profound perplexity as to whether an alleged discovery is a fragment of eternal truth or a nebulous emanation of chaos. To make many mistakes of judgment in regard to such matters would mean a book rich in misinformation. If all of the alleged improvements of recent years were gathered together, the company would be decidedly mixed, and one would have to be cautious in receiving and in making introductions. In that company we would find the productions of the mistaken enthusiast, of the brilliant confidence man, of the deluded observer, of the conscientious worker, of the dull pretender, of the man with occasional flashes of genius, of the profound scholar, and of the grandee of science.

In a book like this the author is held by a short tether. He must strive to attain a happy medium between undue length and undue brevity. The former tends to become prolixity, the latter a Gradgrind catalogue of undigested and unexplained facts. Almost every very long book, like almost every very long congressional speech, contains many words which tell little. Too brief a book is like Mrs. General's mind, free from opinions. She had "a little circular set of mental grooves or rails on which she started little trains of other people's opinions, which never overtook one another and never got anywhere." Many times I have set forth opinions, but as want of space

has forbidden argument and discussion, the imperative mood has dominated the text, and many of the views must appear dogmatic and oracular.

I have tried to keep out of these pages the ornaments of plagiarism. I have sought to do so by giving the authority for every important statement. When possible I have used the author's words, believing that his description of his own work must be better than any description that could be written for him. In consequence, the book contains numerous quotations. As I must use other men's work, it is only fair that I should acknowledge it. The vital necessity for using the work of others is emphasized in Mr. Story's lines, which are printed on page 7.

The author wishes to thank warmly several gentlemen who have given able, unselfish, and highly valuable aid. To the sections on Orthopedic Surgery, The Surgery of the Bones, of the Joints, of the Muscles, and of the Tendons, Dr. J. Torrance Rugh, Associate in Orthopedic Surgery in the Jefferson Medical College, contributed some of the results of his extensive experience.

The section on Röntgenology was revised by that most skilful operator, Dr. Willis F. Manges, the Röntgenologist of the Jefferson College Hospital. Dr. Thomas C. Stellwagen, Jr., assistant Genito-urinary Surgeon of the Jefferson College Hospital, was of great help to me in the revision of the sections on Genito-urinary Diseases.

Dr. Hubley R. Owen, Surgeon to the Philadelphia Hospital, has efficiently and in many ways lessened the burdens of the author.

To Dr. Chevalier Jackson, of Pittsburgh, the distinguished laryngologist, I extend sincere acknowledgments. He did me the honor to contribute to this book. He wrote a section upon Tracheobronchoscopy and Esophagoscopy, describing the very valuable methods he devised.

A contemplation of the achievements of modern surgery must fill the surgical student with hope and confidence, must inspire him with the conviction that we are on the threshold of great events, and that the first few hesitating words of truth have as yet but scarce been lisped by the baby lips of Science.

John Chalmers DaCosta.

2045 Walnut Street, Philadelphia.

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# MODERN SURGERY

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## I. BACTERIOLOGY

Bacteriology is the science of micro-organisms. Though a science in the youth of its years, bacteriology has not only profoundly altered, but it has also revolutionized, pathology, and our views of surgery would be incomplete, misleading, and erroneous without its aid.

**Micro-organisms,** or **microbes,** are minute non-nucleated vegetable cells closely connected with fungi and algæ, many of them being visible only by means of a highly powerful microscope and after they have been brightly stained. The contents of these cells are protoplasm and nuclear chromatin enclosed by an albuminous structure which in some cases contains cellulose. There is considerable evidence that certain diseases are caused by micro-organisms so minute as to escape detection even by the most powerful microscope. The French Yellow Fever Commission asserted that the yellow fever micro-organism (which is perhaps protozoal) passes through a porcelain filter ("Annals of the Pasteur Institute," Nov., 1903). The micro-organism of rabies probably does the same thing.

Simon Flexner believes that many diseases are due to submicroscopical parasites (Ether Day Address, 1911).

Even in the most remote times some have believed that "the mysterious cause of contagious and epidemic diseases must be sought in living entities" (Monti on "Modern Pathology"), but all such beliefs were ingenious guesses or unproved theories, unsustained by a scrap of experimental demonstration. Bacteria were discovered in 1683 by the Dutch optician Leeuwenhoek, of Delft. In his researches he used the simple microscope, that is, single lenses of short focal length. By means of this primitive instrument he saw spermatozoids, capillaries, blood-corpuscles, the structure of the crystalline lens, yeast-cells, and certain large bacteria. The Dutch observer regarded bacteria as animalculæ.

In 1762 Plenciz, of Vienna, impressed by the publications of Leeuwenhoek, asserted that each disease is caused by a special organism, that decomposition is caused by micro-organisms, and that bacteria can grow in living tissue.

In 1832 Bassi claimed that a certain disease of silkworms was due to a fungus. In 1839 Schönlein discovered the vegetable parasite causative of favus.

In 1840 Henle came to the conclusion that fungi cause all infectious diseases. In 1843 Oliver Wendell Holmes published his famous essay on the Contagiousness of Puerperal Fever, and in 1847 Semmelweiss, of Vienna, strongly maintained the same thesis.

In 1847 Professor John K. Mitchell put forth the theory that malarial and epidemic fevers have a "cryptogamic origin." In 1849 Malmsten pointed to a fungus as the cause of ringworm.

Cohn, who for over twenty years subsequent to 1850 contributed actively to science, described round bacteria (cocci), rod-shaped bacteria (bacilli), and in 1872 admitted disease-producing bacteria to his classification.

In 1863 Davaine found bacilli in the blood of victims of splenic fever and made a strong argument to prove that the bacilli caused the disease. Absolute proof was furnished by Koch in 1876. He inoculated animals with pure cultures of the bacilli and produced the disease ("Bacteria and Their Products," by Sims Woodhead).

The first definite knowledge of bacteria and their products came from a chemist and not a physician, and dates from the study of fermentation by the illustrious Frenchman Pasteur.

Before his day "bacteria were known, theories of infection had been elaborated, and vaccination practised," but he "definitely established the importance of bacteria in putrefaction, fermentation, and disease, and gave to vaccination a scientific basis" ("Research in Medicine," by Prof. Richard M. Pearce, in "Popular Science Monthly," July, 1912).

In 1858 Pasteur asserted that every fermentation has invariably its specific ferment; that this ferment consists of living cells; that these cells produce fermentation by absorbing the oxygen of the substance acted upon; that putrefaction is caused by an organized ferment; that all organized ferments are carried about in the air; and that the entire exclusion of air prevents putrefaction or fermentation.

In 1860 Pasteur published the observation that sterile liquids will not be contaminated by air if the air gains entry only through a long curved tube, the reason being that dust and growths fall from the entering air by gravity ("Comptes rendus," 1860).

In 1863 Pasteur published experiments which proved that beer cannot ferment without yeast and that wine received in sterile vessels and defended from external contamination will not undergo ammoniacal change.

Most of the subsequent life of Pasteur was passed in seeking the causes, the prevention, and the cure of infectious diseases in man and animals.

The views of Pasteur, which were radical departures from accepted belief, inaugurated a bitter controversy, and in that controversy were born the microbic theory of disease, the doctrine of preventive inoculation, antiseptic surgery, and serum-therapy.

The word *microbe*, which signifies a small living being, was introduced in 1878 by the late Professor Sédillot, of Paris. At that time the nature of these bodies was in doubt; some thought them animal, and called them *microzoaria;* others thought them vegetable, and called them *microphyta;* the designation "microbe" does not commit us to either view. We now know them to be vegetable, but the term "microbe" has remained in use.

The micro-organisms connected with disease in man are divided into:

1. Yeasts, Saccharomyces, or Blastomycetes;
2. Molds, or Hyphomycetes;
3. Bacteria, or Schizomycetes.

**Yeasts,** or **budding fungi,** include most of those fungi which can cause alcoholic fermentation in saccharine matter. They consist of small round or oval cells, which are devoid of chlorophyl, which can live without free oxygen, and which multiply by *gemmation* or *budding*. Definite nuclei are not demonstrable in the cells. When a cell multiplies a small bud of protoplasm projects from or near the end, or buds project from or near both ends of the cell. Buds increase progressively in size and a constriction appears between each bud and the parent-cell. Each constriction deepens as the corresponding projection enlarges, until the bud attains a considerable size and is cast off as a daughter-cell. In some cases buds are not cast off, but remain attached, a chain or series of rounded yeast-cells being formed. Yeast-cells contain spores when nourishment is insufficient. Under certain conditions yeast fungi can form interwoven threads called *mycelial threads*.

**Molds,** or **filamentous fungi,** consist of filaments, each filament being composed of a single row of cells arranged end to end, and all filaments springing from a germinal tube which grows from a germinating spore. A thread grows by increase at the apex, and this area eventually gives origin to new spores. The yeast fungi are the common, but not the only cause of fermentation. Mold fungi are connected with processes of decomposition. Putrefaction is due to bacteria and retards the growth of yeasts and molds.

Most yeasts and molds grow best upon dead organic matter, some attack plants, a few the lower animals, and a very few grow upon or in the tissues of the human body. The term *mycosis* means an infection with budding fungi or with filamentous fungi.

The *oïdium albicans* is a fungus which by growing in the mucous membrane produces the disease known as *thrush.* Some observers believe that the thrush fungus is a mold. Others maintain that it is a budding fungus which may develop filaments. Thrush attacks especially the mucous membrane of the tongue, lips, cheeks, gums, and pharynx, but occasionally the growth takes place upon the esophagus, the vocal cords, the stomach, the vagina of a pregnant woman, the respiratory tract, and the areola of the breast of a nursing woman. The fungus has been found in areas of bronchopneumonia. The proliferating fungus presents the appearance of milky white spots which by thickening and coalescence form curd-like masses, the superficial layer of epithelium being raised and cast off. Thrush is particularly common in infants during the second week of life, and in infants suffering from marasmus, but it may occur in older children and even in adults who have been weakened by some exhausting disease like typhoid fever or tuberculosis.

*Blastomycetes dermatitis* is an inflammation of the skin due to yeast fungi and bearing a resemblance to tuberculosis or syphilis. Pharyngomycoses, keratomycoses, otomycoses, pneumomycoses, and mycoses of the liver, kidney, etc., have been reported. Sanfelice and others maintain that pathogenic yeasts are responsible for the growth of *malignant tumors.* It is certain that yeasts may exist in a carcinoma and can be cultivated, but proof is entirely lacking that they are anything but a contamination. Many skin diseases are due to fungi; among them should be mentioned favus, pityriasis versicolor, herpes tonsurans, parasitic sycosis, and eczema marginatum.

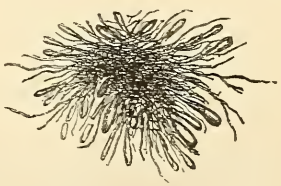


Fig. 1.—Actinomyces (Ziegler).

**Actinomycosis** (**streptotrichosis**) is a disease due to infection with some variety of streptothrix. Usually the streptothrices are regarded as molds, but they possibly constitute a sort of transition stage between filamentous fungi and bacteria.

It was long believed that the ray fungus (Fig. 1) was the only cause of actinomycosis. We now know that other members of the streptothrix group may be responsible (see page 309).

**Madura-foot,** or **mycetoma,** is due to the *Streptothrix maduræ.*

**Schizomycetes,** or **bacteria,** chiefly claim our attention. It is important to remember that the term "bacteria," though applied to the class *schizomycetes,* has also a more restricted application—that is, to a division of the class; it may mean either *schizomycetes* in general, or rod-shaped *schizomycetes,* whose length is not more than twice their breadth. In this work it is employed to designate *schizomycetes* as a class.

Bacteria are minute, unbranched, non-nucleated, vegetable cells, free from chlorophyl, varying in shape and occasionally presenting locomotive flagella. Though devoid of chlorophyl (leaf coloring-matter), many of them

produce pigment. The cell consists of a *cell membrane*, a *layer of protoplasm*, and some *central fluid*. No true nucleus has yet been demonstrated, but granules are found within the cells which some call *metachromatic bodies* (Babés) and others *nuclei* (Ernst). The cell membrane varies greatly in thickness, and when it is very thick the cell is said to have a *capsule*. The round cells have a smooth outer surface, but some of the rod-shaped cells show many *flagella* or at the end a single flagellum (Fig. 2). Flagella enable some bacteria to move (*motile bacteria*), but all organisms which possess them are not motile, and under certain conditions bacteria without flagella may develop them, or organisms which possess flagella may lose the power to develop them.

Some bacteria, known as *non-pathogenic*, cannot grow and produce poison either in the tissues, in wound-fluid, or in the fluid moistening a mucous surface. Others grow upon dead organic matter, but are not able to invade living tissues. They can live and multiply in dead material, as the discharge from a wound or in the fluid covering a mucous surface, and are called *saprophytes*, *saprophytic microbes*, or *putrefactive bacteria*. *Obligate* saprophytes only live in dead matter and never become parasites. *Facultative* saprophytes can become parasites under certain circumstances, but normally grow in dead organic matter. Bacteria, known as the *pathogenic*, under certain conditions invade living tissue and cause various diseases. Harmless bacteria are *non-pathogenic*. *Parasitic* bacteria can grow on or in the tissues of the body.

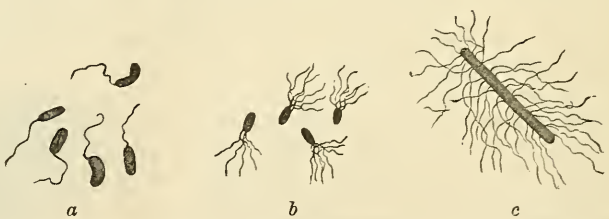



Fig. 2.—Types of flagella: *a*, Vibrio choleræ, one flagellum at the end—monotrichia type; *b*, Bact. syncyaneum tuft of flagella at the end, rarely at the side—Lopotrichia type; *c*, Bact. vulgare, flagella arranged all about—Peritrichia type (Lehmann and Neumann).

*Obligate* parasites are those which have not been cultivated outside of the body (as the spirilla of relapsing fever). *Facultative* parasites usually live outside the body, but may enter into the body and produce disease. The schizomycetes vary much in shape, size, color, arrangement, mode of growth, and action upon the body. One form cannot be transformed into another, but each maintains its specific identity. Every organism comes from a pre-existing organism, this being true of all forms. Pasteur proved that spontaneous generation is impossible. Although numerous attempts have been made to overthrow this view it still stands unshaken. The protoplasm of these cells can be stained with anilin colors, and the cell-wall is more readily detected after treating it with water, which causes it to swell.

Many bacteria are colored; others are colorless. Some move (*motile bacteria*); others do not move (*amotile bacteria*). The bacilli of anthrax and tuberculosis and all cocci are amotile. Most bacteria can change from motile to amotile, or from amotile to motile, when subjected to certain changes of soil and environment. The oscillations of cocci are physical in nature, not vital; they are Brunonian or Brownian movements, movements due to alterations in equilibrium because of currents or changes of level in the fluid in which the micro-organisms are contained. Bacteria seem to possess the power of attracting elements necessary for their nutrition (*positive chemiotaxis* or *chemotaxis*) and of repelling harmful elements (*negative chemiotaxis* or *chemotaxis*).

**Bacterial Products.**—Bacteria when active produce many different products. Among them are gases (H, $H_2S$, $CO_2$, $NH_3$), water, alcohols, fatty acids, carbohydrates, phenol, coloring-matter, toxins, enzymes, etc. Some of these materials are given off from the living cell, some are found only when the cell is dead. Some of them are excretions, some of them secretions. Some are formed within the cell (intracellular), others are excreted by the bacteria into the material in which the cell lies (extracellular).

**Forms of Bacteria.**—The three chief forms of bacteria are—

1. The *Coccus* or *Micrococcus*—the berry-shaped, oval, or round bacterium (Fig. 3);

2. The *Bacillus*—the rod-shaped bacterium (Fig. 4);

3. The *Spirillum* or *Vibrio*—the corkscrew-shaped or spiral bacterium (Fig. 5). A short spiral organism is called a *comma bacillus*. Spirochetes are sharply bent curved rods twisted like the thread of a screw.

Fig. 3.—Micrococci.

Fig. 4.—Bacilli.

Fig. 5.—Spirilla.

De Bary compares these forms, respectively, to the billiard-ball, the lead-pencil, and the corkscrew.

**Cocci and Bacilli.**—As surgeons we have to do chiefly with *cocci* and *bacilli*. Cocci may be designated according to their arrangement with one another; namely, when existing singly they are called *monococci* (Fig. 3); in pairs they are called *diplococci* (Fig. 8, A); arranged end to end in a chain they are called *streptococci* (Fig. 8, C); in group side by side clustered like a bunch of grapes they are called *staphylococci* (Fig. 8, B); in groups of four they are called plate *cocci* or *tetracocci;* in cubic groups they are called *sarcinæ* or *wool-sack* cocci (Fig. 6). Irregular masses, resembling frog-spawn, constitute *zoöglea masses* (Fig. 9). The gelatinous matter in such a mass is formed by a trans-

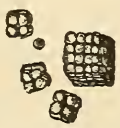


Fig. 6.—Sarcinæ forming bales of packets. Single packets regularly grouped together (Lehmann and Neumann).

Fig. 7.—Ascococcus Billrothii Cohn (after F. Cohn).

formation in the walls of the bacteria. The term *ascococci* is applied to a group of cocci enclosed in a capsule (G. S. Woodhead) (Fig. 7).

The cocci are often named according to their function, as, for example, "*pyogenic*," or pus-forming. Cocci may be named according to the color of the culture. The name may embody the form, arrangement, color of culture, and function; for instance, *Staphylococcus pyogenes aureus* signifies a round micro-organism, which arranges itself with its fellows in the form of a bunch of grapes, which produces pus, and which gives golden-yellow cultures.

The *bacilli* are long, staff-shaped organisms. Long, delicate, jointed bacilli having wavy outlines are known as *leptothrix* forms. Chain-like bacilli are called *streptobacilli*. Bacilli give origin to many surgical diseases.

*Dichotomy or Branching.*—It is very seldom that a side bud appears upon bacteria except in the bacteria of tuberculosis and diphtheria.

*Pseudodichotomy* is by no means unusual. It occurs when one end of a bacillus grows by the end of the adjacent bacillus or when a bacillus in a chain divides in a line parallel to the chain and thus begins another chain (Fig. 10).

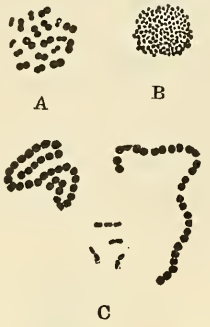



Fig. 8.—Forms of cocci: A, Diplococci; B, staphylococci; C, streptococci.

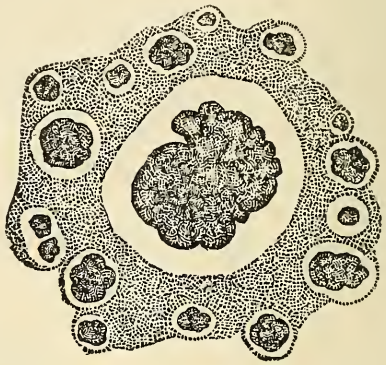


Fig. 9.—Zoöglea (Ball).

**Multiplication of Bacteria.**—Bacteria multiply with great rapidity when placed under suitable conditions. They can multiply by transverse fission or by spore-formation. Some bacteria multiply by both methods. In

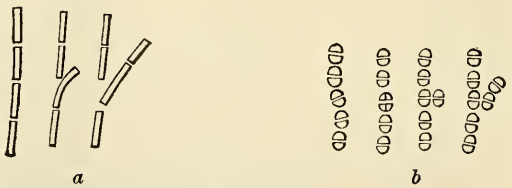



Fig. 10.—Pseudodichotomy: *a*, In bacilli; *b*, in streptococci (Lehmann and Neumann).

*fission*, or *segmentation*, a bacillus undergoes an increase in size and length; a coccus does not increase in size, but slightly elongates. In either case about the middle of the cell a transverse constriction begins, which deepens until the cell has divided into two parts, each of which soon grows as large as its parent (Figs. 11, 12). As a rule, the micro-organisms separate after division of the cell; but they may not do so; and if they do not separate, the special grouping receives a particular name (diplococci, streptococci, etc.). If the division is invariably in the same direction, and if the new cells remain in contact, streptococci or streptobacilli are formed. Tetracocci and sarcinæ are formed when a number of cocci "divide in two or three successively vertical directions" ("Clinical Bacteriology," by Levy and Klemperer), forming four quadrants (*tetracocci*) or eight octants (*sarcinæ*). All cocci and most bacilli multiply by fission.

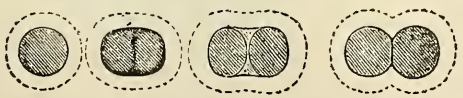


Fig. 11.—Divisions of a micrococcus (after Macé).

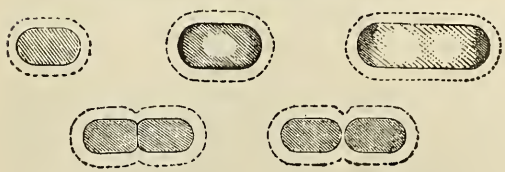


Fig. 12.—Divisions of a bacillus (after Macé).

Hence the common term of *fission fungi*. The time required for the multiplication of a bacterium varies. Some varieties, when placed under favorable conditions, undergo fission in two hours. The cholera bacillus requires only twenty minutes to divide. The tubercle bacillus requires several days. If segmentation of a single cell and the growth to maturity of its products require one hour, a single cell in a single day, if the conditions for increase were ideally favorable, would have 16,000,000 descendants, and in three days the mass of new cells would weigh 7500 tons (Cohn). In order, however, for such enormous multiplication to occur conditions would have to be absolutely favorable for the cells, and conditions are never absolutely favorable. Were it otherwise, all other forms of life would be destroyed. During growth in a culture-medium inhibitory substances are formed, and these substances are detrimental to the bacteria themselves and to all bacteria of the same type. Such substances are known as *autotoxins* (Conradi and Kurpjuweweit, in "Muenchen. med. Woch.," No. 32, September 12, 1905). In a culture of cholera bacilli the number of living microbes begins to lessen after twenty-four hours, and after forty-eight hours the diminution is distinct.

**Spores.**—A *spore* is a germ, and corresponds with the seed of a plant. Some bacilli, a few spirilla, and it may be sarcinæ, multiply by spore-formation. Cocci do not undergo spore-formation after the manner of bacilli, though some observers maintain that cocci occasionally undergo an alteration that makes them very resistant to any destructive influences. When spore-formation is about to occur in a bacillus, a point of cloudiness or an area of bright refraction appears in the protoplasm and the cell generally elongates. When a row of cells sporulate, the segments, each of which contains a lustrous area or a region of cloudiness, look like parts of a necklace of beads (Fig. 13). The spore enlarges, the cell membrane bursts, and the young bacillus emerges through the opening. A cell usually contains but one spore, which may be situated at the end of the cell (*endspore*) or in the middle of the cell (*endospore*). Sometimes a single cell contains several spores. If an endspore exists, the end of the cell containing the spore is swollen or club shaped (*drumstick bacterium*). If an endospore exists, the cell becomes spindle shaped (*clostridium*). When multiplication is by a single endospore, the bacillus does not elongate. When multiplication takes place by a process of combined spore-formation and fission, the mother-cell divides into a number of daughter-cells, which are called *arthrospores*. Organisms which when active multiply by fission take on spore-formation when subjected to certain conditions.

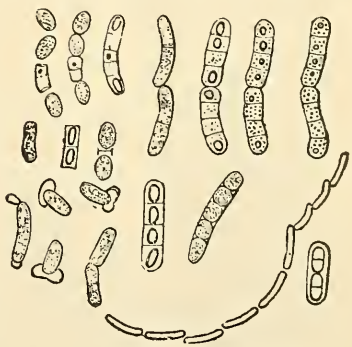


Fig. 13.—Sporulation (after De Bary).

Spore-formation tends to occur when bacilli are about to die for want of nourishment or when there is an excess of oxygen present. The spore has a dense envelope or covering which is very resistant to destructive agents. So resistant is the covering that twice the amount of heat is necessary to kill a spore as to kill an active adult cell. Spores when placed under conditions unfavorable for development may remain inactive for an indefinite period, just as seeds remain inactive when unplanted. Drying, even drying for years, may not destroy them. A dry temperature of nearly 300° F. destroys the spores of anthrax, but only after acting for three hours. Steam or boiling water kills most spores in a few minutes. Some spores are able to withstand the action of live steam for several or perhaps for many hours. Direct sunlight quickly

destroys spores ("Clinical Bacteriology," by Levy and Klemperer). When spores encounter favorable conditions, they develop very rapidly into adult cells, just as seeds develop when planted. It seems probable that spores occasionally remain dormant in the human body for long periods, and finally awaken into activity because of injury or disease of the tissue in which they lie.

**Chemical Composition of Bacteria.**—The protoplasm of bacteria consists of water, salts, albuminous material, extractives soluble in alcohol, and extractives soluble in ether.

**Life-conditions of Bacteria.**—In order to grow and to multiply, bacteria require a suitable soil and the favoring influences of heat and moisture. The soil demanded consists of highly organized compounds rather than crude substances, and slight modifications in it may prove fatal to some forms of bacterial life, but highly advantageous to others. Some organisms require albuminous matter, others need carbohydrates; they all require water, carbon, nitrogen, oxygen, hydrogen, and certain inorganic materials, especially lime and potassium (Woodhead). All organisms require water. If dried, no micro-organisms will multiply, and many forms will die. The fluids and tissues of the individual may or may not afford a favorable soil for the germs of a disease, or, in the same person, may afford it at one time and not at another. Some individuals seem to possess indestructible immunity from, and others are especially prone to, certain bacterial diseases, and these immunities and predispositions may be hereditary. The Japanese show high immunity to scarlet fever and negroes to yellow fever. Drunkards are predisposed to pneumonia. Some families exhibit high susceptibility to scarlet fever. Negroes are very susceptible to tuberculosis and small-pox. Impairment of health, by altering some subtle condition of the soil, may make a person liable who previously was exempt. The insane are predisposed to infections. Injury or disease of a tissue may increase local liability.

Again, some bacteria which under normal conditions are harmless may become virulent under certain conditions. Colon bacilli, which under normal conditions seem to be putrefactive organisms inhabiting the intestine, may attack a point of least resistance in the intestine itself: this point being established by congestion, strangulation, inflammation, or injury, and descendants of the bacteria which attacked the point of least resistance may become so virulent that they can live and develop in tissues distant and apparently normal and cause disease in them.

The presence of oxygen influences microbic growth. Most organisms thrive best when exposed to the oxygen of the air, and they are known as *aërobic*. The term *anaërobic* is employed to designate organisms that can grow and multiply and produce particular products only when air is absent, free oxygen being fatal to them. They may need oxygen; but if they do, they are able to obtain it from the tissues when air is excluded. Tetanus bacilli and the bacilli of malignant edema are anaërobic. An organism which grows as well in the presence as in the absence of oxygen is called a *facultative-anaërobic bacterium*. Most microbic diseases in man are due to facultative-anaërobic bacteria.

**Effect of Motion, Sunlight, the X-rays, Radium, Cold, and Heat.**—The majority of fungi grow best when at rest; violent agitation retards the growth of some. Sunlight antagonizes the growth of certain bacteria, especially tubercle bacilli and the bacilli of typhoid fever. Direct sunlight even destroys spores. Ordinary daylight is very slowly germicidal to bacteria. Direct sunlight is quickly fatal to them. Sunlight is bactericidal even when heat rays are intercepted. The active agent is the light. The blue and violet are the most active, while the rays of the red end of the spectrum are devoid of power to kill bacteria. The electric arc-light acts on bacteria like sunlight. It is claimed by some that the *x*-rays retard bacterial growth. Radium rays are bactericidal.

Sunlight, the arc-light, radium emanations, and *x*-rays stimulate tissue activities, and so aid the tissues to rid themselves of bacteria. Temperature influences bacterial growth. Some organisms will grow only within narrow temperature limits, while others can sustain sweeping alterations, but most grow best between the limits of from 86° to 104° F. Freezing renders bacteria motionless and incapable of multiplication, but it does not kill them; they again become active when the temperature is raised. Prudden showed that typhoid bacilli can live in ice one hundred and three days. The absurdity of employing cold as a germicide is evident when the fact is known that a temperature of 200° F. below zero is not fatal to germ-life, cell-activities by such a temperature only being rendered dormant. Bacteria have been placed in hermetically sealed tubes and the tubes immersed in liquid air for seven days. The germs were thus subjected to a temperature of —190° C., but there was no change produced in their virulence (A. MacFayden and S. Roland, in "Lancet," March 24, 1900). High temperatures are fatal to bacteria; moist heat is more destructive than dry heat, and adult cells are more easily killed than spores. A temperature less than 212° F. will kill many organisms, and boiling will kill every pathogenic organism that does not form spores. Some spores are not destroyed after prolonged boiling, and some will withstand a temperature of 120° C. As a practical fact, however, boiling water kills in a few minutes all cocci, most bacilli, and all pathogenic spores; though the spores of anthrax, tetanus, and malignant edema are killed with more difficulty than are the spores of other bacteria.

**Effect of Bacteria Upon Bacteria.**—Some bacteria are antagonistic to others, some are synergistic to others. When certain bacteria favor the growth of other bacteria the process is called *symbiosis*. When certain bacteria retard the growth of other bacteria the process is known as *antibiosis*. The streptococcus of erysipelas is antagonistic to the bacillus of anthrax and also to syphilis and tuberculosis. The Bacillus prodigiosus makes the streptococcus of erysipelas more active, and the bacillus of anthrax less active. The growth of some microbes in culture-media makes a soil favorable or unfavorable for other microbes, and the same process may occur in the human body. Influenza renders the lungs prone to infection with pneumococci. Aërobic organisms prepare a wound for tetanus bacilli. Saprophytes on mucous surfaces are antagonistic to certain pathogenic bacteria. The organisms productive of lactic acid fermentation are destructive to many injurious intestinal bacteria. We are not yet able to cure a microbic disease by inoculating the sufferer with antagonistic microbes, on the principle of sending a thief to catch a thief, although Hankin ("British Medical Journal," August 14, 1897) suggests purifying water infected with colon bacilli or typhoid bacilli by means of the Micrococcus Ghadiallii, which is fatal to them.

**Latent Bacteria.**—Sometimes pathogenic organisms remain latent in the body for a considerable time. They are not destroyed, but produce no symptoms, or only local symptoms, possibly because the individual is immune for the time being. Pneumococci, staphylococci, and typhoid bacilli may become latent. Tubercle bacilli may remain long latent in a lymph-gland or in any old area of caseation. Syphilis may remain latent for a long time.

Latent bacteria may take on active growth when the tissue containing them is damaged by injury or disease. I have seen active disease arise in an apparently cured and stiff tuberculous joint as a result of forcibly breaking up adhesions. An attack of bronchitis may light up an old and latent area of pulmonary tuberculosis. The administration of ether or chloroform by inhalation may render active a previously inactive tuberculous focus in the lung. A partial or incomplete operation on a quiescent tuberculous lesion is apt to be followed by active spread and may result in wide dissemination of disease.

**Mixed Infection.**—A fact of practical importance to the surgeon is that an area infected by one form of micro-organism may be invaded by another form. This is known as a *mixed* infection, and consists in a *primary* infection with one variety of organism, and a *secondary* infection with another, or in an infection at the same time with different micro-organisms. Mixed infection is especially common on surfaces exposed to air and wound infection is usually mixed. Koch found both bacilli and micrococci in the same lesion of tuberculosis. A soil filled with pneumococci favors the growth of pus cocci and tubercle bacilli. Tuberculous or syphilitic lesions may be attacked by erysipelas. Chancre and chancroid can exist together. A syphilitic ulcer is a good culture-soil for tubercle bacilli (Schnitzler). Suppuration in lesions of tuberculosis is due to secondary infection with pus organisms. Occasionally in empyema and other conditions due to pus organisms the diseased process ceases to be active, the pyogenic bacteria having lost much of their virulence, but a mixed infection with some germ usually harmless may break down surrounding barriers, intensify the virulence of bacteria, and aggravate the disease into an acute outburst. When secondary infection occurs the primary infection may remain as before, may be aggravated, may be mitigated in intensity, may be destroyed, or may be disseminated.

**Intra-uterine or Placental Infection.**—The infection of the embryo from the diseased ovum or the diseased sperm-cell occurs only in syphilis. Such an embryo is diseased at the first moment of life. The direct transmission of bacteria from parent to fetus is a problem still in course of solution. Certain it is that some diseases may follow the transmission of the micro-organism through the septum of separation between the circulations of the mother and child. Placental transmission may occur in syphilis, scarlatina, pneumonia, anthrax, measles, pyogenic conditions, and tuberculosis (Hektoen). A child born of a woman recently the subject of pneumonia may be born with that disease, and a child may be born with pneumonia when the mother has never had it. Mothers free from infectious disease may give birth to infected children. It is stated that Mauriceau (a noted French obstetrician of the 17th century) was born with small-pox, though his mother had never had a sign of the disease. Few cases of congenital tuberculosis have been reported, but Rosenberger claims to have found the bacilli in the umbilical vein from the placenta of a tuberculous mother. A child of a tuberculous parent may not be born tuberculous, but may have weakened tissue-cells that easily fall a prey to the tubercle bacillus when it reaches them by any avenue. Placental transmission of bacteria is favored by disease or injury of the placenta.

**Chemical Antiseptics and Germicides and Aseptic Agents.**—It is necessary to make a distinction between deodorizers, antiseptics, and germicides, although the two latter terms are usually regarded as being interchangable. In the methods of antiseptic surgery we use germicides.

A *deodorizer* is an agent which destroys an offensive odor. It is true that an offensive odor may be due to microbic growth. It is also true that nasty odors may prove injurious to those who inhale them. But, nevertheless, the odor is the result of microbic action, and destroying an odor does not render harmless the bacteria which caused it. Charcoal is a well-known deodorizer.

An *antiseptic* is an agent which retards or prevents putrefaction. It acts by weakening or killing saprophytic organisms, but is not fatal to spores.

A *germicide* or *disinfectant* is an agent which is fatal to adult bacteria and spores. The destruction of the germs of disease on the skin, in clothing, in excreta, in a wound, etc., is known as *disinfection*. Disinfection of the skin, of a wound, of dressings, or of instruments is called also *sterilization*.

Antiseptics and germicides should not be used in surgically clean wounds. Repair will occur more quickly if they are not used. Tillmanns has pointed

out that when antiseptics are used cell-division begins late and progresses slowly. Germicides are not efficient in fatty tissue, as bacteria surrounded with oil cannot be reached by the drug, and the chemical is irritant and apt to induce fat necrosis (Haenel, in "Deutsch. med. Woch.," 1895, No. 8).

**Corrosive Sublimate.**—Many chemical agents will kill bacteria, one of the most popular of them all being corrosive sublimate. Koch showed that corrosive sublimate is an efficient test-tube germicide when present in the proportion of only 1 part to 50,000. It is used in surgery in strengths of 1 part of the salt to 1000, 2000, 3000, or more parts of water. Badly infected wounds are occasionally irrigated with solutions of a strength of 1 : 500. Contact with albumin precipitates from a solution of corrosive sublimate an insoluble albuminate of mercury which forms a white layer upon the surface of the wound, is not a germicide, and prevents deep diffusion of the mercurial fluid. In surgical operations by the antiseptic method the mercurial salt should be combined with tartaric acid in the proportion of 1 to 5, which combination prevents the formation of the insoluble albuminate of mercury.

But though corrosive sublimate under certain conditions is extremely powerful, it is not always absolutely reliable. Many spores are very resistant to its action. Even a 1 per cent. solution of bichlorid of mercury is not certainly destructive to the spores of anthrax. Geppert tells us that anthrax spores may be active after a twenty-five-hour immersion in a 1 : 100 solution of sublimate (Schimmelbusch). In the presence of hydrogen sulphid corrosive sublimate is useless, inert and insoluble sulphid of mercury being precipitated; hence corrosive sublimate is without value as a rectal antiseptic; in fact, Gerloczy has proved that a concentrated aqueous solution of sublimate will not disinfect an equal quantity of feces. Corrosive sublimate contained in dressings after a time undergoes decomposition and ceases to be a germicide. It is not germicidal in fatty tissues because it is unable to attack bacteria which are coated with oil. Corrosive sublimate is very irritating to the tissues and causes copious exudation. Hence, if an extensive wound has been irrigated with this agent, drainage must be employed to obtain exit for the wound fluid. In some wounds which have been irritated by corrosive sublimate the tissues seem to loose to a great extent their power of resistance to bacteria and infection may be actually facilitated by irrigation with bichlorid of mercury. In rare instances corrosive sublimate is absorbed and produces poisoning. In spite of these shortcomings and drawbacks it is a valuable aid to the surgeon and is very frequently used, especially upon the skin of the patient and the hands of the operator and his assistants. It should be dissolved in distilled water. Ordinary water causes a precipitate to form (common salt prevents the formation of this precipitate).

Because of the fact that corrosive sublimate is poisonous and very irritant, it should not be used upon serous membranes. It is absorbed quickly from serous membranes and destroys the endothelial cells and should not be introduced into the pleural sac, into joints, or into the peritoneal cavity. It should never be put within the dura, and should not be applied, in strong solution at least, to mucous membranes. It should not be introduced into the rectum for three reasons: First, it is intensely irritant and causes pain and inflammation. Second, it is useless, being largely and promptly converted into insoluble and inert sulphid of mercury. Third, a poisonous dose may be absorbed. Instruments cannot be placed in corrosive sublimate without being dulled, stained, and corroded. It is better to make the solution at the time it is needed, so as to have it fresh, for in old solutions much of the soluble corrosive sulbimate has been converted into insoluble oxychlorid of mercury, and the fluid has ceased to be germicidal. In order to make up fresh solutions use tablets, each of which contains about 7½ grains of the drug—one of these tablets added

to a pint of water makes a solution of a strength of 1 : 1000. Tablets which also contain ammonium chlorid are more soluble than those which contain corrosive sublimate only. Hot solutions of the drug are more powerfully germicidal than cold solutions. As corrosive sublimate is irritant, leads to profuse exudation, and may produce tissue necrosis, it should never be introduced into an aseptic wound. In such a wound it can do no good and may do much harm.

Griffin, in Foster's "Practical Therapeutics," sets forth the strengths of solutions applicable to different regions:

For disinfection of the surgeon's hands and the patient's skin, 1 : 1000; for irrigating trivial wounds, 1 : 2000; for irrigating larger wounds and cavities, 1 : 10,000 to 1 : 5000; for irrigating vagina, 1 : 10,000 to 1 : 5000; for irrigating urethra, 1 : 40,000 to 1 : 20,000; for irrigating conjunctiva, 1 : 5000; for gargling, 1 : 10,000 to 1 : 5000.

*Corrosive Sublimate Poisoning.*—Corrosive sublimate may be absorbed from a wound, a serous surface, or a mucous membrane, ptyalism and diarrhea resulting. The absorption of bichlorid of mercury may be followed by cramp in the limbs and belly, feeble pulse, cold skin, extreme restlessness, and even collapse and death. At the first sign of trouble withdraw the drug and treat the ptyalism (see page 338).

**Lithiomercuric Iodid.**—This material was prepared and tested by Dr. Rosenberger and Mr. England ("American Medicine," 1904, p. 1021). It is asserted that the iodid of mercury and lithium is more powerfully germicidal than corrosive sublimate, does not form inert albuminate when placed in a wound, and is not precipitated by alkalis. It is not nearly so irritant nor is it so poisonous as corrosive sublimate. I have given it an extensive trial in my clinic and am satisfied that it is superior to corrosive sublimate as a germicide, is less irritant, and is less poisonous. Its only objection is that it is more expensive.

**Carbolic acid** is a valuable germicide in the strength of from 1:40 to 1: 20. It is certainly fatal to pus-organisms, but weak solutions fail to kill most bacteria and do not destroy spores. Unfortunately, this acid attacks the hands of the surgeon; consequently in the United States dilute carbolic acid is chiefly employed as a solution in which to place the sterilized operating instruments, or as a germicide to prepare the skin of the patient before the operation is performed.

Carbolic acid is very irritant to tissues, and carbolized dressings may be responsible for sloughing of the wound or dry gangrene (see p. 182). Because of its irritant properties wounds which have been irrigated with it should be well drained. Carbolic acid, like corrosive sublimate, is inert in fatty tissues.

*Pure carbolic acid* is a reliable disinfectant for certain conditions. It is used to destroy chancroids, to purify infected wounds and abscess cavities, to disinfect the medullary cavity in osteomyelitis, to stimulate granulation after the open operation for hydrocele, or to purify sloughing burns and ulcerated areas. The pure acid rarely produces constitutional symptoms, but it occasionally causes sloughing. Its application causes pain for a moment only, and then analgesia ensues. Even dilute solutions of carbolic acid greatly relieve pain when applied to raw surfaces. The local action of carbolic acid can be at once antidoted by the application of alcohol (Seneca D. Powell). When carbolic acid is applied to a wound, the area about the wound should first be moistened with alcohol. After the application of pure carbolic acid to the interior of a joint, a wound, the medullary canal, or an infected area the surgeon should wait about one minute and then apply alcohol.

Dilute carbolic acid acts more slowly and less certainly than corrosive sublimate. It requires twenty-four hours for a 5 per cent. solution to kill anthrax

spores. Pus or blood (albuminous matter) greatly weakens the germicidal power of carbolic acid, and fatty tissue cannot be disinfected by it. It is not even the best of agents in which to place instruments, as it dulls them. After operation upon the mouth it may be used as a wash or gargle, 1 to 2 per cent. being a suitable strength. It is used sometimes to irrigate the bladder and often to cleanse sinuses, but is not employed in the peritoneal cavity, the pleural sac, the rectum, or the brain. It is occasionally injected into tuberculous joints. Carbolic solution should never be used in clean wounds.

*Carbolic Acid Poisoning.*—Carbolic acid is readily absorbed, and may thus produce toxic symptoms. Absorption is not uncommon when the weaker solutions are used, but seldom occurs when a wound has been brushed over with pure acid, because the pure acid at once forms an extensive zone of coagulated albumin, which acts as a barrier to absoprtion. One of the early indications of the absorption of carbolic acid is the assumption by the urine of a smoky, greenish, or blackish hue. This hue appears a little time after the urine has been voided, whereas the smoky hue of hematuria is noted in urine at once after it has been passed. The condition produced by carbolic acid is known as *carboluria*, and examination of such urine shows a great diminution or entire absence of sulphates when the acidulated urine is heated with chlorid of barium. The diminution of precipitable sulphates is explained by the fact that these salts are combined with carbolic acid, forming soluble sulphocarbolates. Such urine is apt to contain albumin. If during the use of carbolized dressing or the employment of carbolic solutions the urine becomes smoky, the use of the drug in any form must be at once discontinued, otherwise dangerous symptoms will soon appear. These symptoms are subnormal temperature, feeble pulse and respiration, muscular weakness, and vertigo. If death occurs, it is due, as a rule, to respiratory failure. The treatment of slow poisoning by carbolic acid consists in at once withdrawing the drug, giving stimulants and nourishing food, administering sulphate of sodium several times a day, and atropin in the morning and evening. (For Carbolic Acid Gangrene, see page 182.)

**Boric Acid.**—This drug is a very mild antiseptic. It is used to dust wounds. A solution of it is used to irrigate wounds and as the fluid for hot antiseptic fomentations. The solution should be concentrated.

**Acetate of Aluminum.**—This is a mild antiseptic, useful as a constituent of irrigating fluids and of hot fomentations. Its prolonged use hardens the tissues. A strength of 1 or 2 per cent. is employed.

**Saline Solution.**—Sodium chlorid solution of normal strength (0.9 of 1 per cent.) does not damage the cells of serous surfaces or of a wound, hence it is used as an irrigating fluid, and it is the best fluid for such a purpose. In intravenous infusion, in shock, or hemorrhage it is very valuable. It does not damage the blood-corpuscles as plain water does. It is, however, irritant to the kidneys when used by hypodermoclysis or intravenous infusion; hence if the kidneys are diseased saline fluid of one-half normal strength should be used for either of the latter purposes. Normal salt solution is prepared as follows: A quart of water is filtered and sterilized and in this 1½ drams of table salt are dissolved, and the fluid is again boiled (see pages 466 and 467).

**Thiersch's Fluid.**—This fluid is used upon mucous and serous surfaces and is employed to irrigate wounds. It is non-toxic and non-irritant. It consists of 1 gr. of salicylic acid and 6 gr. of boric acid to 1 oz. of sterile water.

**Alcohol** is a germicidal agent, which is most powerful when of the strength of 70 per cent. It may be used on the hands of the surgeon or the skin of the patient in a strength of 70 per cent. and may be used plain or mixed with corrosive sublimate, of the strength of 1 part of corrosive to 1000 parts of alcohol. Pure alcohol is used to arrest the local action of pure carbolic acid.

**Boiled water** is used to dissolve antiseptic materials; to inject by hypodermoclysis; to irrigate wounds, mucous cavities or serous surfaces, and as a fluid in which to keep instruments during the operation. It damages somewhat the tissue-cells of the surface of a wound and injures the cells of serous surfaces, hence for irrigation and hypodermoclysis salt solution is to be preferred.

**Creolin**, which is a preparation made by the dry distillation of English coal, is a germicide without irritant or powerful toxic effects. It is less powerful than carbolic acid, but acts similarly. It is not soluble in water, but is used in emulsion of a strength of from 1 to 5 per cent. It does not irritate the skin like carbolic acid.

**Peroxid** or **dioxid of hydrogen** is an excellent agent for cleansing a purulent or putrid area, but it is never applied to a sterile wound. It is prepared in a 10-volume solution, which should be diluted one-half to two-thirds before using. A 30 per cent. solution is known as *perhydrol*. It probably destroys the albuminous element upon which bacteria live, and thus starves the fungi. When peroxid of hydrogen is applied to a purulent area ebullition occurs, liberated oxygen bubbling up through the fluid and the pus being oxidized. The peroxid reaches every cranny and diverticulum containing pus. The peroxid of hydrogen is not fatal to tetanus bacilli; in fact, tetanus bacilli can be cultivated in a strong solution of it. It is very valuable as a mouth-wash to cleanse the mouth before and after operations in the oral cavity. Some surgeons use it to wash out appendicular abscesses (R. T. Morris). It must not be injected into a deep abscess in any region unless a large opening exists, as otherwise the evolved gas may tear apart structures, dissect up the cellular tissue, and spread infection. The use of peroxid should not be too long continued, for if used for a considerable period it makes the granulations edematous and retards healing. In fact, its continued use may actually prevent a sinus closing.

**Iodoform** is largely used by surgeons in spite of the fact that laboratory workers have assured us it is not truly a germicide, as bacteria will grow upon it. Clinical evidence, however, is in its favor and surgeons long ago concluded that it at least hinders the development of bacteria, directly antagonizes the action of the toxic products of germ-life, and stimulates the production of connective tissue. It is of the greatest value when applied to putrid foci, suppurating areas, and tuberculous processes. In putrid foci it probably combines with toxins and renders them less poisonous or even inert.

It attenuates the virulence of pus cocci and organisms of putrefaction. It renders its greatest service in tuberculous processes and is infinitely more powerful when oxygen is excluded than when it is present. The laboratory workers who condemn it have in many cases used nutrient material in which it does not dissolve (P. F. Lomry, "Archiv für klin. Chir.," 1896). D. B. Heile ("Proceedings of the German Surgical Congress of 1903") insists that iodoform is a valuable germicide if oxygen is excluded. He says, if iodoform is mixed with tissue juice, oxygen being excluded, the mixture becomes powerfully germicidal, even to streptococci, in from three to five days, although, as he maintains, neither constituent of the mixture when alone is germicidal. Tissue juice decomposes iodoform, liver juice decomposing it most rapidly, brain and fat decomposing it slowly. Granulation tissue decomposes it and tuberculous granulation tissue acts upon it most rapidly.

The conclusion of Heile is that this study confirms the clinical observation that iodoform is valuable in cavities, but not in free surfaces. My own belief is that it is more valuable in cavities than upon free surfaces, but when we are dealing with putrefactive areas, even on free surfaces, it is of great value. When iodoform decomposes on a free surface it sets free I, which we now know is a powerful germicide. When it decomposes in tissue juice Heile says it forms a powerful germicide which is rendered inert by oxygen. Clinically,

no real substitute for iodoform has yet been found. It can be rendered sterile by several washings with a solution of corrosive sublimate. It need not be applied to clean wounds, but the powder is very useful when dusted into infected wounds. It prevents wound discharges from decomposing and distinctly allays pain. Gauze impregnated with iodoform is used to keep abscesses open after evacuation, to drain the belly after certain operations, to pack aside the intestines and prevent their infection during some abdominal operations, and as packing to arrest intracranial hemorrhage. Iodoform gauze will drain serum well, but will not drain pus. In fact, it blocks up a pus-cavity, and if long retained leads to the collection of purulent matter behind and about the supposed drain. If used in an abscess, it must be replaced in twenty-four or thirty-six hours. Tuberculous joints and cold abscesses are injected with iodoform emulsion, which is made by adding the drug to sterile glycerin or olive oil. The emulsion contains from 4 to 10 per cent. of iodoform. Dunham's iodoform emulsion is valuable in suppurating cavities ("Annals of Surgery," May, 1909). In order to prepare it he adds to 100 c.c. of glycerin, 1 gm. of iodin, and 1 gm. of iodid of potassium, sets the mixture in an Arnold sterilizer, and boils the fluid in the sterilizer. By shaking the mixture the iodin goes into solution in the glycerin. When the mixture cools, 10 gm. of iodoform are added and ground into the mixture by use of a sterile mortar. A solution in ether of a strength of 10 per cent. may be used to inject the cavity of a cold abscess, but it is dangerous, may rupture the wall, and is more apt to produce poisoning than is the emulsion. Iodoform wax is used to fill cavities in bone (see p. 502).

*Iodoform-poisoning.*—The drug must be used with some caution. Absorption from a wound sometimes happens, producing toxic symptoms. These symptoms are frequently misinterpreted, being usually attributed to infection. R. T. Morris has pointed out that in iodoform-poisoning the wound seems to be in excellent condition, whereas in sepsis the wound appears unhealthy. The symptoms in some cases are acute and arise suddenly, and consist of hallucinatory delirium, nausea, fever, watery eyes, contracted pupils, metallic taste in the mouth, yellowness of the skin and eyes, an odor of iodoform upon the breath, the presence of the drug in the urine, the outbreak of a skin eruption resembling measles or one which is erythematous, vesicular, bullous, or petechial. There is often nephritis and always excessive loss of flesh and strength. Patients with such acute symptoms usually pass into coma and die within a week. Such attacks are most apt to arise in those beyond middle life (see Gerster and Lilienthal, in Foster's "Practical Therapeutics"). Iodin can be recognized in urine by adding a few drops of commercial nitric acid and a little chloroform. When the mixture is shaken the chloroform will take up the free iodin and become purple, and on standing the purple layer will settle to the bottom of the tube. Another method is as follows: Put a little urine in a saucer, add a little calomel, and stir. If the urine contains iodoform a brown color will be noted (R. T. Morris). The finding of iodin in the urine, however, is not proof that the patient is poisoned. We may find it when no sign of poisoning exists. In chronic cases of iodoform-poisoning the first symptoms usually observed are moroseness, bewilderment, and irritability, followed by depression, with unsystematized persecutory delusions, delirium, coma, and even death.

In systemic poisoning by iodoform, discontinue the use of the drug, sustain the strength of the patient, and favor the elimination of the poison.

Iodoform sometimes produces great local irritation of the cutaneous surface, the dermatitis being eczematous or else being manifested by crops of vesicles filled with turbid yellow serum or even bloody serum. These vesicles rupture and expose a raw, oozing surface, looking not unlike a burn. The dermatitis usually exists only in the region with which iodoform was in contact, but in some cases it spreads widely. The use of the drug must be at

once abandoned, for to continue it will not only increase the dermatitis, but may produce constitutional symptoms. Wash the vesiculated area with a stream of normal salt solution to remove iodoform, open each vesicle, and dress the part for several days with gauze wet with normal salt solution. After acute inflammation ceases apply zinc ointment or cosmolin.

**Aristol** is an odorless iodin compound used by some as an antiseptic dusting-powder.

**Loretin** is an antiseptic powder which is odorless, germicidal, non-irritant, and which is said to be non-toxic.

**Euphoren** is a powder containing iodin, and the iodin separates from it slowly when the powder is applied to wounds or burns. It does not produce toxic symptoms readily, if at all, and is a valuable substitute for iodoform. It is used especially in the treatment of ulcers and burns.

**Nosophen** is a pale-yellow powder containing 60 per cent. of iodin. Its bismuth salt is known as **antinosin.** Nosophen is not toxic, is free from odor, and is the best of the substitutes for iodoform.

**Acetanilid** is frequently used as a substitute for iodoform. It is of value when applied to suppurating, ulcerating, or sloughing areas, but it does not benefit tuberculous conditions. Sometimes absorption takes place to a sufficient extent to cause cyanosis, sweating, and weakness of the pulse and respiration. If cyanosis arises, suspend the administration of the drug and administer stimulants by the stomach.

**Airol** is a substitute for pure iodoform, and is composed of gallic acid, bismuth, and iodoform. It is non-irritant and non-toxic.

Among other powders we may mention iodol, amyloform, subiodid of bismuth, and dermatol or subgallate of bismuth.

**Silver** is a valuable antiseptic. Halsted and Bolton have shown that metallic silver exerts an inhibitive action upon the growth of micro-organisms and does not irritate the tissues. Credé has also demonstrated the same facts. These statements indicate one great reason why silver wire is such a useful suture material (see page 74). Halsted is accustomed to place silver foil over wounds after they have been sutured, and Credé employes as a dressing a fabric in which metallic silver is intimately incorporated.

Credé considers silver lactate (**actol**) an admirable antiseptic. It does not form an insoluble albuminate when introduced into the tissues and is not an irritant. Silver citrate (**itrol**) is said to be even a better preparation than silver lactate, and it is a useful dusting-powder. A preparation of metallic silver, known as *colloidal silver* or *collargolum,* is made. This preparation is soluble in water and in albuminous fluids. It is said to remain as metallic silver when in solution and to be powerfully germicidal. It certainly seems to cause temporary leukocytosis, but so do some other drugs which are not antagonistic to infections. It comes put up in 1- and 2-gr. tablets. A solution of the strength of from 1 to 5 per cent. is used. In severe cases of sepsis some advocate injecting this solution into a vein which has been rendered prominent by applying a bandage above the elbow. The dose is from 1 to 2 gr. of the drug. One injection or more may be given. I have never seen it do the slightest good and I believe that intravenous injections are dangerous. Some have given it subcutaneously, some by the mouth, others by enema. Subcutaneous injections are often very irritant and it is doubtful if the drug is absorbed from either the stomach or rectum. The most extraordinary claims have been put forth regarding the therapeutic value of collargolum. Its use has been advocated in the most diverse general infections. I believe it is of no real value. Its claims, in my opinion, have been shattered by the majority report of the committee of the American Medical Association ("Jour. Am. Med. Assoc.," March 13, 1909). **Credé's ointment of silver,** I believe, is of use in

infections of the skin and lymphatic vessels. I have used it repeatedly in such cases. In a child, 15 gr., in an adult, 45 gr. of the ointment are rubbed into the skin at one time, and the rubbing should be kept up from ten to thirty minutes. There is said to be no risk of argyria. **Protargol** is a silver salt much used in gonorrhea. A solution in water is made. It is not precipitated by albumin, alkalis, nor acids. In gonorrhea a 1 to 5 per cent. solution is used. **Argyrol** is a new and valuable preparation of silver which I have used frequently with much satisfaction. It is known as silver vitelline, is not irritant, and contains 30 per cent. of metallic silver. It is not precipitated by albumin. In a strength of 5 per cent. it is a very useful injection for gonorrhea, as it has powerful gonococcidal properties. In some types of chronic cystitis several drams of a 3 per cent. solution may be injected into the bladder from time to time, and much stronger solutions can be used with safety. Inflamed mucous membranes may be painted with a solution of a strength of from 20 to 50 per cent. A sinus or a sluggish area of granulation may be stimulated by touching with a solution of a strength of from 25 to 50 per cent. I have found it of much service in sinuses.

**Formaldehyd,** or **formic aldehyd,** has valuable antiseptic properties. **Formalin** (liquor formaldehydi) is a 37 per cent. solution of the gas in water. Solutions of this strength cauterize the tissues, but 1 per cent. solutions can be used to disinfect wounds. A solution of a strength of 0.5 per cent. is used to irrigate sinuses, tuberculous areas, abscess cavities, and suppurating joints. A strong solution is used to asepticize chancroids and other ulcers. A 2 per cent. solution disinfects instruments. The vapor of formalin can be so applied as to disinfect wounds, and Wood suggested its employment in septic peritonitis as a means of disinfection after the abdomen has been opened. Formic aldehyd gas thoroughly disinfects catheters.

**Formalin-gelatin** was introduced by Schleich as an antiseptic powder. The commercial preparation is known as **glutol.** When applied to a clean wound it gives off formalin and keeps the wound aseptic. When it is applied to a sloughing surface it will not give off formalin unless it is mixed with pepsin and hydrochloric acid. Formalin-gelatin has been used to fill bone cavities.

**Lysol** is a clear, brownish, oily fluid with an odor like creasote. It is a valuable germicidal agent. It is saponified phenol and is used in a solution of a strength of from 1 to 3 per cent. It does not attack the hands like carbolic acid and is much less poisonous.

**Mustard** is an excellent emergency germicide. Its value has been demonstrated by Roswell Park, who uses a mixture of soap, cornmeal, and mustard flour to scrub the surgeon's hands or the patient's skin. I have used it repeatedly with entire satisfaction. Mustard removes the odor of decay at once.

**Commercial gasolene** is used by Riordan and others to clean wounds and ulcers, and to prepare the field of operation. Its vapor is so inflammable that the material must not be used when gas or lamp light is necessary. It is used only in the daytime or in a room lighted by electricity, and on free surfaces where evaporation is rapid. It is sterile, non-irritant, and on evaporation leaves a dry, clean surface.

**Iodin.**—This drug was strongly endorsed by the late Prof. Nicholas Senn ("Surgery, Gynecology, and Obstetrics," July, 1905). He regarded it as the most powerful and the safest of antiseptics, and claimed that in solutions of a strength of 1 per cent. it is non-irritant and causes a protective phagocytosis. It may be used in great dilution or the tincture may be applied to an infected wound in the same manner as is pure carbolic acid; a method advocated by Carl Beck. In dilute solution it is used to irrigate sinuses. The proper dilu-

tion for irrigation of a sinus is obtained when the tincture is diluted to the color of sherry wine. Its employment for sterilizing the skin is described on page 68.

**Nucleins,** especially protonuclein, possess germicidal powers. Nuclein is composed of nucleinic acid and protein material. When injected hypodermatically and to a less degree when taken by the mouth it increases the germicidal power of the blood-serum, causes leukocytosis and increased phagocytosis, and thus prevents or opposes infection. Mikulicz has used nucleinic acid to increase vital resistance as a preliminary to operation (see page 42). A 1 per cent. solution of nucleinic acid is on the market. This acid is made from yeast. The dose of the preparation is from 10 to 60 minims hypodermatically, once or several times a day. *Protonuclein* probably contains nucleinic acid and is of some value when applied locally to areas of infection, particularly when sloughing exists.

**Heat.**—The best germicide is heat, and the best form in which to apply heat is by means of boiling water (which is even better than steam). One can use boiling water upon instruments and dressings, but seldom upon a patient. Jeannel, of Toulouse, uses boiling salt solution in abscess cavities, and some other surgeons employ steam or boiling water to disinfect the medullary canal in osteomyelitis. Nevertheless, boiling water is seldom applied to a patient, and in many cases a chemical germicide must be used.

Among other antiseptics and germicides of more or less value we may mention trichlorid of iodin, chlorid of zinc (10 per cent. solution), chlorid of iron, salol, oxycyanid of mercury, fluorid of sodium, argonin, sugar, lannaiol, bichlorid of palladium (in very dilute solution), thymol, potash soap, salicylic acid, sulphate of copper, arsenite of copper, camphor, eucalyptol, cinnamon, bromin, chlorin (as gas or as chlorin-water), cinnamic acid, permanganate of potassium or of calcium, chlorate of potassium, and oxalic acid. The surgeon before operating should always scrub his hands in a germicidal solution.

**Distribution of Bacteria.**—Microbes are very widely distributed in nature. They are found in all water except that which comes from very deep springs; in all soil to the depth of 3 ft.; and in air, except that over the desert, over the open sea, and that about lofty mountains. Dust-free air does not contain them; the more dust, the more microbes, hence they are present in greatest number in the air of towns. There are more in narrow courts than in broad highways, more in crowded attics than in roomy apartments. Bacteria are present on and in the skin, in the alimentary canal, in the nose, mouth, and pharynx, and in the blood and lymph. As Adami points out, under normal conditions the bacteria which enter the blood are very quickly killed.

Microbes may be useful. Some of them are scavengers, and clean the surface of the earth of its dead by the process known as "*putrefaction*," in which complex organic matter is reduced to harmless gases and to a mineral condition. The gases are taken up from the air by vegetables, and the mineral matter is dissolved in rain-water and passes into the soil from which it came, there again to be food for plants, which plants will become food for animals. Other organisms purify rivers; others cause bread to rise; still others give rise to fermentation in liquors. Microbes may be harmful. They may poison rivers and soils; they may be parasites on vegetable life; they cause diseases of the growing vine, and also of wine; they produce the mold on stale, damp bread; they occasionally form poisonous matter in sausages, in ice-cream, and in canned goods; and they produce many diseases among men and the lower animals.

With so universal a distribution of these *fungi*, man must constantly take them into his organism. They are upon the surface of his body, he inhales them with every breath, and he swallows them with his food and drink. Most

of them, fortunately, are entirely harmless; others cannot act on the living tissues; but some are virulent, and these are generally, but not always, destroyed by the cells of the human body. The alimentary canal always contains bacteria of putrefaction, which act only upon the dead food, and not upon the living body; but when a man dies these organisms at once attack the tissues, and postmortem putrefaction begins in the abdomen. Even pathogenic bacteria may exist for long periods in the tissues without causing illness in the host, but when such bacteria do persist, they may at any time and from a variety of causes become active in producing disease in the carrier, or when they pass from the host they may perhaps infect other people (see Typhoid Carriers, page 38). Sternberg found pneumococci in healthy sputum. In fact, pneumococci can be found in the mouths of 25 per cent. of people free from pneumonia. Pneumococci obtained from the mouth of an individual free from pneumonia "are just as pathogenic as the diplococci obtained from a culture from the sputum of a pneumonic patient" (Sir Thomas Oliver, in "British Medical Journal," April 30, 1910). If the lungs become irritated, insufflation of diplococci from the mouth may be followed by pneumonia. Every infection is at first local. Some infections remain local, but others become general. In a general infection the micro-organisms are in the blood, though often we cannot find them because of imperfect methods (Ball, in "Lancet," June 8, 1912).

Welch long ago pointed out that the human skin normally contains the Staphylococcus epidermidis albus, even after the most careful surgical cleansing. Dudgeon, in the Horace Dobell Lecture for 1908 ("Lancet," Dec. 5, 1908, "Latent Persistence and the Reactivation of Pathogenic Bacteria in the Body"), says that healthy organs may contain various bacteria, that the tissues of the fetus are sterile, but in childhood and adult life "bacteria are found in various parts of the human body; that Staphylococcus albus can be cultivated from the human omentum in cases in which the peritoneal cavity is apparently healthy"; that pus cocci may persist for long periods in a scar; that virulent diphtheria bacilli may be "found in the throats of persons who have come in contact with diphtheria patients, but show no signs of the disease"; that the Bacillus proteus is frequently found in the urine; that colon bacilli normally inhabit the intestinal tract and appendix and frequently exist in the urinary tract without giving rise to inflammation or symptoms of disease, and that typhoid bacilli tend notably to persist (see page 38). As previously stated, the organisms of tuberculosis and of syphilis may long rest latent.

**Koch's Circuit.**—To prove that a microbe is the cause of a disease it must fulfil Koch's circuit. It must always be found associated with the disease; it must be capable of forming pure cultures outside the body; these cultures must be capable of reproducing the disease; and the microbe must again be found associated with the artificially produced morbid process.

**Infection or Disease Production by Micro-organisms.**—Most infections are caused by bacteria, some result from molds, a few from protozoa. Pathogenic organisms cannot enter through the sound skin and the unbroken skin without causing the formation of lesions at the point of entrance. The sound skin is the very best antiseptic covering for tissue, as ordinary bacteria cannot pass it at all. Some bacteria by entering the ducts of cutaneous glands may cause disease. Disease-producing organisms which enter the body may reach the focus in which they act from outside of the body, entering by inoculation, inhalation, or ingestion. In most instances organisms which enter the body from without are rapidly destroyed. When they enter in large numbers, or when they are very virulent, or when the vital resistance of the individual is at a low ebb they cause disease. Bacteria may reach the region in which they become active from some other part of the body. Bacteria seldom dwell in the body long without inducing disease, but spores can lie dormant in the

system for years. When bacteria or spores from some other part of the body reach a region of injury or disease they may become active; this area is a damaged and weakened part, in it the circulation is abnormal, it is a so-called *point of least resistance* (a *locus minoris resistentiæ*) which affords a nest for them to develop and to multiply, the cellular activities of the weakened part being unable to cope with the activities of the germs. Even large numbers of pathogenic organisms may induce no trouble in a healthy man; but let them reach a damaged spot, and mischief is apt to arise. Kocher established subcutaneous bone injuries in dogs, and these injuries pursued a healthy course until the animal was fed upon putrid meat, whereupon suppuration took place. This experiment proves that micro-organisms can reach a damaged area by means of the blood, and it enables us to understand how a knee-joint can suppurate when we merely break up adhesions, and how osteomyelitis can follow trauma when the skin is intact. A given number of organisms might produce no effect on a healthy man, whereas the same number might produce disease in an individual who was weak or ill-nourished, suffering from depression or fear, or debilitated by the habitual use of alcohol. The personal equation plays a great part in disease production. Some individuals seem to be immune to certain diseases; and these immunities and liabilites may be hereditary or acquired, temporary or permanent.

The local infection may be violent and yet never be generalized. Sometimes the local reaction at the point of bacterial entry is so trivial as to be overlooked, or by the time general infection occurs the initial point of local infection may have healed.

It is not at all unusual to observe lymphangitis or lymphadenitis "above a healed focus" (J. C. Bloodgood, in "Progressive Medicine," Dec., 1911). A few infections generalize primarily by way of the blood—most do so by the lymphatics. From the moment there is lymphangitis or lymphadenitis the infection is to be regarded as general, and the blood contains toxins or bacteria, the lymph-glands filtering out and holding comparatively few micro-organisms. (See J. C. Bloodgood, in "Progressive Medicine," Dec., 1911, and Noetzel, in "Beitr. z. klin. Chir.," 1909, lxv).

A general infection in which the blood contains toxins but not bacteria is called a *toxemia*. A general infection in which the blood contains bacteria is called a *bacteremia*.

The intensity of an infection may often be estimated by the degree of leukocytosis and anemia (see page 42).

**Enzymes.**—Bacteria contain and excrete ferments, and these ferments are known as enzymes. Bacterial ferments resemble pepsin and trypsin, the digestive ferments. The digestive ferments convert albumin into peptone, starch into sugar, and break up fat. Some enzymes are proteolytic (dissolve albumin); some are diastatic (change starch into sugar); some convert cane-sugar into grape-sugar; some coagulate milk. When microbic infection of the tissues occurs the enzymes of the bacteria act upon the tissues just as the digestive ferments act upon the food, and form microbic albumoses. The enzymes are the weapons of micro-organisms. By means of these ferments bacteria not only prepare substances for assimilation, but seek to destroy antagonists and cell enemies. It is probable that enzymes when absorbed are frequently productive of toxemia.

**Toxins** are poisons produced by microbic action. The action of pathogenic bacteria upon the tissues is of great importance. In the first place, they invade the tissue, the capacity for invasion varying greatly and depending on their power to sweep aside the defenses of the body. When they invade the tissues they abstract from the blood, the lymph, and the cells certain elements necessary to the body—as water, oxygen, albumins, carbohydrates, etc.—

and thus cause body wasting and exhaustion from want of food. In the second place, bacteria produce a vast number of compounds, some harmless and others highly poisonous. The symptoms of a microbic disease are largely due to the absorption of poisonous materials from the area of infection. These poisons may be formed from the tissues by the action upon them of the bacterial ferments (see page 36), may be excreted by the bacteria (*extracellular toxins*), or may be liberated from the bodies of degenerating microbes (*bacterial protein*, *intracellular toxins*, or *endotoxins*). Intracellular toxins are very insoluble. Bacteria contain and secrete ferments; and as albumoses are formed in the alimentary canal by the action of digestive ferments upon proteins, sugars, and starches, so microbic albumoses are formed by the action of microbic ferments upon tissues. Just as the albumoses formed in digestion are poisonous when injected, so the albumoses of microbic action are poisonous when absorbed. The albumoses of microbic action are called *toxalbumins*, and these albumoses often operate as virulent poisons to the body-cells.

A number of compounds formed during the microbic destruction of tissue are *alkaloidal* in nature. These poisonous alkaloids are readily diffusible and, many of them, very virulent. It is probable that every pathogenic organism has its own special toxin which produces its characteristic effects, although the effects are modified by the nature of the soil—that is to say, by the condition of the tissues. Again, one micro-organism may produce several toxins. The absorption of toxins may be very rapid; for instance, the toxins of cholera may kill a man before the bacilli have migrated from the intestine. Brieger uses the term *toxin* to designate all of the poisonous products of bacterial action. He divides toxins into alkaloidal or crystallizable and amorphous, the latter being called toxalbumins.

**Ptomains.**—By many writers the term "ptomain" is used to designate these toxins, but, in reality, a ptomain is a form of toxin produced by the action of saprophytic bacteria. A ptomain is a putrefactive alkaloid and a toxin is any poison of microbic origin. Among these putrefactive alkaloids may be mentioned tetanin, typhotoxin, sepsin, putrescin, tyrotoxicon, muscarin, and spasmotoxin. The poison which occasionally forms in cheese, ice-cream, sausage, and canned goods is composed of ptomains. Poisoning by any putrid food is called ptomain-poisoning.

**Leukomains** must not be confounded with the above-mentioned bodies. Leukomains are alkaloidal substances existing normally in the tissues and not produced by bacteria, but arising from physiologic fermentations or retrograde chemical changes. They are natural body constituents, in contrast to toxins, which are morbid constituents. Leukomains are found in expired air, saliva, urine, feces, tissues, and the venom of serpents. If not excreted, these bodies may induce illness, and when injected may act as poisons. Ordinary colds and some fevers result from leukomains; they play a great part in uremia, and when excretion is deficient the retained leukomains make the system a hospitable host for pathogenic bacteria. Sickness due to the retention and absorption of leukomains is known as *auto-intoxication*. Among leukomains may be mentioned adenin, hypoxanthin, and xanthin, allied to uric acid, and other substances allied to creatin and creatinin. The surgeon should never forget the possibility of harm being done by retained leukomains, and should endeavor to prevent auto-intoxication in all cases by keeping the skin, the bowels, and the kidneys active.

**Immunity.**—*Resistance* is the fight of the body against bacteria. Even a person with high resistance may be infected, but even though infected the body still fights the bacteria. If a person cannot be infected with a certain disease, he is said to be immune to it. Some persons seem naturally immune to certain diseases (*natural immunity*). Immunity to some diseases may be produced

artificially. When the body itself produces the materials which render it immune the immunity is called *active*. When immunity is produced by the introduction of substances artificially produced the immunity is called *passive* or *artificial*. It has long been known that when a person recovers from certain diseases he has become immune to the disease from which he suffered (*acquired immunity*). Immunity may be transitory, prolonged, or permanent. Acquired immunity may be compared to fermentation. When fermentation ceases, the addition of more ferment is without result. When a person recovers from certain diseases, the addition to his blood of more of the causative bacteria is also void of result.

Immunity was long believed to arise from the exhaustion of some unknown constituent of tissue necessary to the life of the bacteria. This theory was advanced by Pasteur. It has been abandoned because of the demonstration that though an animal may become immune to a disease caused by certain bacteria, these bacteria may continue to live in the host. It is true that when recovery ensues upon infections, as a rule, the causative bacteria disappear, but there are enough exceptions to this rule to invalidate the theory of Pasteur. It is well known that even for years after an attack of typhoid fever the bacilli may exist in the gall-bladder or the bone-marrow, or be passed in the urine or feces. A person apparently well, yet holding, for instance, in his gall-bladder infectious bacteria, is called a "*bacteria carrier*." From 1 to 2 per cent. of persons who have had typhoid fever years ago pass bacilli in the stools, and such carriers are often responsible for the spread of the disease. Hutchinson ("British Medical Journal," March 26, 1910) reports a "carrier" whose attack had been fifteen years before. Gregg ("Boston Med. and Surg. Jour.," July 16, 1908) reported the case of a typhoid carrier fifty-two years after recovery from typhoid. Some carriers never knew they had had typhoid, the attack having been very mild. Some have never had it, but have been in contact with it. Such people were immune. A carrier may give the disease to others, but is practically immune. "Certain protective immune bodies, such as opsonins and stimulins, are augmented by the attack; others, such as the bactericidins and bacteriolysins, are not augmented to the point of destruction of the bacilli" (Willard J. Stone, in "American Journal of Medical Sciences," April, 1912). Fromme operated upon 4 typhoid carriers. In each case he removed the gall-bladder. In the bile of each he found bacilli. After operation the bacilli disappeared from the feces of each woman ("Deutsch. Zeitsch. f. Chir.," Nov., 1910). A theory proposed by Chauveau is known as the "retention theory," and is the opposite of Pasteur's "exhaustion theory." According to Chauveau, bacteria growing within the body leave as a legacy excrementitious material, and the accumulation and retention of excrementitious products produce immunity.

Until very recently one set of investigators maintained that immunity depends upon the activity of certain body cells which attack, consume, and destroy bacteria, this is the theory of *phagocytosis* (see page 41). Another set asserted the claims of Nuttal and Buchner, that normal fresh blood-serum is germicidal, the power varying for different bacteria and being limited. A fixed amount of serum is capable of destroying a fixed number of bacteria of a certain variety. Vaughan and others states that the germicidal agent is probably a nuclein furnished chiefly by the white cells and held in solution by the alkaline serum. This germicidal agent of normal serum Buchner called "*alexin*" or *defensive protein*, and explained immunity by its presence. This theory is known as the "*humoral theory*." According to this theory as originally maintained, when an animal is naturally immune to a bacterial disease it is assumed that the blood-serum and body fluids contain enough of this alexin to dissolve or destroy the bacteria. Neither method of defense is the only one.

In all probability both phagocytosis and bacterial solution are occurring in the same patient at the same time, phagocytosis being impossible but for the serum and bacteriolysis being impossible without leukocytes.

Since the above theories were set forth it has been found that when an animal recovers from some bacterial diseases the blood-serum and body fluids contain new protective materials called, in general, *antibodies*. The toxins of bacteria stimulate body cells to the production of antibodies, and antibodies bring the disease to an end and secure immunity. It is thus seen that the very poisons produced by bacteria cause the body cells to produce poison antidotes. The bacteria may be so virulent or the patient so susceptible that poison overwhelms the cells, antibodies are not formed in sufficient quantity, and death ensues. The cells may be badly poisoned and the patient may become very ill, and yet after a time the cells may regain enough vitality to furnish antibodies in sufficient quantity to bring about cure and to secure immunity. The bacteria may be so few in number or so attenuated in virulence or the cells of the patient may be so active that quantities of antibodies are quickly formed under mild stimulation, and the individual does not take the disease at all or takes it very mildly.

The **lytic** or **bacteriolytic antibodies** or **lysins** destroy and dissolve bacteria. All bacteria are not susceptible to lysis, for instance, streptococci, tubercle bacilli, and pneumococci. When recovery ensues the causative bacteria usually but not always disappear (see page 38). These lytic bodies are formed by the leukocytes, bone-marrow, spleen, and lymph-glands (Wassermann, in "Berlin. klin. Woch.," No. 4, 1898, and Levaditi, in "Annales de l'Inst. Pasteur," 1904).

**Agglutinins** and **precipitins** gather in the blood-serum of an animal when the animal has been injected with bacteria or certain cells. When these antibodies appear in blood after an animal has been injected with bacteria they agglutinate and precipitate the bacteria injected. It is probable that agglutinins and precipitins are formed by the endothelium of vessels walls (Kraus and Schiffman, in "Annales de l'Inst. Pasteur.," 1906). These materials only appear after certain infections.

*Opsonins* are materials which by attaching themselves to certain bacteria so alter the bacteria that they easily become the victims of phagocytosis (see Phagocytosis, page 41).

*Antitoxins* are specific bodies secreted, as Roux says, by the body cells. They pass into the serum and body fluids. They fix and neutralize the bacterial toxin by combining with it, but do not dissolve, kill, precipitate, or agglutinate the bacteria. The first antitoxin to be discovered was that of diphtheria. The discovery was made by Behring in 1890.[1] He found that if an animal is injected with gradually increasing amounts of diphtheria toxin the serum comes to contain an antitoxic material. Very soon after this discovery was announced Behring and Kitasato made a like discovery in regard to tetanus toxin. It was pointed out by Kitasato and Behring that animals can be rendered immune to tetanus by artificial means and that the blood-serum of immune animals will, if injected into other animals, render them immune, or perhaps cure the disease if injected into animals suffering from tetanus. The same statements were also proved to be true of diphtheria. Now many experimenters are endeavoring to find the antitoxin of each microbic disease for the purpose of using it therapeutically and also as a preventive agent.

In some infections soluble toxins are not formed and the body resistance depends largely on the formation by the bacteria of substances which finally, when present in sufficient amounts, destroy bacteria.

Surely one of the most important of modern discoveries is that certain

[1] "Deutsche Med. Wochenschrift," 1890, Nos. 49 and 50.

substances introduced into the body cause a reaction which results in the formation of antibodies. Any material which causes antibodies to form is called an antigen. In the preceding section we have spoken of bacterial products as the antigens. But other antigens exist, for instance, blood-corpuscles and other cells, blood-serum, some vegetable poisons, and some animal poisons. Thought is now directed to treating bacterial diseases by the introduction of the proper antigen to produce lysins, opsonins, antitoxins, as the case may be. Streptococci produce no antigen which leads to lysin formation, but do produce antigens which lead to antitoxin and opsonin formation.

The subject is of enormous importance and is vastly complicated.

*Ehrlich's Theory of the Mechanism of Immunity by Antitoxins.*—Ehrlich's theory was advanced in 1898 and is generally accepted at the present time. "Ehrlich's theory of the mechanism of immunity is based upon Weigert's teaching of the process of tissue repair. It is a matter of universal observation that nature is prodigal in her attempts to repair an injury. This is shown in the healing process in an ordinary wound. A much larger amount of material is thrown out to bridge the chasm than is really utilized in the formation of new tissue. The presence of an excessive amount of new material is shown by the fact that the part is raised above the level of the surrounding sound tissue, and this excess is removed gradually as the new-formed tissue becomes stronger and stronger, until finally the wound is marked by a line of white scar-tissue, the excess gradually passing into the blood-current.

"Ehrlich believed that the mechanism of immunity was explainable on a similar basis. It had become evident from the experiments of Wassermann with the tetanus bacillus that its toxin had an especial affinity for the cells of the central nervous system. Experiments with other bacteria pointed to the fact that the toxins of different species of bacteria had an especial affinity for the cells of different organs of the body. When the amount of poison entering the body is not sufficient to destroy the cells which have an especial affinity for it, these cells may be injured only to such an extent as to permit subsequent repair. In order to comprehend Ehrlich's hypothesis it is necessary to conceive the cells of the body as having a complex structure which may be stated diagrammatically as consisting of a central mass or nucleus from which radiate a number of 'lateral chains,' or bonds, each of which serves to bind the cell to other substances. In the case of the cells of the central nervous system one of these lateral bonds has an especial affinity for tetanus toxin and suffers destruction. The cell now finds itself in unstable equilibrium, and at once proceeds to repair the damage wrought. As in the case of tissue repair, the new material produced is far in excess of the required amount. The excess finds its way into the blood-current. This material now circulating in the blood-current has the same affinity for tetanus toxin as when united with the central mass of a cell as its lateral bond, and can, therefore, combine with tetanus toxin floating in the blood-current, thus preserving other cells from injury. The union formed between the lateral bond of the cell (which is really the antitoxin) and the tetanus toxin results in the formation of a compound which is physiologically inert. According to Ehrlich's idea, therefore, the antitoxin is simply the excess of lateral bonds floating in the blood-current. This substance can neutralize the effect of the tetanus toxin in a test-tube just as readily as it does within the body" (D. H. Bergey, "American Medicine," October 11, 1902).

**Phagocytes.**—It was generally believed after Metschnikoff's important discoveries that leukocytes were the agents which protected the body from infection. When other observers found that in blood-serum is material that damages or destroys bacteria, opinion swung to the veiw that the blood-serum contains the protective element, and that the leukocytes are simply scavengers

and remove dead bacteria, but do not destroy living ones. It has recently been shown that under some circumstances leukocytes destroy living bacteria and under other circumstances they do not, and that the presence or absence of this property depends in most instances upon the presence or absence in the blood-serum of substances which act upon bacteria and render them susceptible to the phagocytic action of leukocytes. We say in most instances, not in all instances, because certain bacteria, for instance, influenza bacilli, are phagocytable without the presence of opsonic serum (Ludvig Hektoen, address in "Section of Physiology and Experimental Medicine, American Assoc. for Advancement of Science," 1908; "Science," Feb. 12, 1909). The existence of substances in the serum provocative of phagocytosis was demonstrated by Wright and Douglas in 1903, and they named them *opsonins*. If opsonins are present, they act upon bacteria, and render the bacteria susceptible to phagocytosis. (See Ludvig Hektoen, in "Jour. Am. Med. Assoc.," May 12, 1906.) Opsonins act upon bacteria and alter them, and the altered bacteria are easily eaten up by leukocytes. Very virulent bacteria resist phagocytosis because they have little affinity for opsonin, and such virulent bacteria may grow in opsonic serum. The source of opsonins is not known, but serum normally contains "opsonins for many different bacteria" (Hektoen, in "Jour. Am. Med. Assoc.," May 12, 1906). When experiment determines the fact that an individual's leukocytes are highly phagocytic toward particular bacteria, we believe that a quantity of opsonin for that variety of bacteria is present, and we may say the individual has a high *opsonic index* as regards them. Under opposite conditions we say he has a low opsonic index. "The opsonic index of Wright with respect to a given bacterium is obtained by comparing the number of the bacteria taken up under the influence of the serum of the person or animal in question with the number taken up under the influence of the corresponding standard of normal serum under conditions that are as comparable as they possibly can be made" (Ludvig Hektoen, address in "Section of Advancement and Physiology and Experimental Medicine in Am. Assoc. for Science," 1909; "Science," Feb. 12, 1909).

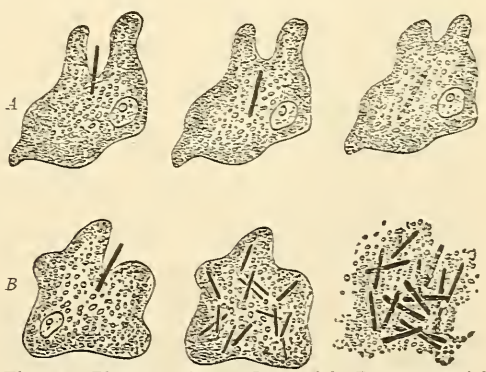


Fig. 14.—Phagocytosis: *A*, Successful; *B*, unsuccessful (Senn).

**The Process of Phagocytosis.**—We have just seen how opsonins stimulate phagocytosis. The process of destruction of bacteria by cells is known as *phagocytosis*, and the destroying cells are called *phagocytes*. The cells active in phagocytosis are the endothelial cells of the blood-vessels, lymph-channels and lymph-spaces, and particularly the leukocytes. When infection occurs, the white blood-cells gather in enormous numbers at the seat of disease, encompass and surround the bacteria, and build a barrier to prevent dissemination of the microbes and general infection of the victim. The force which draws leukocytes to a region of infection also tends to draw them to an area where there is cellular degeneration or death. This force is called *positive chemiotaxis* and is greatly stimulated by opsonins. In very virulent infections the leukocytes may fail to collect and may actually be repelled and scattered under the influence of what has been called *negative chemiotaxis*. Phagocytes at the seat of infection try to eat up, carry away to a gland, and there digest

and destroy bacteria. A battle royal occurs, the microbes fighting the body cells with most active ferments and destroying the opsonic power of the blood-liquor; the body cells endeavoring to devour and destroy the bacteria (Fig. 14), in which effort opsonins give them aid. In some cases the bacteria win absolutely and the patient dies. In other cases they win for a time and overwhelm the system; but presently the body cells, whose movements were inhibited by the poison, regain their activity and are then immune to the bacterial poison. It is probable that the materials thrown out by the white cells during the combat with the microbes tend to destroy bacterial products and to neutralize toxic products of tissue destruction. These materials, which neutralize toxic products, are known as *antitoxins* (see page 39). After the attack of disease has passed away the body cells have been educated to withstand this poison, and new cells in the future retain this capacity; the weak cells were killed, the fittest survived, and the body fluids contain antitoxin. The new cells formed in the body are insusceptible to the poison and the individual is said to be *insusceptible* or *immune*. The theory of phagocytosis immunity assumes an educated white corpuscle and body cell. This view originated with Sternberg, but it is usually accredited to Metschnikoff. Lankester gave us the term "educated corpuscle."

**Leukocytosis.**—In a number of infectious and inflammatory diseases leukocytosis occurs. By this term we mean a notable increase of leukocytes in the blood, the polynuclear neutrophiles being increased relatively and absolutely. Leukocytosis in an infection indicates that the body is trying to protect itself against poisons by furnishing more phagocytes to attack bacteria. The degree of the leukocytosis is a sort of gauge of the virulence of the infection and of the reacting or resisting powers of the individual. In a very trivial infection there may be a slight leukocytosis or no leukocytosis at all. A violent infection, if resistance is high, is accompanied by a high degree of leukocytosis; if resistance is low, there is a low degree or no leukocytosis at all.

In a virulent infection absence of leukocytosis is of unfavorable import. It means that tissue resistance is at an end and that the body cells have ceased to fight the bacteria.

Normally, the blood should contain about 7500 leukocytes per c.mm. From 60 to 75 per cent. of the cells are polynuclear neutrophiles. If the cells number well above 10,000, and if the percentage of polynuclear neutrophiles is increased, the condition is regarded as leukocytosis.

In most cases the leukocyte count is below 20,000. Over 20,000 is high leukocytosis. It is very seldom that a count of over 30,000 is obtained (see page 93).

**Artificial Stimulation of Phagocytosis.**—When active hyperemia is induced by heat, when irritants are applied to an inflamed surface, or when an inflamed joint is treated by Bier's method of passive hyperemia, local leukocytosis is stimulated and phagocytosis becomes more active. A few years ago Issaëff affirmed that the introduction of certain materials, as salt solution, into the peritoneal cavity, lead, for a time, to great increase in the resistance to abdominal infection. This period of increased resistance he called the *resistance period*. It begins a few hours after the injection and terminates by the end of the fifth day. During this period the great increase in intraperitoneal leukocytes saves the animal from infection with bacteria, which would otherwise cause a dangerous or fatal inflammation. Mikulicz believed it possible to establish this resistance period before abdominal operations and was working on the problem just before his lamented death. Mikulicz used diluted nucleinic acid injected twenty-four to forty-eight hours previous to operation (Mikulicz, "Verhandl. d. 33. Congress d. Deutsch. Ges. f. Chir.," 1904). Graf's studies of nucleinic acid used to secure immunity from puerperal sepsis were inconclusive

("Zentralblatt für Gynäkologie," 1910, No. 27). Some surgeons have injected fresh warm horse-serum for the same purpose (Petie, Jayle, and Federmann; see "Le Presse Medicale," 1905). The agents used must not be of a nature to damage opsonins, for leukocytosis without plenty of opsonins would do no good. A true infectious or inflammatory leukocytosis is much more protective than an artificial leukocytosis, however induced. In fact leukocytosis induced, for instance, by injecting such a material as nuclein is of very little use against an established infection. It is probably of more value as a protective against infection. How slight or how great this value may be is not yet certainly determined.

Vital Resistance.—Local resistance is lowered by injury or disease of the skin or mucous membrane. Sound skin and mucous membrane are most important elements in resistance. For instance, disease of the intestinal mucosa may permit colon bacilli or other micro-organisms to be taken through the damaged mucous membrane and be carried to distant regions, in which regions of arrest disease may arise.

In the same manner pulmonary tuberculosis may develop subsequent to disease of the bronchial mucous membrane. The general vital resistance to infection depends in part upon germicidal and opsonic blood-liquor and in part upon active leukocytes.

Vital resistance is increased by agents which cause active phagocytosis without destruction of opsonins.

Anything that lessens the germicidal and opsonic power of blood-serum or the phagocytic activity of corpuscles lessens general vital resistance. Among these causes are ill health, worry, unhygienic life, chronic drug intoxications, alcoholism, chronic visceral diseases, diabetes, Bright's disease, gout, rheumatism, violent and sudden fluctuations of temperature, bodily or mental overwork, improper food, insufficient food, too little sleep, fear, antecedent illness, exposure to cold, and the creation of points of least resistance (see page 36). The general vital resistance to infection is also lowered by inordinate prolongation of a surgical operation, by shock, by hemorrhage, by protraction of the anesthetic state with ether or chloroform. Exham ("Brit. Med. Jour.," Jan. 27, 1912) points out that inflammation is a factor in resistance (this is seen by pus limitation in appendiceal abscess), and so is fever. Elevated temperature means that the body is fighting the infection (see page 129).

Different tissues in the same individual show great differences in resistance. We know that certain bacteria have a predisposition to attack certain tissues. This is notably true of tubercle bacilli. The resistance of an individual varies at different times. Heredity often plays a part in predisposition and resistance and sex has some influence upon it. Race is influential regarding some diseases (see page 24). Resistance varies at different ages. Exham ("Brit. Med. Jour.," Jan. 27, 1912) points out that children are particularly prone to acute infections, but are less apt to die of them than are adults.

Protective and Preventive Inoculations.—Our knowledge of protective inoculations for contagious diseases dates from Jenner's discovery of vaccination against small-pox in 1798. Preventive inoculations with attenuated virus are due to the experiments of Pasteur. This observer discovered the cause of chicken-cholera, and cultivated the micro-organism of the disease outside the body. He found that by keeping his cultures for some time they became attenuated in virulence, and that these attenuated cultures, inoculated in fowls, caused a mild attack of the disease, which attack was protective, and rendered the fowl immune to the most virulent cultures. Cultures can be attenuated by keeping them for some time, by exposing them for a short period to a temperature just below that necessary to kill the organisms, or by treating them with certain antiseptics. It has further been shown that injection of the

blood-serum of an animal rendered immune by inoculation is capable of making a susceptible animal also immune.

A most important fact is that animals may be rendered immune to certain diseases by inoculating them with filtered cultures of the microbes of the disease, the filtrate containing microbic products, but not living microbes. By this method animals can be rendered immune to tetanus and diphtheria. Pasteur's protective inoculations against hydrophobia owe their power to microbic products, and Koch's lymph contains them as its active ingredients. Injections of dead bacteria is the basis of inoculation against typhoid fever. Inoculation with dead bacteria is called *vaccination* and the dead bacteria constitute the *vaccine* (see page 47).

Vaccination against typhoid has been notably successful in the army and navy of the United States. Surgeon-General Stokes of the U. S. Navy reports the entire disappearance of typhoid from the navy as the result of vaccination. During the summer of 1911 an army division of nearly 13,000 men was stationed in Texas for four months. It was in a region where typhoid existed. Every man was vaccinated and only one developed typhoid. The chief feature in acquired immunity is the presence in the blood and tissues of elements which can neutralize the toxic products of bacteria. These elements are "antitoxins" (see page 39). Microbic products are dead and cannot multiply as can living bacteria, hence the human organism is not overwhelmed unless the dose is too large, but the microbic products cause the development of antitoxin as certainly as do the living microbes. The above facts are of immense importance, for on these lines may be solved the problems of the prevention and treatment of microbic maladies.

**Orrhotherapy,** or **serum=therapy,** is an attempt to utilize therapeutically the germicidal properties of blood-serum. It is believed that when a person recovers from an infectious disease the alkaline blood-serum is saturated with protective material, particularly with antitoxin. If this belief is true, a proper deduction is, that blood-serum containing protective material should cure the disease if injected into a patient suffering from an attack. Some sera used therapeutically are antitoxic (antitoxin), that is, they do not kill bacteria, but merely neutralize the toxin. Others are bacteriolytic, destroying and dissolving bacteria. An antitoxic serum is made by injecting a horse with toxin. The horse-serum comes to contain antitoxin. Antibacterial serum is obtained by injecting an animal first with the dead and then with the living bacteria. The serum contains the bacteriolytic material. Instead of using the blood-serum itself, some observers have precipitated the supposed curative material from the serum, have dissolved this material, and have administered the solution in fixed amounts. Instead of using the serum of persons rendered immune by an attack of the disease, many physicians have employed the serum of animals rendered artificially immune by injections of attenuated cultures of the bacteria or injections of bacterial products. Some experimenters have even employed the serum of animals naturally immune to the disease. In some cases the serum is given hypodermatically, in some intravenously, in some by lumbar puncture, in some by intracerebral, and in others by intraneural, injection. Claims have been made that serums are efficient when given by the mouth or by the rectum. Paten, of Melbourne, claimed in 1906 that the oral administration of immune serum raises the opsonic index ("Med. Press and Circular," Jan. 31 and Feb. 7, 1906).

Latham ("Lancet," Feb. 15, 1908) and others claim that clinical and bacteriologic evidences are in favor of the view that serums are efficient when given by the mouth. If these views are proved to be true serum-therapy will receive an enormous impetus. Calmette has perfected an antivenomous serum (antivenene) for use after cobra bites. Pasteur has devised a method which will usu-

ally prevent hydrophobia (see page 306). That Murri, of Bologna, has apparently cured a case of hydrophobia seems proved (see page 307). Hosts of observers believe in the utility of diphtheria antitoxin and many are convinced of the value of tetanus antitoxin. The earlier in the disease the injection of an antitoxic serum is practised and the larger the dose, the more apt it is to prove curative. When the toxin has not yet combined with cells, antitoxin may keep it from doing so, and when it has recently combined and the combination is still unstable, antitoxin may cause dissociation of the combination. When the disease is well established and the cell combination of toxin is firm, antitoxin will, in all probability, fail to cure. If we decide to give serum in an acute infection, give it early and as advised by Ball ("Lancet," June 8, 1912).

A rapid effect will be obtained by mixing the serum with normal salt solution and throwing 100 c.c. of the mixture into a vein. Each day after this 50 to 100 c.c. are given subcutaneously.

Ball advises that we obtain the serum which is most nearly autogenous. The best is obtained from the blood of one who has recently recovered from the identical infection. The next best is obtained from a person who has been artificially immunized. Make a vaccine at once, and as soon as it is made, substitute it for the serum injections.

It is very important to remember that the water of the salt solution must have been recently distilled. Otherwise an intravenous injection of serum and salt solution will be followed by a chill in from one-half to two hours after the injection (Ball, in "Lancet," June 8, 1912). In order to make diphtheria antitoxin a horse is immunized to diphtheria toxin by injecting subcutaneously increasing doses of diphtheria toxin. It requires two or three months for the blood of the animal to acquire sufficient potency. The blood containing antitoxin is withdrawn by bleeding, the serum is separated from the clot, and its antitoxic potency is determined by complicated methods. We signify the degree of potency of a serum by saying that it is of so many "immunizing units," a unit being an arbitrary standard. The average dose for a child is 1000 units and for an adult 2000 units. (Tetanus Antitoxin is considered on page 208; Antivenene is considered on page 300.)

Anthrax in animals and human beings has been treated with success by Sclavo's serum (the serum of an actively immunized animal). The antimeningococcus serum of Flexner and Jobling seems to possess distinct power in the treatment of epidemic cerebrospinal meningitis. It is given by lumbar puncture. Cholera serum seems of no avail therapeutically. Shiga's antidysenteric serum is of value. Claims are made for plague serum. Inconclusive experiments have been made in the treatment of syphilis by the serum of dog's blood and by the blood-serum of men laboring under tertiary syphilis; in the treatment of pneumonia by the blood-serum of persons convalescent from pneumonia; and in the treatment of sufferers from septic diseases by antistreptococcic serum—blood-serum of horses rendered immune to virulent streptococci. The serum treatment of pneumonia is a failure. The real value of antistreptococcic serum is yet uncertain. Occasionally it seems to do great good; at other times it appears to produce no benefit whatever. Some observers claim remarkable results in erysipelas. In several cases of phlegmonous erysipelas and in 2 cases of malignant endocarditis I thought it was of benefit. Tavel, in an elaborate research ("Klinische-therapeutische Wochenschrift," Vienna, August, 1902), states that he obtained brilliant results in some cases, but no results in others. He does not undertake to explain this variability of action. He thinks the serum benefits staphylococcus as well as streptococcus infections. Antistreptococcic serum often fails completely. This is supposed to be due to the fact that there are many different families of streptococci. It was hoped that a polyvalent serum would prove efficient, but the hope is still

only a hope. It has been proved that some antistreptococcic sera have not as high an opsonic index for streptococci as has normal horse-serum, and "the opsonin content of an antistreptococcic serum seems to be the factor that gives the serum whatever" value it possesses (Jordan's "General Bacteriology"). Tavel and Moser, believing that scarlatina is a streptococcic malady, prepare serum by using cultures of streptococci obtained from a number of cases of scarlet fever. Van de Velde uses cultures of streptococci obtained from various streptococcic infections. According to Burkard antistreptococcic serum destroys neutrophiles in the blood. This destruction is not harmful if leukocytosis follows the injections, and it does follow them in all cases when the body is able to react to the serum ("Archiv. f. Gynäk.," lxxx, No. 3). Before removing a tongue or an upper jaw it is my custom to give antistreptococcic serum, and I believe that it lessens the tendency to toxemia and to septic bronchopneumonia. Malignant tumors (both sarcomata and carcinomata) have been treated with the blood-serum of dogs, which animals had been injected with fluid expressed from malignant growths (Richet and Hericourt). Von Leyden and Blumenthal obtain a serum by compression of a recent cancerous growth and treat human victims of cancer with it. They claim that the results are encouraging ("Deutsche medicinische Wochenschrift," Sept. 4, 1902). Many claims made for serum-therapy in surgical diseases are exaggerated, sensational, and unscientific. It does not seem possible to obtain an antitoxin for each bacterial malady, and the bacteria of most specific diseases are potent for harm for more reasons than because they form crystalloidal toxic matter. That there is truth in the method seems highly probable, but how much truth there is, is not yet definitely ascertained. It is our duty to study, experiment, and observe, and to reach a conclusion only after honest, careful, and thorough investigation. A little skepticism is as yet a safe rule.

**Anaphylaxis, or Untoward Effects of Serum Injections.**—Anaphylaxis is a term introduced by Richet in 1904 to designate a state of hypersusceptibility (congenital or acquired) "to a strange protein or antigen with a reaction body formed in the body of the organism undergoing immunity" (St. George T. Grinnan, in "Jour. Amer. Med. Assoc.," Jan. 20, 1912). It has been known for a considerable time that guinea-pigs which had been injected with antitoxin frequently died when injected with the serum again some time later; the curious fact is that the first dose does no harm, but the second dose, given after several days, produces the trouble. In man unpleasant or even dangerous effects may follow the injection of any serum. They occur in certain hypersensitive individuals. They may occur from a first dose, but are far more apt to arise from the second, the third, or some later injection. They are most apt to occur when there has been an interval of two or three weeks between injections. In some cases where death followed a first injection an enlarged thymus existed. In a recent case in Philadelphia this was proved by autopsy.

The symptoms may be trivial and not arise for several hours. The most common ones are joint pains, weakness, depression, dyspnea, urticaria or erythema, cough, itching, sneezing, edema of the face, and swelling of the tongue ("Progressive Medicine," Dec. 1, 1908).

The symptoms may be serious and arise in a few minutes. In such a condition any of the previously mentioned symptoms may exist, but the dyspnea is urgent, the face is often cyanosed, and collapse occurs. In some cases death occurs in a few minutes after an injection. When untoward results follow a first injection the condition is regarded as *hypersusceptibility to serum.* When it follows a later injection it is called *serum disease.* We fear fatality from an initial dose when there is hypersusceptibility. Serum disease is usually made manifest by minor symptoms developing from eight to thirteen

days after a first injection or almost at once after an injection given from fourteen days to four months after the first one. There is no way of knowing beforehand that a person is hypersensitive or that he is liable to serum disease except that asthma is ominous and makes us fear some untoward effect. The dyspnea in some cases may have been due to enlarged thymus. In using diphtheria antitoxin or tetanus antitoxin the serum should be given at close intervals and not at intervals of several days.

**Vaccine Therapy, or Treatment of Infections by Bacterial Vaccines** (*Bacterines*, as S. Solis Cohen calls them).[1]—The studies of Wright and Douglas upon opsonins led to the adoption of this plan of treating certain infections.

By the injection of an antitoxic serum we seek to directly neutralize toxic products. By the injection of the bacterial vaccines we seek to stimulate the body cells to produce antibodies and particularly opsonin. An injection of antitoxic serum has only a temporary effect. Injections of bacterial vaccine cause a much more enduring effect. After such an injection the opsonic index usually begins to rise in from twelve to twenty-four hours. Additional doses gain more pronounced response. The injections appear to be free from all danger. Bacterial vaccine consists of dead bacteria and their endotoxins made into emulsion in normal salt solution. Each individual has his own response to such an injection, but this response varies at different times. An antitoxic serum contains other antibodies besides antitoxin. Bacterial vaccine is made up with salt solution and is truly specific. A vaccine made up from a certain variety of organisms is valuable only in infections from that variety of organism. In some cases stock cultures are used, but it is better whenever possible to obtain the bacteria from the infected person and obtain our cultures from them (**autogenous vaccine**). In some cases, however, we cannot wait for the development of a culture and must then use stock vaccines. In erysipelas we cannot use autogenous vaccine. In a mixed infection it is sometimes uncertain which organism is the main factor in causing the trouble and danger, and yet that is the main factor against which the vaccine must be leveled. Until recently it was believed that the dose must be determined by the opsonic index. This plan is now seldom followed. Each cubic centimeter of Wright's stock vaccine contains 600,000,000 dead bacteria. The first dose is $\frac{1}{2}$ c.c. and the second dose is 1 c.c. Hartwell and Lee repeat the full dose every fourth or fifth day until the lesions are cleared up ("Publications of Mass. General Hosp.," Oct., 1908). Each injection is made in the subcutaneous tissue, the skin having been previously scrubbed with soap and water and washed with alcohol. In many cases there is a trivial reaction after injection. This reaction is not febrile, is of brief duration, and is manifested by headache, backache, and languor. It might well be asked, Why inject dead bacteria to stimulate resistance when live bacteria in the individual have failed to do it? The theory is that the bacteria causing the disease have died too quickly in the blood or, for some other reason, have failed to produce enough stimulation to result in the copious production of antibodies.

There is much testimony as to the value of this plan of treatment. The temperature of some cases of streptococcic infection may be rapidly lowered by vaccine treatment, pus formation may be lessened, and delirium be abolished. It is particularly serviceable in superficial infections from the Staphylococcus aureus (boils and carbuncles). In many cases pain and tenderness begin to abate a few hours after the first injection, a profuse discharge flows from the lesion if it is open, and gathers in the tissues if there is no opening. If the focus

[1] In this connection see particularly article by Roger J. Lee and article by H. F. Hartwell and Roger J. Lee in "Publications of Mass. General Hosp.," October, 1908; also article by Ball, in "Lancet," June 8, 1912. I have used these articles freely.

of infection is closed it should be incised, but Wright insists that antiseptics must not be used, as they destroy the activity of opsonins. The treatment is of little or no value in abscess, pyemia, septicemia, and mixed infections. It seems certain that in an overwhelming infection a vaccine can do no possible good. In such a condition it cannot possibly cause the patient's tissues to produce antibodies. We may lay it down as a rule that vaccines are particularly indicated for chronic and for local infections, and serums for general infections. In an acute infection give serum at once and autogenous vaccine as soon as it can be made (Ball's rule).

**Tuberculin.**—(See page 220.)

**Special Surgical Microbes.**—**Suppuration** (see page 132) is caused by microbes. Does it ever exist without them? The answer is, "Practically no." Injection of a sterile fluid containing dead organisms, or the injection of the sterile products of the growth of pyogenic cocci, will form a limited amount of pus. Injection of an irritant causes the formation of a thin fluid which may resemble pus, but is not pus. In surgery pus very seldom forms without the actual presence of living micro-organisms (see page 133), and the presence of pus is regarded as proving the presence of living micro-organisms.

**Pyogenic Bacteria.**—*Pus microbes*, or *pyogenic microbes*, are strongly proteolytic, that is, they possess the property of peptonizing albumin, and thus forming pus. The peptonizing action is brought about by bacterial products. Some believe that pus is not formed by a peptonizing action of the bacteria, but that the bacteria furnish a poison (leukolysin) which breaks up the leukocytes, and that the breaking up of leukocytes liberates an enzyme which dissolves albumin. The inflammation which surrounds an area of pyogenic infection is caused by the irritant products of bacterial action (toxalbumins, ammonia, etc.). In the presence of the pyogenic peptones the coagulation of inflammatory exudate is retarded or prevented. Bacteria which ordinarily cause suppuration may fail to cause it, producing instead a non-suppurative inflammation. Non-suppurating inflammation may arise if bacteria are present in small numbers or if the tissue resistance is at a high level, or if the virulence of the bacteria has been modified by adverse antecedent conditions. Bacteria which ordinarily do not cause suppuration may do so under certain conditions of increased bacterial virulence or lessened tissue resistance. The typhoid bacillus is at times pyogenic, but, as a rule, it is not pyogenic. The usual causes of suppuration are the following micro-organisms.

The term *Micrococcus pyogenes* (Fig. 15) includes the Staphylococcus aureus, the Staphylococcus albus, and the Staphylococcus citreus. These forms are deviations from one form and are not specifically different. The albus and citreus may be grown from the aureus, and they may remain white and yellow or may revert in part to the aureus form ("Atlas of Bacteriology," by Lehmann and Neumann). Some observers maintain that these forms vary greatly in virulence and hence are specifically different, but the varying virulence has been disputed, and it seems to have been proved that virulence may be lessened greatly even when the color does not change. Eighty per cent. of acute abscesses are due to staphylococci. Staphylococci are found also in osteomyelitis, in carbuncle, in boil, in acne, in pemphigus, in periostitis, in septicemia, and in pyemia, and in some cases of empyema and peritonitis. Some toxic products of staphylococci destroy leukocytes. All of the staphylococci are non-motile.

*Staphylococcus pyogenes aureus* (Plate 1, Figs. 1 and 15), is the golden-yellow coccus. When grown in the air it produces orange-yellow pigment. This is the most usual cause of abscesses (circumscribed suppurations). The Staphylococcus pyogenes aureus grows best in air, but can grow when air is ex-

1 2 3

1. Staphylococcus pyogenes aureus.
2. Staphylococcus pyogenes albus.
3. Bacillus tuberculosis on glycerin-agar.

(Warren's *Surgical Pathology*.)

cluded. As it can thus grow it is a facultative anaërobic parasite. It is widely distributed in nature, and is found in the soil, the dust of air, water, the alimentary canal, under the nails, on and in the superficial layers of skin, especially in the axillæ and perineum, in the mouth, the nasal cavities, the vagina, and human milk. It forms the characteristic color only when it grows in air (Plate 1, Fig. 1). It is killed in ten minutes by a moist temperature of 58° C. and is instantly killed by boiling water. Carbolic acid (1 : 40) and corrosive sublimate (1 : 2000) are quickly fatal to this coccus.

*Staphylococcus pyogenes albus* (Plate 1, Fig. 2), the white staphylococcus, acts like the aureus, but is usually more feeble in power. When this organism is found upon and in the skin it is called the *Staphylococcus epidermidis albus*, an organism which Welch proved to be the usual cause of stitch-abscesses.

*Staphylococcus pyogenes citreus*, the lemon-yellow coccus, is found occasionally in acute circumscribed suppurations, but less often than are the other two forms. Its pyogenic power is even weaker than that of the albus.

The *Staphylococcus cereus albus* and the *Staphylococcus cereus flavus* are found occasionally in acute abscesses, but these forms cannot be sharply differentiated from the Micrococcus pyogenes and the names should be abandoned.

*Staphylococcus flavescens* is occasionally found in abscesses. It is intermediate between the aureus and albus.

*Micrococcus pyogenes tenuis* rarely takes the form of a bunch of grapes. It is occasionally found in the pus of acute abscesses.

Fig. 15.—Micrococcus pyogenes aureus (× 1000) (Lehmann and Neumann).

Fig. 16.—Streptococcus pyogenes (× 700) (Lehmann and Neumann).

The *Micrococcus tetragenus* is thought to be the bacterium chiefly responsible for the suppuration of tuberculous pulmonary lesions.

*Streptococcus pyogenes* (Fig. 16).—This coccus, known as the chain coccus, grows best in air and can also grow when air is excluded. It is non-motile and does not bear spores. It is found in the healthy human body in the nasal cavities, urethra, mouth, vagina, and on the skin. It has been found in spreading inflammation and suppuration, erysipelas, pneumonia, otitis, puerperal fever, pyemia, septicemia, lymphangitis, some very acute abscesses, and some cases of meningitis, empyema, peritonitis, ulcerative endocarditis, pericarditis, osteomyelitis, diarrhea, and in certain sore throats. It varies very greatly in virulence and the intensity of its action is strongly influenced by the nature of the soil in which it is implanted. Streptococci are apt to cause serious local lesions, violent constitutional involvement, and frequently death. Not only do streptococci produce virulent toxins, but they also produce a non-toxic material called hemolysin, which dissolves red corpuscles. Some bacteria always get in the blood during the existence of a streptococcic infection. In a mild case those which enter the blood are soon killed. Even in a very severe case we may be unable to demonstrate them, but they are surely there. Woodhead tells us (Treves's "System of Surgery") that six organisms, each of which bears a separate name, are discussed under this designation. Three of these organ-

isms he places in one group, two in another, and says the sixth may be a separate species.

1st Group.—*Streptococcus pyogenes* (Fig. 16), found especially in spreading suppuration. Such suppurations spread because streptococci only feebly attract leukocytes and prevent the coagulation of exudate. Streptococci are also found in very acute abscesses. About 15 per cent. of acute abscesses contain streptococci. The Streptococcus pyogenes is easily killed by boiling, and can be destroyed by carbolic acid and corrosive sublimate. These organisms are normally present in the nasal passages, vagina, mouth, and urethra.

*Streptococcus pyogenes malignus*, an uncommon organism found in splenic abscess.

*Streptococcus septicus* has a strong tendency to break up into diplococci.

2d Group.—*Streptococcus of erysipelas* is found in the capillary lymph-spaces in erysipelas. Many bacteriologists believe it to be identical with the Streptococcus pyogenes. These bacteria tend particularly to gather in the lymph-spaces. They rarely produce pus and when they do it is usually watery. When ordinary thick pus forms there is a mixed infection with staphylococci.

*Streptococcus of Septicemia and Pyemia.*—Most observers maintain that it is identical with the Streptococcus pyogenes and the streptococcus of erysipelas.

3d Group.—*Streptococcus articulorum*, found in the false membrane of diphtheria (see the article by Woodhead in the "System of Surgery," by Sir Frederick Treves).

**Other Pyogenic Organisms.**—The various forms of colon bacillus, the typhoid bacillus, the Streptococcus intracellularis, the Micrococcus tetragenus, and the pneumococcus, are at times pyogenic. Pneumococci may produce arthritis (see page 638), peritonitis (see page 1030), cholecystitis, empyema, necrosis of bone, or wound infection. A case of wound infection due to pneumococci was reported by J. H. Beaty ("Northwestern Lancet," July 1, 1907). In many persons pneumococci exist in the mouth. A common form of colon bacillus is the Bacillus pyogenes fetidus: it is found in stinking peritoneal pus and in the pus of ischiorectal abscesses. The gonococcus is also pyogenic.

The Bacillus pyocyaneus may be the sole cause of a suppuration, but usually when it appears it constitutes a secondary infection in a suppurating area. It causes a blue or blue-green hue in pus and wound discharges.

It is normally found in water and exists in the mouth, intestine, and skin.

**Other Surgical Microbes.**—*Streptococcus of erysipelas* (Fehleisen's coccus), as stated before, is thought by many to be identical with the Streptococcus pyogenes. Their difference in action is believed by Sternberg to be due to difference in virulence induced by external conditions and by the state of the tissues of the host. The coccus of erysipelas is somewhat larger than the ordinary form of Streptococcus pyogenes. Infection takes place by a wound, often a very trivial wound of the skin or mucous membrane. The cocci multiply in the small lymph-channels. This coccus will cause puerperal fever in a woman in childbed when it gains access to any area in the genital tract from which absorption can occur. The streptococcus seldom causes suppuration in erysipelas; when it does so the pus is usually watery. Thick pus means mixed infection.

Fig. 17.—Micrococci gonorrhœæ, highly magnified, schematic (Lehmann and Neumann).

The *gonococcus*, or the *Micrococcus gonorrhœæ* (the bacillus of Neisser) (Fig. 18), is the diplococcus which causes gonorrhea. Neisser, in 1879, observed this bacillus in pus from gonorrheal ophthalmia and urethral gonorrhea. Bumm, in 1887, proved the causative influence of the gonococcus. He reproduced the disease in a healthy female urethra by inoculation with the twentieth genera-

tion in descent from a pure culture. These micrococci are in pairs, and each member of a pair is kidney shaped (Fig. 17). Gonococci grow best in air, but can grow when air is excluded. Diplococci are found often in the secretions of apparently healthy mucous membranes, and simulate very closely gonococci, but genuine gonococci are not so found. The gonococcus is a pure parasite and is not found outside of the organism except upon articles contaminated with gonorrheal discharge. In male gonorrhea the gonococci are in the urethra and prostate; in female gonorrhea they are in the urethra, glands of Bartholin, and cervix uteri. These cocci may cause gonorrheal conjunctivitis, lymphangitis, lymphadenitis, rhinitis, otitis, proctitis, endometritis, salpingitis, oöphoritis, cystitis, peritonitis, bursitis, thecitis, pleuritis, malignant endocarditis, arthritis, periostitis, abscess, and parotitis. In chronic urethral gonorrhea the gonococci may at times be absent from the discharge, returning when there has been sexual or alcoholic excess, traumatism, or contact with an irritant secretion. In such a case there could have been but a very few gonococci in the urethra before the irritation was applied, and the discharge was kept up, in part at least, by irritant toxins. If a part in such a condition is irritated, active multiplication begins and the cocci reappear in the discharge. Gonococci cannot be cultivated upon ordinary media, but grow best upon human blood or human blood-serum. In gonorrhea the organisms are found both within and outside of pus-cells and on mucous cells (Fig. 18). The gonococci infect a surface covered with cylindrical epithelium much more readily than a surface covered with pavement-epithelium. They pass into the submucous tissue, cause inflammation, and spread by way of the lymph-paths. It seems certain that the gonococcus is pyogenic, although mixed infection with other pyogenic organisms may exist in this disease. Their presence inside of pus-cells means phagocytosis. Gonococci stain easily by methylene-blue and are readily decolorized by Gram's method.

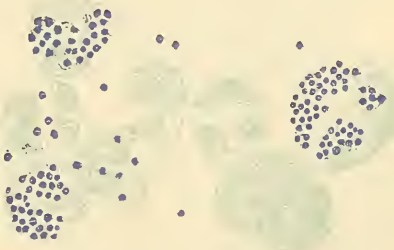


Fig. 18.—Gonococci from gonorrheal pus.

In noma *streptococci* are found. No specific organism has been isolated for traumatic spreading gangrene or hospital gangrene.

The *bacillus of tetanus* or the *Bacillus tetani* (Nicolaier's bacillus) (Fig. 19) was discovered by Nicolaier in 1884. In 1889 Kitasato obtained a pure culture. It is an obligate anaërobic organism. In recent cultures at least it ceases to grow in the presence of free oxygen. It grows *within* the tissues of the animal body. In a wound to which air has access the bacilli may lie so surrounded by fluid that air cannot reach the bacteria. Pyogenic or saprophytic bacteria may consume the air or the bacilli may lie in a laceration of the tissue the outlet of which is sealed by exudate or blood. The bacillus of tetanus is a facultative saprophyte, that is, under certain conditions it can grow in dead organic material. It is possible to develop by cultivation bacilli which will live in air, but such bacilli have lost their virulence.

The bacilli of tetanus are widely distributed. They are found particularly in hay, in the soil of gardens, in the dust of old buildings, in street dust and dirt, and in the sweepings of stables. The feces of healthy horses, cattle, and men may contain the bacilli. Tetanus develops after a wound and the bacilli remain in the wound and do not enter the blood. They furnish deadly toxins which are absorbed. The symptoms are due to intoxication, not to infection. The toxin of tetanus is alkaloidal, not albuminoidal. These bacilli stain by

Gram's method. Cultures are made in agar-agar punctures, the air being excluded, or on gelatin-containing glucose and in an atmosphere of hydrogen. These bacilli when placed under somewhat unfavorable conditions sporulate with great rapidity, and the spores are seen at the ends (Fig. 19). The spores are far more resistant than the adult bacilli, and it is difficult to kill them in a wound. A drug which is very fatal to tetanus bacilli is bromin.

Fig. 19.—Bacillus of tetanus, with spores.

The *Bacillus tuberculosis* (Koch's bacillus) (Fig. 20). This bacillus is the cause of all tuberculous processes. It was discovered and cultivated by Koch in 1882.

It is non-motile and requires oxygen in order to grow, but may obtain this from the body cells or fluids. It stains by Gram's method and by fuchsin. These bacilli are cultivated upon glycerin-agar or solid blood-serum (Plate 1, Fig. 3).

They are found in dust containing the dried sputum of victims of phthisis and dried discharges and secretions of tuberculous patients. This infected dusty air is influential in conveying infection (inhalation tuberculosis). Infection can also be conveyed by inoculation of bacilli (inoculation tuberculosis) and by eating the meat and drinking the milk of tuberculous animals (ingestion tuberculosis). Tuberculin is discussed on page 220.

Koch maintains that the human type of bacilli is almost altogether responsible for tuberculosis in human beings. Behring and many other observers dispute this statement, and assert that bovine bacilli are frequently responsible for tuberculosis in human beings. It seems certain that in children many cases of glandular tuberculosis and abdominal tuberculosis are due to bovine bacilli. The bacilli of cattle are not so long or thick and are straighter than human bacilli. It is not yet quite certain that the bovine bacilli are identical with human bacilli. They are at least very close blood relations.

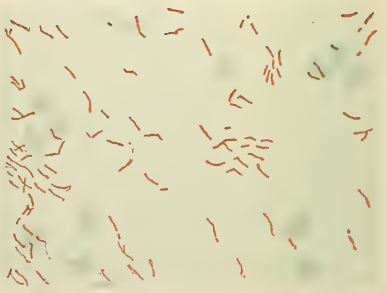


Fig. 20.—Tubercle bacilli in sputum (Ziegler).

*Bacillus anthracis*, or the *bacillus of anthrax* (Fig. 21), is the cause of malignant pustule, anthrax, or splenic fever. This bacillus was first observed by Pollender in 1849, and its causal influence was first strongly asserted by Davaine in 1863. Davaine's contention was proved by Koch in 1876. It is non-motile. Tissue containing it is stained by Gram's method. Cover-glass preparations are stained by a watery solution of an analine dye. It will grow without oxygen, but grows best in air. In the presence of air sporulation occurs, but it does not occur in the infected animal. It grows upon or in gelatin or agar. Only outside of the diseased body are spores found, and they exist in the hides and hair of infected animals and in stalls and pastures in which diseased animals were kept.

*Bacillus mallei*, or the *bacillus of glanders*, is the cause of glanders. It was

discovered by Löffler and Schütz in 1882. It is non-motile and grows best in air, growing with great difficulty when air is excluded. It grows well upon glycerin-agar and does not stain by Gram's method. It is never found except in the body of a diseased man or other animal. It is best cultivated in solid blood-serum. Under certain circumstances some few of the bacilli contain spores.

The *pneumococcus*, called also the *Diplococcus pneumoniæ*, *Fränkel's bacillus*, and the *Streptococcus lanceolatus*, was discovered by Sternberg in 1880. It is often found in the saliva of healthy individuals. It is not found outside of the

Fig. 21.—Bacillus anthracis (× 1000) (Lehmann and Neumann).

Fig. 22.—Bacillus of malignant edema (Lehmann and Neumann).

body. It varies greatly in virulence, but when virulent can establish inflammation and even suppuration, particularly of mucous and serous surfaces. It is especially apt to lodge and multiply in the lung, but it may lodge in a joint, in the brain membranes, in the peritoneum, or in other parts. It may cause croupous pneumonia, catarrhal pneumonia, pleuritis, meningitis, conjunctivitis, arthritis, peritonitis, periostitis, osteomyelitis, parotitis, salpingitis, empyema, cholecystitis, perinephric and other abscesses, nephritis, tonsillitis, mastoiditis, and septicemia. In any of these conditions it may appear in the blood. Pneumococci in the blood constitute pneumococcemia. In fact, it may appear in the blood when the lungs have not been diseased. Pneumococcic arthritis, peritonitis, cholecystitis, or empyema may arise without coexisting or antecedent pneumonia. The pneumococcus grows best in bouillon cultures, in blood-serum and in glycerin-agar. Sir Thomas Oliver ("Brit. Med. Jour.," April 30, 1910) points out the interesting fact that in pneumococcic meningitis, although symptoms are largely cerebral, the patient looks like one suffering from pneumonia.

Fig. 23.—Bacillus coli communis.

The *Bacillus coli communis*, called also the *Bacterium coli commune*, the *colon bacillus*, or the *bacillus of Escherich* (Fig. 23), was discovered in feces by Emmerich in 1885. Under ordinary conditions this is a putrefactive bacillus inhabiting the intestinal canal and feces invariably contain it. It is found in the mouth, nose, and vagina, on the skin, and under the nails. The bacillus is normally found in water, even in water regarded by the users as pure. It has already been stated that this ordinarily harmless micro-organism may, under certain conditions, acquire pathogenic power and enter the circulation. This bacterium grows best in air, but it can also grow when air is excluded. It is not

stained by Gram's method and has pyogenic power. It stains with anilin dyes and is decolorized by iodin solution. There are numerous forms of colon bacilli, and some of them are motile, some are amotile. This bacillus may be responsible for appendicitis, peritonitis, inflammation of the genito-urinary tract, pneumonia, inflammation of the intestine, leptomeningitis, perineal abscess, cholangitis, cholecystitis, myelitis, puerperal fever, wound infection, and septicemia. It is the cause of many abscesses about the intestine, and is responsible for many ischiorectal abscesses. From the pus of an appendiceal abscess we may perhaps obtain a pure culture of Escherich's bacillus, but usually find also streptococci or staphylococci, and sometimes pneumococci. Colon bacilli introduced into the system by tainted food may be responsible for epidemic pneumonia. A few years ago there was such an epidemic in Middlesbrough, England (Oliver, in "Brit. Med. Jour.," April 30, 1910).

Lehmann and Neumann point out that there are occasionally encountered "gaseous phlegmons and similar diseases of internal organs, in which are found the bacterium coli alone or usually in combination with other varieties, but without any anaërobes being present" ("Atlas and Principles of Bacteriology," vol. ii, edited by Geo. H. Weaver).

The *Bacillus œdematis maligni*, the *bacillus of malignant edema*, or the *vibrione septique* of Pasteur (Fig. 22), was discovered by Pasteur in 1875. This bacillus is found especially in stagnant water and certain varieties of soil and exists in putrefying material. It is sometimes motile, but is often amotile, and multiplies by spore formation. It is anaërobic and in its growth produces bubbles of gas. In the disease known as malignant edema there is usually a mixed infection with the bacilli of malignant edema and saprophytic organisms, and the latter also form considerable quantities of gas in the tissues. The bacilli of malignant edema may cause either spreading bloody edema containing gas-bubbles or spreading emphysematous gangrene. The bacilli enter the blood and produce septicemia. The bacillus is grown in the interior of a stab in gelatin agar-agar or solid blood-serum when the mouth of the stab has been sealed up.

The *Bacillus aërogenes capsulatus* of Welch was described by Welch and Nuttal in 1892. This bacillus is found sometimes in abscesses containing gas. It is causative of some cases of gangrenous cellulitis, which is a spreading gangrene with gas formation.

It has a capsule and very seldom forms spores. It stains by Gram's method and grows well upon blood-serum.

The *Bacterium typhi*, the *typhoid bacillus*, or *Eberth's bacillus*, was discovered by Eberth in 1880. It is sometimes found in water or soil contaminated by typhoid fecal matter. It never exists in the healthy human body (except in typhoid carriers). It causes typhoid fever and in this disease can be obtained and cultivated, particularly from the spleen and lymphatic glands and frequently from the blood. It has been found in the urine, kidney, bone-marrow, and bile. It is difficult to cultivate typhoid bacilli from feces because of the presence of multitudes of other bacteria. The bacillus of typhoid is motile, does not stain by Gram's method, and grows best in air, but can grow when air is excluded. It grows upon all the ordinary nutrient media. This bacillus is particularly apt to be confounded with the colon bacillus, and it is even possible that the former develops from the latter. Besides typhoid fever the typhoid bacillus may cause peritonitis, chronic osteomyelitis, epididymitis, orchitis, gangrene, cholecystitis, thrombosis, embolism, synovitis, arthritis, and pulmonary inflammation. If pneumonia is caused by the typhoid bacillus, there are the ordinary physical signs of pneumonia and there are no abdominal symptoms, but the appearance of the patient and the duration of the disease are suggestive of typhoid fever. It is interesting to note that relapse may occur (Oliver, in

"Brit. Med. Jour.," April 30, 1910). This bacillus, under certain conditions, is pyogenic. Typhoid bacilli are agglutinated and lose motion by contact with a 1 to 50 dilution of the blood-serum of a patient with tyhpoid fever or convalescent from typhoid fever (the *Widal reaction*).

**Putrefactive Bacteria.**—By putrefaction we mean the decomposition of albuminous matter with the production of materials possessed of a foul odor. The bacilli of putrefaction act upon dead tissue exposed to air and are most active when the supply of air is somewhat limited. The surgeon encounters these bacteria in areas of necrosis or in tissues previously destroyed by other microbes. In the latter case they cause a mixed infection. An instance of such a mixed infection is putrid pus. Some of the products of putrefactive bacteria are highly poisonous (ptomains). Absorption of a small amount of putrid toxin causes surgical fever and absorption of a large amount causes putrid intoxication.

The chief putrefactive bacteria are: The colon bacillus (when under normal conditions); the bacillus of malignant edema; the Proteus vulgaris; the Proteus mirabilis; the three forms of the Bacillus saprogenes; and the Proteus zenkeri.

We may mention, in conclusion, as of occasional surgical importance the bacillus of influenza, bacillus of diphtheria, bacillus of bubonic plague, bacillus of leprosy, bacillus of rhinoscleroma, bacillus of fetid ozena, bacillus of hemorrhagic septicemia, and the Bacillus lactis aërogenes, which is an unusual cause of peritonitis.

The *ray-fungus* is considered on page 309.

**Infections by Protozoa.**—Protozoa is the name given to the lowest forms of animal life. This group of organisms shows transitions from forms certainly animal toward forms certainly vegetable. The protozoa are minute unicellular organisms. The cell has a definite nucleus and is composed of protoplasm and a more or less dense cell wall. Many species have organs of locomotion (cilia or flagella). Most parasitic protozoa are sporozoa. The sporozoa multiply by spore formation, feed by osmosis, and, when freely formed, possess neither cilia nor flagella. Pébrine or silkworm disease is due to protozoa, so is trypanosomiasis. Protozoa are known to cause malaria (the plasmodium malariæ), tropical dysentery (the Entameba histolytica), and syphilis. Some observers maintain that they cause cancer, others assert that they are responsible for hydrophobia; and it is thought probable that they may produce measles, small-pox, yellow fever, scarlatina, and spotted fever.

*The Spirochæta Pallida* (the *Treponema Pallidum*).—A bacterial cause of syphilis has long been sought. Lustgarten thought he had found it in a bacillus resembling the tubercle bacillus, but this view has been disproved. Schaudinn and Hoffmann have described an organism constantly present in the initial lesion of syphilis and in secondary lesions and which they call the Spirochæta pallida ("Arbeiten aus dem Kaiserlichen gesundheitsamte," Berlin, April 10, Heft 2). The studies of Schaudinn and Hoffmann were confirmed by Metchnikoff ("Bull. Acad. de med. de Paris," May 16, 1905). These organisms are found in great numbers in the juice of syphilitic glands, in condylomata, and in chancres. They are motile, are without flagella, curve from three to twelve times, are stained with difficulty, and are transported by the lymph and blood (Blaschko, in "Berlin klin. Woch.," No. 11, 1907). The organism is thought by many to be a protozoön. The fact that the cell divides longitudinally and not transversely suggests that it is not a bacterium (Noguchi). Many observers place the organism with bacteria. The matter is still in doubt. The spirochetes were originally discovered by Bordet and Gengou in 1903. These observers found them in chancres, but thought their presence was inconstant. Schaudinn and Hoffmann show that it is constant. Very positive claims are now made as to the causal influence of the pale spirochete, and it seems prac-

tically certain that syphilis is a chronic spirillosis. The micro-organism is present in primary syphilis and all early secondary lesions and in congenital syphilis. It is not found in gummata. Noguchi obtained pure cultures of the spirochete, and inoculation of monkeys with these cultures caused the development of sores resembling chancres.

## II. ASEPSIS AND ANTISEPSIS

The effort in all operations is to secure and maintain scrupulous surgical cleanliness. What is known as the antiseptic method we owe to the splendid labors of Lord Lister, and the aseptic method is but a natural evolution of the antiseptic method. It is true that Agostino Bassi, over half a century ago, convinced that various maladies were due to parasites, treated wounds with a solution of corrosive sublimate. It is true that Oliver Wendell Holmes in 1843 insisted on the contagiousness of puerperal fever. It is also true that Semmelweiss in 1847 demonstrated the infectiousness of puerperal fever and the method of preventing it; that Jules Lemaire in 1863 published a treatise on carbolic acid and advocated the use of this drug in the treatment of wounds in order to destroy living germs; and that Bottini in 1866 employed carbolic acid in the treatment of putrid and suppurating wounds because he believed germs to be responsible for such conditions (Monti, on "Modern Pathology"). In spite of the above facts, Lister is the real father of asepsis and taught all nations how to prevent infection. Monti says: "But Lister, with that practical spirit which forms one of the best characteristics of English genius, from the scientific studies of Pasteur deduced the general laws of antisepsis and the rules for their methodical application to practical surgery." Lister called the attention of the profession to a new method of treating wounds, compound fractures, and abscesses in 1867.[1] The processes first employed were extremely complicated, but have been made in the last few years simple and easy of performance. Lister believed the chief danger to be from air. It is now believed that the chief danger is from actual contact of hands, instruments, dressings, or foreign bodies with a wound. Air carries but few micro-organisms unless it is filled with dust. Infection through air is most apt to occur if the air is dusty, and is more common after an aseptic than an antiseptic operation.

Of course, some bacteria from the air must settle in every wound, but the majority of the air fungi are harmless. Comparatively few reach the wound unless the air is dusty, and these few the tissues are usually able to destroy. Schimmelbusch made experiments in v. Bergmann's clinic when the students were present. He found that "the number of bacteria which settle upon the surface of a wound a square decimeter in extent, in the course of half an hour, is about 60 or 70," and thousands are usually required to produce infection.

There is no danger of infection being produced by the breath of spectators. Air which comes from the lungs is germ free, and even a large class will not infect the air by breathing, but will rather help to free it from bacteria, for the lungs are filters for air laden with micro-organisms. If a surgeon talks while he is operating, he may spray droplets of saliva into the wound *and thus produce infection.* In order to obviate this danger some surgeons wear masks of gauze before the nose and mouth. A conversational assistant is a danger, and a surgeon should direct his remarks away from the wound and not toward it. The surgeon and his assistant should wear caps to keep hair from falling in the wound. The clean shaven face is not a peril to the patient, the face "bearded like the pard" may be. A bearded man should wear a mask.

[1] "Lancet," March 16, 1867; "Brit. Med. Jour.," August 9, 1867.

The more simple the operative technic, the better and the more certain is it to be carefully carried out. Desault said, "The simplicity of an operation is the measure of its perfection." This is as true to-day as when the great French surgeon said it. The fewer assistants that are used the better, and no hands but the surgeons should enter the wound unless others are absolutely required.

In performing any surgical operation cutting is better than tearing by blunt dissection. The former method makes an incised wound, the latter a lacerated wound. In an incised wound there is a minimum amount of damage and there will be rapid repair. In a lacerated wound some necrosis occurs and there is great lowering of tissue resistance, hence a lacerated wound is much more apt to become infected than an incised wound.

Surgical cleanliness may be obtained by either the *aseptic* or the *antiseptic* method. In the *aseptic method*, heat, chemical germicides, or both are used to cleanse the instruments, the field of operation, and the hands of the surgeon and his assistants, the surface being freed from the chemical germicide by washing with boiled water or with saline solution. After the incision has been made no chemical germicide is used, the wound being simply sponged with gauze sterilized by heat; if irrigation is necessary, boiled water or normal salt solution (at 110° F.) is used, and the wound is dressed with gauze which has been rendered sterile by heat. The effort of the surgeon is simply to prevent the entrance of micro-organisms into the tissues. Some micro-organisms must enter, but the number will be so small that they will be destroyed by healthy tissues. The aseptic method should be used only in non-infected areas. If chemical germicides are not used in the wound, there will be a minimum amount of irritation, few cells will be destroyed, the amount of wound-fluid will be small, the surgeon can often dispense with drainage, and repair will be rapid. If a wound is to be closed without drainage, every point of bleeding must be ligated. Many wounds are closed by interrupted through-and-through sutures of silkworm-gut. Some wounds are closed in layers by catgut sutures. If a wound is closed in layers, muscle being against muscle, fascia against fascia, etc., the skin may be closed by interrupted sutures or by Halsted's subcuticular stitch (Fig. 116). If this stitch is employed, the skin staphylococcus does not obtain access to stitch-holes, and stitch-abscesses are not apt to arise. This suture may consist of catgut, silk, or, preferably, silver wire, this latter agent being capable of certain sterilization by heat and exercising a definite inhibitory action on micro-organisms. If a wound is closed without drainage, firm compression is applied over the wound to obliterate any cavity which may exist. Such a cavity is called a *dead space*. If a dead space is allowed to remain wound-fluid will gather, tissue resistance will be lowered, and the wound-fluid, the tissue, or both may become infected. Drainage must be used if the wound is very large, if its shape or structure prevents the obliteration of the cavity by pressure, if there is any doubt as to the perfect cleanliness of the part, if the patient is very fat (for in such individuals fat necrosis predisposes to sepsis and to fat embolism), or if the skin is so thin that we fear pressure will produce sloughing ("A Manual of Surgical Treatment," by Cheyne and Burghard). In some regions of the body wounds are sealed with collodion or iodoform-collodion. If irrigation is not practised and the wound is dressed with dry sterile gauze, the procedure is said to be by the "*dry*" *aseptic method*. In the *antiseptic method* the same preparations are made for the operation as in the aseptic method, but during the operation gauze sponges impregnated with a chemical germicide are used, and the wound is dressed with gauze containing corrosive sublimate or some other chemical germicide. If the wound is not flushed with a chemical germicide, and is dressed with dry antiseptic gauze, the operation is said to be by the "*dry*" *antiseptic method*. The anti-

septic method is preferred in infected areas. Dry dressings are usually preferable to moist dressings in treating aseptic wounds, because they are more absorbent and do not act as poultices, and dry dressings may be used, even when the wound has been flushed. Some surgeons question the value of antiseptic irrigation in a septic wound, but I believe it removes many bacteria and much poisonous matter and also antidotes toxic material. In suppurating areas it is often best to use moist dressings in the form of antiseptic fomentations. Year by year the aseptic method becomes more popular. Surgeons have learned that the most important factor in asepsis is mechanical cleansing by means of soap and water. The chemical germicide plays a secondary rather than a vital part. By mechanical cleansing of the skin great numbers of micro-organisms are removed along with dirt, grease, and epithelium. Many bacteria remain, but vast hordes are washed away, and the danger of infection is greatly lessened by thus diminishing the number of bacteria. If a chemical germicide is used without preliminary mechanical cleansing it is useless, because it cannot destroy bacteria in the epithelium and in masses of oily matter. After mechanical cleansing the germicide is active in destroying the comparatively few bacteria which are naked on the surface. In many regions a strong chemical germicide must not be used (in the abdomen, in the brain, in joints, in the pleural sac, and in the bladder), and in other regions (mucous surfaces and fatty tissue) it is productive of harm rather than good.

**Preparation for an Operation.**—If the operation is to be performed in a hospital there is, of course, an operating room always ready. If it is to be done in a private house, much careful preparation is desirable. The operating room should be warm, but not too warm. The desirable temperature is 78° to 80° F. Over 80° F. is too warm, it causes vascular relaxation in the patient, and makes the surgeon perspire and wear out. The patient is kept warm by certain special methods. He may be placed on a table heated by hot water or electricity or he may be surrounded by hot-water bags. Any large raw surface is kept covered as far as possible with pads of gauze wrung out of hot salt solution and frequently changed. Protruding intestines are treated in the same way. Every effort is made to avoid soaking the patient's skin with fluids, because as they cool they will chill him. A room in which an operation is to be performed should be well lighted and well ventilated. The northern light is the best. It is advantageous to have an open grate in the room, for then a wood fire can be quickly made to take a chill off the air and ventilation is improved. The morning before the operation the furniture should be removed, the carpet taken up, and the curtains and hangings taken down. If the ceiling and walls are papered, they must be thoroughly brushed. If they are painted, they must be washed with soap and water. Dust is thus removed and the danger of dust falling into the wound is averted. The floor is scrubbed with soap and water. The windows should be opened for many hours to thoroughly dry and freshen the room. On the morning of the operation the windows are closed and newspapers are tacked up so as to cover the lower half of each window. Plenty of light is admitted and the curiosity of neighbors across the street cannot be satisfied. The patient's bed is brought into the room and placed in a position where there will be plenty of light for future dressings, and where the surgeon will have access from either side. In order that there may be access from each side the bed must not be in a corner or against the wall. Never use a big broad bed; use a narrow bed. Never have a feather bed, but insist on Treves's advice being followed, and employ a metal bed with a wire netting and hair mattress.

A piece of carpet or rug is spread upon a portion of the floor and the table is set upon it. The table should be so placed that there will be a good light on the field of operation. There are several tables which are very satisfac-

tory. The best for a private house operation is Lilienthal's (Figs. 24 and 25). This table can be folded into a small compass, can be carried in a case with a handle, and is comparatively light and easily transportable. It can be

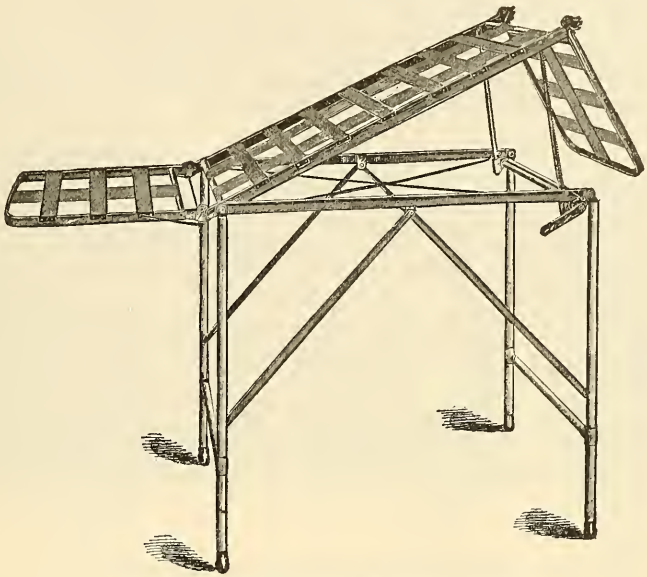


Fig. 24.—Lilienthal's portable operating table.

rapidly set up, is firm, and it enables the surgeon to obtain the Trendelenburg position at any moment. A kitchen table does very well. If a kitchen table is used and the abdomen is to be opened a frame should be at hand which,

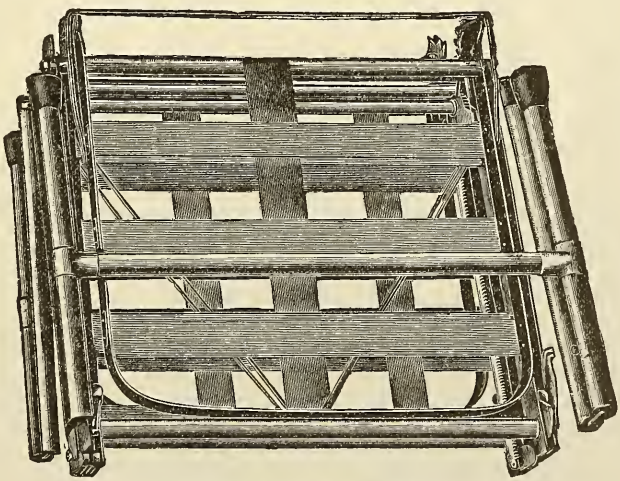


Fig. 25.—Lilienthal's portable operating table, folded.

when slipped under the patient, enables the surgeon to obtain the Trendelenburg position. Dr. Joseph Price was accustomed to use two trestles and a board like an ironing board. In hospital work I use Boldt's table (Figs. 26

and 27). On the table or board is placed a folded comfortable or several folded blankets. A rolled blanket is placed under the hollow of the back to

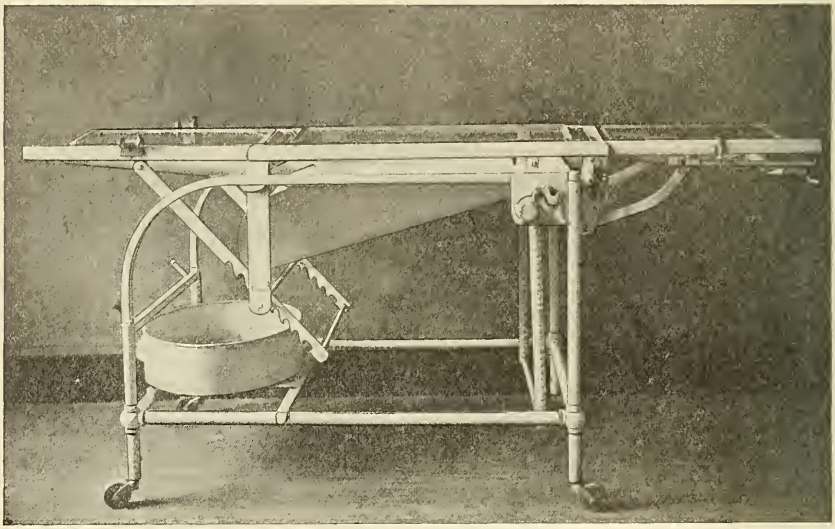


Fig. 26.—Boldt's operating table.

prevent strain of the sacro-iliac joints and postoperative backache. Kelly's pad to catch fluids is laid upon the blankets and is so placed that fluid used in

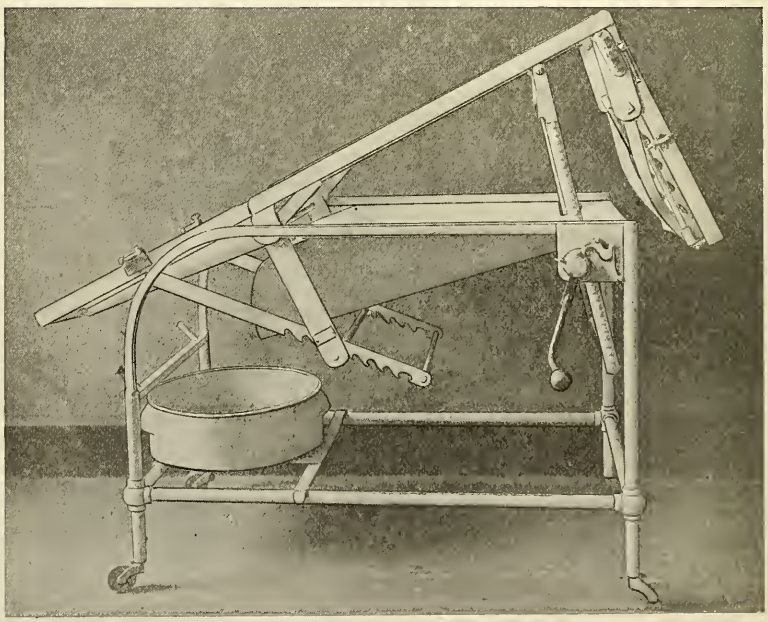


Fig. 27.—Boldt's operating table.

irrigation will flow into it and will be conducted by it to a suitable receptacle.

Around the operating table at proper distances are arranged a table for instruments, ligatures and sutures, a table for dressings, a table for pads, packs, gauze sponges, and a basin of bichlorid, and a table for soap and a basin of water. Ordinary wooden tables may be used if they are covered with towels wet in corrosive sublimate solution. In a hospital special tables are used. They are of iron with glass tops. Ordinary basins may be used, but enameled or glass basins in stands (Figs. 28 and 29) are the most satisfactory. A couple of buckets should be placed on the floor near at hand. Enameled buckets are the best ones to use. The nurses and assistants should have ready the ether cone, wrapped in a clean towel, sterile sheets, sterile gowns, sterile towels, sterile gauze for sponges and dressings, trays

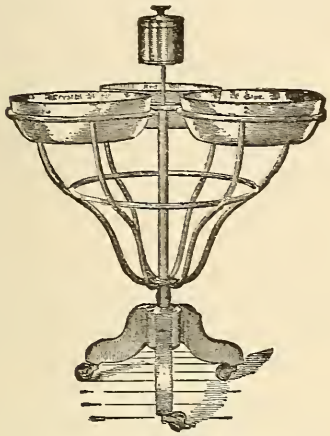


Fig. 28.—Revolving wash-stand.

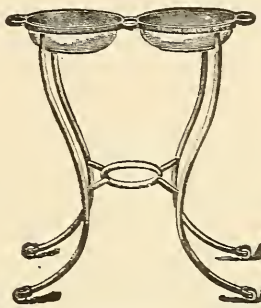


Fig. 29.—Plain double wash-stand.

for instruments (Figs. 30 and 31), iodoform gauze, catgut, silk, silkworm-gut, hot normal salt solution, etc., according to the nature of the operation. The surgeon should pick out the instruments required. The anesthetist should lay out a mouth-gag, tongue-forceps, hypodermatic syringe *in working order*, ether or chloroform, brandy, tablets of strychnin and also of atropin, and a cylinder of oxygen.

Most surgeons have the operation field sterilized the day before the operation, except in an emergency case. For several years I have been doing it in

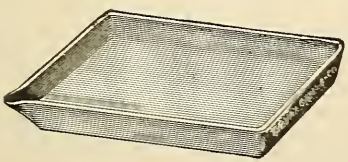


Fig. 30.—Porcelain surgical tray.

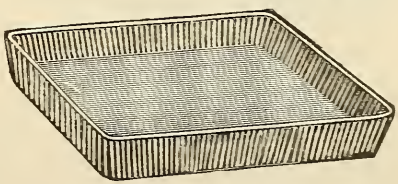


Fig. 31.—Glass surgical tray.

most cases after the patient has been anesthetized, and find this plan more comfortable for the patient, less troublesome, and equally effective.

When the time for the operation arrives, the surgeon and his assistants remove their clothing and put on duck trousers and thin, short-sleeved shirts of white muslin. After sterilizing the hands and forearms they envelop themselves in aseptic or antiseptic sheets or gowns, to protect the patient and themselves. The gowns should have sleeves long enough to cover the forearms and wrists. Sterile muslin caps should always be worn. The caps prevent hair, dandruff, and sweat from falling into the wound. Many operators wear over the mouth and nose a respirator or piece of gauze in order to prevent saliva or mucus being projected into the wound while the surgeon talks.

**Danger from the Hands.**—It is a difficult or impossible matter to absolutely sterilize the hands, but it is fortunate, as Mikulicz and Flügge say, that most of the bacteria of the skin are harmless. The Staphylococcus epidermidis albus, however, is constantly present in the epidermis. The hands of some persons are more easily sterilized than those of others. For instance, a hairy, creased hand is more difficult to sterilize than a smooth and almost hairless one; a hand grossly neglected, than one reasonably clean. Germs abound in the epidermis, in the fissures and creases, under and around the nails, on hairs, and in ducts of glands. The surface of the hands may be thoroughly sterile at the beginning of an operation and become infected later, because germs in gland ducts are forced to the surface. Hence, in a prolonged operation the surgeon, if he does not wear gloves, should from time to time stop operating and wash his hands, first in alcohol and then in corrosive sublimate solution (Leonard Freeman).

In view of the difficulty of cleansing the hands, every student must be taught how to do it, and he must become impressed with the fact that the surgical hand is to be regarded as reaching to the elbow. The more the fingers enter a wound, the greater is the danger of infection of the wound. The surgeon uses retractors and forceps whenever possible, but in most cases his fingers must at times enter the wound. The fingers of no other person should enter unless absolutely necessary. The basis of all plans of sterilization and the most important part of any plan is mechanical cleansing by scrubbing with soap and water. By this means a quantity of loose epidermis is removed and with it great numbers of bacteria.

**Mechanical Cleansing of the Hands and Forearms.**—The hands and forearms may be sterilized in several ways. Any method is preceded by mechanical cleansing, which is carried out as follows: Scrub for five minutes with soap and hot running water, giving special attention to the nails and creases in the skin. The water should be as hot as can be borne with comfort, as hot water stimulates the sweat glands and the flow of sweat washes out the ducts. If the ducts are washed out before the operation by copious sweating, during the operation the secretion will be slight. The brush is rubbed both in the long axis of the extremity and transversely. The creases on the back of the hands and fingers will be partially opened by flexing the fingers, and transverse scrubbing will clean the furrows. The furrows on the palmar surface will be opened by extending the fingers, and will be best cleaned by transverse scrubbing (George Ben Johnston). An excellent soap is ethereal soap, which is a solution of castile soap in ether. Castile soap can be used. I am accustomed to use green soap. Some surgeons prefer to use green soap in the form of a tincture. Synol soap has advocates. There is no particular advantage in using soap containing a germicide, as such soap is practically without germicidal power. The brush employed should be kept in a 1 : 1000 solution of corrosive sublimate or should have been recently sterilized with steam and kept in a sterile glass box (Fig. 32). The nails are cut short, are cleansed with an orange-wood stick, which does not scratch them, and the hands are again scrubbed. Very prolonged or very rough scrubbing, especially with harsh agents like marble dust or sand, is actually harmful, as it tends to crack the hands and make them rough and it extensively loosens epidermis which may drop into the wound. Epidermis may contain bacteria within it and may infect the wound.

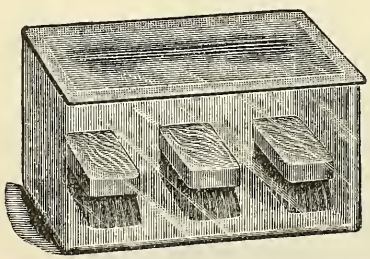


Fig. 32.—Glass brush-box with cover.

**Sterilization of the Hands and Forearms.**—After mechanical cleansing a germicide is employed to render the parts sterile. Whatever method is adopted it is desirable that it shall not unduly irritate the skin. An occasional operator may use without injury tolerably strong chemicals, but the busy hospital surgeon, who operates perhaps several times or many times a day, cannot use them. Any method which inflames, cracks, or roughens the skin makes future sterilization difficult or impossible, hence such a method is undesirable. Four methods are described here:

*Fürbringer's Method.*—After washing off the soap in sterile water the hands are dipped in 95 per cent. alcohol and held there for two or three minutes while the forearms, hands, fingers, and nails are being rubbed with alcohol. Alcohol removes the soap which has entered into follicles and creases, removes desquamated epithelium, enters under and about the nails, is germicidal, and favors the diffusion of the corrosive sublimate under the nails and into the follicles, when the hands are placed later in the mercurial solution. Alcohol also hardens epithelium and keeps it from desquamating into the wound. After using the alcohol the hands are then dipped in a hot solution of corrosive sublimate (1 : 1000), and with the forearms are scrubbed for at least a minute, the nails receiving especial care.

*The Welch-Kelly Method.*—After the hands and forearms have been cleansed mechanically and have been rinsed in sterile water they are immersed for two minutes in a warm solution of permanganate of potassium (a saturated solution in distilled water). This solution causes the cutaneous surface to assume a very dark brown color. The hands and forearms are then immersed in a warm saturated solution of oxalic acid and are held there until decolorized. They are then well washed in sterile water, are next immersed for two minutes in a 1: 500 solution of corrosive sublimate, and finally are rinsed in sterile water and dried on a sterile towel. The solutions for use in the above method should be contained in jars of the shape of a druggist's percolator, so that both the hands and forearms can be immersed at the same time. In this method the permanganate of potash is merely an oxidizer and the oxalic acid is the active germicide. Some persons find that the skin tolerates the plan very well, others, among whom is the author, find the oxalic acid decidedly irritant when used several times in a day.

*The Weir-Stimson Method.*—This method was suggested by Mr. Rauschenberg, the pharmacist of the New York Hospital, and it was applied practically by Doctors Weir and Stimson. The process is as follows: The hands should be cleansed mechanically as previously directed. Place about a tablespoonful of chlorinated lime in the palm of the hand, place upon the lime a piece of crystalline carbonate of soda (washing soda) 1 inch square and ½ inch thick, add a little water, and rub the creamy mixture over the arms and hands until the rough granules of sodium carbonate are no longer felt. This requires from three to five minutes. At first there is a sensation of heat usually followed by a sensation of coolness. Place the paste under and around the nails by means of a bit of sterile orange wood. Wash the arms and hands in hot sterile water.[1] Remove the odor of chlorin by washing the hands and arms in sterile ammonia water of a strength of from ½ to 1 per cent. (McBurney, Collins, and Oastler, in "International Text-Book of Surgery"). The combination of carbonate of sodium and chlorinated lime is said to set free nascent chlorin, a most efficient germicide. I used this method for several years in the clinic of the Jefferson Medical College Hospital and found it efficient. When employed several times a day it may prove decidedly irritant. It is important that crystalline washing-soda be employed. If the bicarbonate is used, nascent chlorin will not be produced, but hydrochloric acid gas will

[1] "Medical Record," April 3, 1897.

be formed, and the latter gas irritates the skin and is not a satisfactory germicide.

*The Sublimate-alcohol Method.*—This is the method I personally prefer. It is rapid, efficient, and reasonably non-irritant. It is as follows: Cleanse the hands with soap and water as previously directed. Use 95 per cent. alcohol as in Fürbringer's method (see p. 63). Dip the hands in 70 per cent. alcohol containing 1 part to 1000 of corrosive sublimate, and rub the hands, forearms, and nails with a piece of sterile gauze wet with this fluid for three minutes. Rinse these parts in the fluid and then rinse in sterile water.

**The Use of Gloves.**—Most surgeons are so impressed with the impossibility of sterilizing the hands that they wear gloves in operations. Over sixty years ago, at King's College, Sir Thomas Watson in a lecture on puerperal fever suggested that obstetricians wear gloves. He said: "In these days of ready invention a glove, I think, might be devised which should be impervious to fluids, and yet so thin and pliant as not to interfere materially with the delicate sense of touch required in these manipulations. One such glove, if such shall ever be fabricated and adopted, might well be sacrificed to the safety of the mother in every labor" ("Watson's Lectures on Physic").

Fig. 33.—Showing rubber glove applied.

Professor Halsted was a pioneer. He began to use rubber gloves in 1889. Some surgeons used cotton and others silk gloves, but it has been proved that cotton and silk are not impervious to micro-organisms, and that rubber is. The thin, seamless rubber gloves which are now made are very satisfactory. They are sterilized by boiling, are then dried, and are wrapped in a sterile towel. In order to insert the hand in them the hand should be dried, the interior of the glove should be dusted with sterile starch or talc powder, and then the nurse should fold forward the wrist part and hold the glove open while the surgeon inserts his fingers into the proper compartments and pushes the hand in. The custom of filling the glove with sterile fluid and then inserting the hand is troublesome and objectionable, because the fingers soon become sodden like those of a washwoman, the sense of touch is impaired, considerable discomfort is occasioned, and the skin is apt to crack.

If, during an operation, a glove becomes infected, a clean one can be substituted for it. Gloves somewhat impair the sense of touch, but a surgeon soon learns to work with them. If they are to be used, the hands should be sterilized just as carefully as when they are not to be used, because, during the operation, the gloves may tear or be punctured by a needle. I always wear gloves, but that it is absolutely necessary to wear gloves in all cases has not been

proved. Their use does contribute to success in brain operations, abdominal operations, and joint operations. They are of great value in military surgery, for the military surgeon may not have time to prepare his hands, and sterile gloves can be always kept ready prepared.

When a surgeon is obliged to place his fingers in an area of virulent infection he may be poisoned. Gloves will save him from this danger. Again, a surgeon should try to avoid bringing his hands unnecessarily in contact with putrid or purulent matter. Though it may not poison him, it grossly infects the surface, renders subsequent cleansing difficult, and endangers other patients. Gloves will prevent this danger. A surgeon should wear gloves if he is making an examination or performing an operation which is sure to infect the bare hands, and he should wear gloves in an operation if in a previous operation his hands were infected. A surgeon whose hands are very hairy or sweat much will contribute to the patient's safety by wearing gloves.

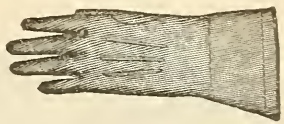


Fig. 34.—Half-long rubber glove.

Gloves should be worn if the surgeon has a wound or sore upon his hand or chapped hands. When using gloves in a prolonged operation dip the covered hands now and then in corrosive sublimate solution, because the glove may have been punctured or dust may have settled upon it from the air.

Gloves make the hands sweat, and if one should be punctured considerable sweat may emerge from the puncture and enter the wound, and sweat contains bacteria. The entry of any considerable amount of sweat is more dangerous to the patient than are well cleaned naked hands, hence gloves may actually favor the infection they are meant to prevent. When they are used the surgeon must take scrupulous care not to puncture them with a needle, clip them with forceps, or tear them with a ligature or suture.

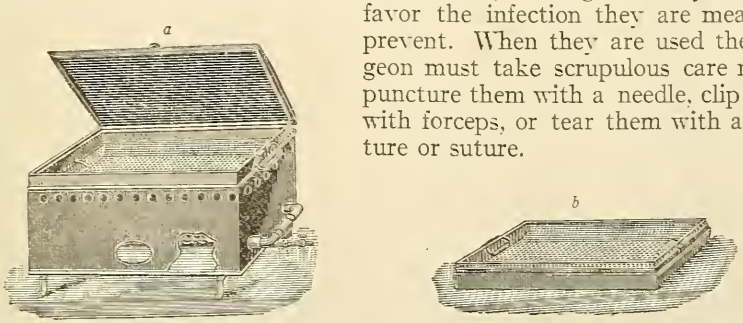



Fig. 35.—*a*, Schimmelbusch's gas-heated apparatus for sterilizing instruments: *b*, wire basket.

The closer they fit the less the danger of puncture, and one should know accurately what size he requires to fit closely and smoothly without being so tight as to make the fingers numb.

*Preparation of Gloves.*—Wash with soap and water containing a little ammonia, rinse in sterile water, boil for thirty minutes in a 1 per cent. solution of carbonate of soda. Dry the glove and wrap in a dry sterile towel and keep until it is needed. A glove should stand about twenty boilings. The surgeon should carry a number of pairs of prepared gloves in his bag, for the use of himself and assistants in private house operations.

**Metal instruments** are disinfected by subjecting them to the action of steam in a special sterilizer or, better, by boiling them for fifteen minutes in a 1 per cent. solution of carbonate of sodium. They are wrapped into a bundle by means of a towel or piece of gauze and are dropped into the solution. The blades of knives should first be wrapped in cotton to prevent scratching and dulling. After boiling, the instruments should be rinsed in hot sterile water or in a 5 per cent. solution of carbolic acid and be kept until needed in pans

of sterile water. The carbonate of sodium prevents rusting. In a clinic the boiling is carried out in a Schimmelbusch sterilizer (Fig. 35). In a private house it can be done in a sterilizer such as that shown in Fig. 36, or in a pan, a kettle, or a wash-boiler. A sterilizer with a tray is better than an ordinary pan or kettle, because, when the latter is used, the metal instruments lie in the bottom of the vessel, where the heat is very great and the temper may be impaired. Boiling, unfortunately, destroys to some extent the keenness of cutting instruments, the ebullition throwing them about. After the completion of the operation the instruments should be scrubbed with soap and water, boiled in soda solution, dried, and placed in a closet with glass shelves so that they will be protected from moisture and dust. Instruments can be partially disinfected by keeping them for thirty minutes in a 5 per cent. solution of carbolic acid or, better, in a 2 per cent. solution of formalin. Instruments with handles of wood, bone, ivory, or tortoise shell must not be boiled. Such instruments should not be used. If such instruments are used, they can be partially disinfected by the use of carbolic acid or formalin. Metal instruments, whenever possible, should consist of one smooth piece. Grooves and letters are objectionable, as dirt gathers in such depressions.

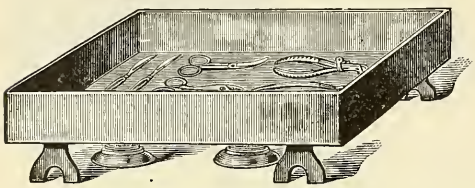


Fig. 36.—Portable sterilizer.

**Preparation of the Patient.**—Whenever possible give the patient some days' rest in bed before a severe operation. This is not possible in an emergency. It is seldom desirable in the case of a highly nervous and excitable woman. Such a patient is sleepless and frightened and loses ground by delay. In most cases this preliminary rest is advisable. It is particularly desirable in a strong, active working man suddenly translated from labor to bed. We wish to prepare him to meet operative shock. During the wait the patient is apt to adjust himself to his surroundings, he becomes accustomed to diminished activity and to sick-room routine, forms an acquaintance with his nurses and physicians, and, as a rule, becomes less nervous and more calmly confident of the result. He also learns to use the bed-pan and to micturate while recumbent. A patient while waiting is to have a general bath several times. Some weak and emaciated patients are treated and built up for weeks before the operation is attempted. During this preliminary rest the surgeon should study the disease, and study the individual in order to learn his tendencies, peculiarities, etc. The condition of the lungs, the heart, the blood, and the kidneys should be accurately determined. The amount of urine passed in twenty-four hours should be ascertained, and the percentage of urea should be estimated from a sample of the twenty-four-hour urine. The urine is carefully examined for sugar, albumin, casts, acetone, diacetic acid, indican, etc. By the above examinations the surgeon may be able to anticipate and provide against certain calamities. Sometimes such a study leads us to postpone or abandon an operation. Furthermore, such a study gives the information which is necessary in order to intelligently select the proper anesthetic. The presence in the urine of acetone and diacetic acid forbids any but an emergency operation. Sugar or granular and fatty casts in the urine, or a considerable quantity of albumin, make us hesitate to operate.[1] A hemoglobin percentage of under 50 makes us seek to avoid operation in most cases not associated with bleeding. The anesthetist should, during this preliminary period, examine the heart, pulse, and blood-pressure, so as to know the natural

[1] The question of operation on diabetics is discussed on p. 69.

character of each when the patient is free from excitement. Without this preliminary knowledge he cannot accurately appreciate or intelligently interpret some changes induced by the anesthetic. Constipation must be amended by mild laxatives or enemas, and all fermented matter should be removed from the alimentary canal. Constipation increases the probability of wound infection and greatly impairs the comfort of the patient. As previously shown, the putrefactive bacteria in the intestinal canal, which are usually harmless and are what Adami calls "potential parasites," may escape into the tissues. The retention of fermented matter causes catarrhal inflammation and bacteria escape more easily. If they escape they may lead to damage in the wound, and even if wound infection from within does not occur, constipation lessens vital resistance and increases the liability to wound infection from without. Purgatives must not be violent, as anything which greatly depresses a person lessens vital resistance, and powerful purgatives are powerful depressants. The diet should be bland and nutritious, but not bulky. The night before the operation give a saline cathartic, and the morning of the operation employ an enema. Not only do we empty the bowel to lessen the liability to wound infection, but we wish the rectum empty at the time of operation for another reason. It is desirable that the rectum be empty because in shock the absorbing power of the stomach is greatly diminished or is even abolished for the time, and it may be necessary to utilize the absorbing power of the rectum to take up stimulants given by enema. When a patient is under the influence of an anesthetic or when he is profoundly shocked, of course no attempt is made to give stimulants by the mouth. Whenever possible give a general warm bath the day before the operation. It is a common custom the evening before the operation to shave the region if hairy, scrub the entire field of operation, as well as the adjoining regions, with soap and water; wash with alcohol; scrub with hot corrosive sublimate solution (1:1000); apply a layer of moist corrosive sublimate gauze, and place over this dry antiseptic gauze, a rubber dam, and a bandage. Many surgeons apply a poultice of green soap for many hours before applying a chemical germicide, in order to separate masses of epithelium and with them many germs. This method is particularly useful in cleansing the scalp. On removing the dressings to perform the operation, the part is scrubbed with soap and water, washed with sterile water, then with alcohol, and then with corrosive sublimate solution. I have become convinced that the teachers in Johns Hopkins Hospital are right, and that cleansing the day before operation is not necessary except for a brain case. Neither is it desirable, as it often gives the patient a restless night. It is my custom to have a hairy region shaved the day before operation. In all cases the field of operation is disinfected the morning of the operation. Disinfection is again practised when the patient is on the operating table, *after* anesthetization. In emergency cases disinfection can only be practised just previous to the operation. When the field of operation has been prepared, surround it with dry sterile sheets and towels. In a head operation I stitch the towels to the skin to keep them in place and at the termination of the operation remove these stitches. Murphy prevents infection from the cutaneous surface by spreading a specially prepared sterile solution of rubber over the sterilized operation area. The rubber is dissolved in acetone. The incisions are made through the artificial skin of rubber and the rubber is removed when the surgeon is ready to introduce the sutures. Thus infection of the wound with contaminated secretion of the skin glands is prevented. If iodin has been used upon the abdomen, the surgeon must be scrupulously careful not to bring intestine in contact with the skin. If iodin comes in contact with intestine, it irritates the gut and becomes responsible for subsequent adhesions.

If disinfecting an emergency case in which a wound exists, tincture of

iodin, unless well diluted, should not be put in the wound. Hindenburg had a case in which local gangrene followed such an application ("Münchener medizinische Wochen.," July 5, 1911). It is not probable that the very dilute alcoholic solution of iodin used by many to prepare the skin would endanger a wound.

Of recent years iodin has been largely used and warmly praised as a disinfectant for the skin. It was introduced by Grossich in 1908. If iodin is to be used the skin may be scrubbed and shaved the night before, but must *never be scrubbed* within several hours of the impending operation. Were it done, it would swell the surface epithelium and keep the iodin from entering into the skin. Two hours before operation apply iodin, and apply it again when the patient is on the table. In an emergency case the skin is *dry shaved*, the field of operation is painted with a 2 per cent. alcoholic solution of iodin (which is allowed to dry and is not wiped away), and covered with sterile towels or gauze. When the patient is placed upon the table the field is again painted with iodin. This method is of great value in emergency cases, especially in out-patient and accident work. I have abandoned it in the axilla and perineum because the constant moisture of those regions makes iodin inefficient. The method is less efficient in summer than in winter, because in summer the skin is apt to be wet with sweat.

During an operation the patient must be carefully protected from cold by wrapping him in blankets, and often by having him wear specially prepared drawers with feet. After the completion of an operation and the application of the dressings the patient is returned to his room or the ward, care being taken to protect him from cold or drafts.

**Disinfection of Mucous Membranes.**—It is impossible to thoroughly disinfect mucous membranes. We must not scrub forcibly and we must not use powerful germicides, because they are irritant and also because they may be absorbed. The best that can be done in the *vagina* is to rub lightly, when possible, with a bit of moist absorbent cotton and irrigate with a solution of boric acid or with normal salt solution. Another method is to sponge the vagina with creolin and ethereal soap (1 and 16) and irrigate with hot saline fluid or boric acid.

The *rectum* is prepared by washing out all retained feces by the use of copious high injections and by irrigating with salt solution or boric acid.

The *mouth* is prepared by having snags of teeth and tartar removed and decayed teeth removed or plugged. For several days before the operation scrub the teeth twice a day with a soft brush and castile soap; and every three hours, when the patient is awake, rinse the mouth with peroxid of hydrogen and spray the nares and nasopharynx with a saturated solution of boric acid.

The *urethra* is prepared by administering by the mouth for several days salol or urotropin and by frequent irrigation of the urethra with boric acid solution, normal salt solution, a solution of permanganate of potash (1:6000), or a 1:5000 solution of silver nitrate.

*Preparation of a Patient for An Operation Upon the Stomach.*—(See p. 1079.)

**The Time of Day to Operate.**—A hard-and-fast rule cannot be set as to the time of day when operations should be done. Emergency operations must be performed at once without any consideration as to time. It is often necessary, because of other professional obligations, to set an afternoon hour for an operation. Whenever possible, however, if the nature of the case admits of it, operate in the morning and, preferably, in the early morning. By doing this the patient is saved some hours of dread and worry and the surgeon is enabled to operate when he is fresh, active, and alert; in other words, when he is at his best. A tired mind, like a tired hand, tends to become shaky, and a tired mind may mean incorrect observation, careless technic, impaired judgment, disastrous timidity, or calamitous recklessness.

*Operations on Diabetics.*—Surgical operations upon diabetics are regarded as very dangerous and are employed by most surgeons only in emergencies. In operations upon such subjects gangrene may arise in the wound or diabetic coma may develop. It is important to remember that glycosuria may result from a surgical condition (head injury, sepsis, etc.), and this temporary diabetes may be relieved by operation. I have seen it in appendicitis, and in such cases operation is not contra-indicated, but is imperative. Llewellyn Phillips ("Lancet," May 10 and 17, 1902) refers to the temporary glycosuria produced by injury and sepsis. He thinks that diabetes may directly cause cataract and balanoposthitis, but produces gangrene indirectly by causing nerve degeneration and arteriosclerosis. Phillips points out that a surgical condition and glycosuria may exist independent of and uninfluenced by each other, and many such cases can be operated upon, although operation should be avoided if there is serious disease of some important organ (the liver, for instance). Phillips, in the valuable article referred to, insists that the percentage of sugar is not a measure of the degree of danger; that albuminuria adds greatly to the danger; that the presence of acetone in the urine, and also the presence of ammonia, gives a bad prognosis. Phillips's conclusions as to when to operate and when to refuse operation are as follows ("Lancet," May 10 and 17, 1902): An operation for malignant disease in a diabetic can be performed if the operation would be proper on a non-diabetic individual. Large abdominal tumors can be removed. Cosmetic operations are justifiable if the general health is good and there is not marked arterial disease or nerve degeneration. Operation is justifiable in all emergencies without regard to the condition of the urine. In a diabetic with a surgical malady it is often possible to lessen danger by preliminary treatment. Only an operation of the greatest urgency should be performed if over 1 gm. of ammonia is excreted during twenty-four hours; and if aceto-acetic acid or much albumin is present, every case but the most urgent should be postponed and subjected to medical treatment.

I would add to the conclusions of Phillips that the anesthetic is a danger to the kidneys irritated by the secretion of sugar, and it is desirable, when possible, to use local anesthesia, or, as Robt. T. Morris advises, nitrous oxid and oxygen ("Medical News," June 29, 1901), or spinal anesthesia, or to block the nerves with cocain. In 3 cases I used spinal anesthesia, but in 1 of them the patient died in coma. If sugar diminishes in the urine but increases in the blood, the condition is one of danger. Further, if the urine contains sugar, but neither acetone nor diacetic acid, we should restrict carbohydrates and administer opium or codein. Too strict and too prolonged exclusion of carbohydrates is not justifiable, because it favors the occurrence of acetonuria. If acetonuria exists or arises, a pure meat diet is inadmissible, as it favors diabetic coma. The risk of coma is diminished by giving bicarbonate of soda by the mouth, before and after operation. If coma arises give carbonate of soda intravenously.

**Irrigation** is often practised in septic wounds, but is not required in aseptic wounds. In a septic wound gentle irrigation is often desirable. Irrigation removes many bacteria and much toxin and antiseptic irrigation perhaps antidotes retained toxins. Irrigation must never be forcible for fear it may disseminate infection. Among irrigating fluids we may mention corrosive sublimate, carbolic acid, peroxid of hydrogen, boric acid solution, acetate of aluminum, and normal salt solution. Hot normal salt solution is the best agent with which to irrigate the peritoneal cavity, the pleural sac, the interior of joints, and the surface of the brain. This solution contains 0.9 per cent. of sodium chlorid.

**The Dry Method.**—Many surgeons employ Landerer's dry method in oper-

ating aseptically, no fluid being applied to the wound. As the wound is enlarged gauze sponges are packed in to arrest hemorrhage. On the completion of the operation the sponges are removed, bleeding points are ligated, and the wound is often closed without drainage.

**Ligatures and Sutures.**—In using sutures always remember that they must be tied firmly, but never tightly. A tight suture will cut when the wound swells and will thus fail of its purpose; further, it produces an area of tissue necrosis, which is a point of least resistance in and about which infection is prone to occur. We had far better use many very fine sutures than a less number of thick ones. The individual fine suture is weak, but in numbers they give firm support. A fine suture cannot be tied too tight. If we try to make it tight the attempt is frustrated by the breaking of the suture.

*Catgut.*—The favorite ligature material is catgut. Catgut undergoes absorption in the tissues. Years ago attempts were made by Scarpa, Crampton, and Physick to use absorbable ligatures. Sir Astley Cooper tried catgut. These attempts failed because the material employed was septic, suppuration ensued, the wound gaped, and the ligature was cast off prematurely. Surgeons remained content with non-absorbable ligatures of silk or linen. These ligatures were not cut short, but a long end was left to each one, and the ends were allowed to hang out of the wound. The ligatures were lightly pulled upon from time to time, and when they loosened or cut through were removed. Catgut is the submucous coat of the intestine of the sheep, and is the material from which violin strings are made. It was reintroduced into surgery by Lister. It is usually obtained in the following manner: The small intestine, after separation from the mesentery, is washed in water, laid upon a board, and scraped by a metal instrument. Thus the mucous coat and the muscular coat are scraped away, and the submucous coat only remains. The submucous coat is cut into strips, and each strip is twisted into a coil. Raw catgut is an infected material. It is difficult to sterilize, because in the twisting many organisms get into the interior of the strand, where it is impossible for antiseptics to reach them. Raw catgut obtained from animals dead of splenic fever contains spores of anthrax. If not thoroughly disinfected catgut is dangerous, and some surgeons consider its cleanliness always a matter of grave question and will not use it. Cases of tetanus after operation have been traced directly to infected catgut. The safest raw catgut is obtained from fresh intestines (as advised by Kuhn), is not twisted, is made into strands in sterile machines so as to prevent handling, and is put up in aseptic bundles. Surgeons' catgut is usually obtained from the dealer in skeins containing 30 yards. It should be rough and yellow. The smooth white variety should not be purchased. It has been rubbed smooth with a piece of glass and bleached with a chemical, and in consequence is weak and unreliable. The smallest size is known as double zero, then come single zero, No. 1, No. 2, No. 3, and No. 4. The usual ligature size is No. 2. Nos. 3 and 4 are only used for tying thick pedicles. Nos. 1 and 2 are used for suturing the dura and peritoneum and No. 1 for tying small vessels in the brain. When catgut is used to tie delicate tissue (omental masses, intestinal surfaces, etc.) it must first be softened by immersing for half a minute in normal salt solution. If this precaution is neglected and wiry catgut is used, the ligature or suture will cut and hemorrhage will occur. The greater the diameter of the gut, the more uncertain is the sterilization. Nos. 3 and 4 are of doubtful cleanliness, no matter what method of sterilization is employed, and a strand though clean upon the surface may be infected in its interior. When a strand which is infected within is used by the surgeon the tissues are not infected promptly, but after some days when the catgut has been partially absorbed and the spores or bacteria within the

strand have been set free. Many late infections are due to catgut infected in the interior of the strand. The smaller sizes I believe can usually be satisfactorily sterilized. I am very uncertain as to the surgical cleanliness of the larger sizes.

If catgut is thoroughly freed from bacteria and the wound in which it is used is aseptic, it is a most satisfactory ligature material, is absorbed in the wound after being cut off short, and produces no trouble, although it does increase wound secretion slightly. The smaller sizes are absorbed in four or five days, No. 2 lasts from nine to ten days, Nos. 3 and 4 from ten days to three weeks. Chromicized catgut is absorbed far less rapidly than plain gut.

One of the following methods of preparation may be used:

*Boiling in Alcohol.*—The catgut is soaked in ether for twenty-four hours to remove fat. It is then wound on glass spools, transferred to alcohol, and boiled under pressure. The boiling is conducted in a heavy metal jar with a well-fitting screw-top. The jar is half filled with alcohol. The spools of catgut are placed in the jar, the lid of the jar is screwed down, and the apparatus is immersed in boiling water for half an hour. The gut is kept in this jar until needed. Fowler's catgut is prepared by boiling in alcohol. It is placed in hermetically sealed U-shaped glass tubes. Each tube contains alcohol and twelve ligatures. The alcohol is boiled *by immersing the tube in* boiling water.

The *cumol method* is employed by Kelly in the Johns Hopkins Hospital, and is known as Krönig's method. Cumol is a fluid hydrocarbon which boils at 179° C. Catgut is wound upon spools of glass, and these are placed in a beaker glass, the bottom of which is covered with cotton. A bit of cardboard is placed on top of the beaker, and through a small perforation in the cardboard a thermometer is introduced. The beaker is placed in a sand-bath and the bath is heated by means of a Bunsen burner. The temperature is gradually raised to 80° C., and is kept at this point for one hour, in order entirely to remove moisture from the gut. Cumol, at a temperature of 100° C., is poured into the glass, and the heat is increased until the temperature of the cumol is 165° C., which is a few degrees below its boiling-point. For one hour this temperature is maintained. Then the cumol is poured off and the catgut is allowed to remain for a time in the sand-bath, at a temperature of 100° C., in order to dry. It is transferred for keeping into sterile glass jars or test-tubes.[1]

*The Claudius Method.*—The iodin catgut is prepared by the Claudius method. Mr. Moynihan, of Leeds, makes Claudius catgut as follows: In 10 oz. of sterile water dissolve 1 oz. of crystals of iodid of potassium. When all the crystals are dissolved add 10 oz. of sterile water, and then add 1 oz. of iodin in crystalline form. Dilute the mixture with 4 pints of sterile water. The result is a 1 per cent. solution of iodin and potassium iodid. After the usual preliminary preparation, place the gut in the mixture and keep it in it for for at least eight days before using. It can be kept in it without harm for a number of months. Salkindsohn has modified the Claudius method as follows: Use 1 part of tincture of iodin and 15 parts of proof spirits and immerse the catgut for eight days (J. S. Riddell, in "Brit. Med. Jour.," April 6, 1907).

*Silverized Catgut.*—Blake advocates this form of gut ("Annals of Surgery," January, 1907). He prepares it as follows: He winds four coils of gut on four glass plates, places the plates in a jar containing a 2 per cent. solution of collargolum and keeps them immersed for a week, the jar being shaken once or twice during the period of immersion. At the end of a week the plates are removed from the silver solution and are placed for from fifteen to thirty

[1] See McBurney and Collins, in "International Text-Book of Surgery," and Clark, in "Johns Hopkins Hospital Bulletin," March, 1896.

minutes in 95 per cent. alcohol, then the gut is wound with aseptic care on glass spools and is kept until wanted in 95 per cent. alcohol.

The *formalin method* was advocated by the late Prof. Senn. The catgut is wound on glass test-tubes and is immersed in an aqueous solution of formalin (2–4 per cent.) for twenty-four to forty-eight hours. It is placed in running water for twelve hours to get rid of the formalin. It is boiled in water for fifteen minutes, is cut in pieces and tied in bundles, is placed in a glass-stoppered jar, and is kept ready for use in the following mixture: 950 parts of absolute alcohol, 50 parts of glycerin, and 100 parts of pulverized iodoform. Every few days the mixture should be shaken.

Senn's process is a modification of Hoffmeister's. Even sterile catgut contains a toxic substance, which increases wound secretion, has a poisonous effect on body cells, and favors to some extent limited suppuration. Senn maintains that to counteract this influence gut should not only be sterile, but should be antiseptic, to inhibit the growth of pyogenic organisms which reach the wound from without during operation or subsequently by the blood.

*Dry Heat Method.*—Boeckman wraps catgut in paraffin paper, seals it in a paper envelope, puts it in the sterilizer, and subjects it to dry heat. For three hours it is heated to a temperature of 284° F., and for four hours to a temperature of 290° F. The envelope can be carried in the pocket or the instrument bag. When the gut is wanted the end of the envelope is torn off, an assistant with sterilized hands unwraps the paraffin paper, and the gut is dipped for a moment in sterile water to make it pliable.[1]

*Corrosive Sublimate Method.*—A method which has been largely used is to take raw catgut, keep it in ether for twenty-four hours, soak it for twenty-four hours in an alcoholic solution of corrosive sublimate (1: 500), wind it on sterilized glass rods, and place it for keeping in ether or in alcohol.

*Johnston's quick method* of preparing catgut is as follows: Place it for twenty-four hours in ether; at the end of this period place it in a solution containing 20 gr. of corrosive sublimate, 100 gr. of tartaric acid, and 6 oz. of alcohol. The small gut is kept in this for ten or fifteen minutes, the larger gut from twenty to thirty minutes, but never longer. It is placed for keeping in a mixture containing 1 drop of chlorid of palladium to 8 oz. of alcohol. This gut is strong and reliable. At the time of operation the gut is placed in a solution one-third of which is 5 per cent. carbolic acid solution and two-thirds of which is alcohol.

*Preparation of Chromicized Catgut.*—Chromicized catgut is absorbed less rapidly by the tissues than ordinary catgut. It is used to tie thick pedicles and large arteries, to suture nerves and tendons, and as a suture material in the radical cure of hernia. Chromicized gut, No. 3 and No. 4, will remain unabsorbed in the tissues from four to six weeks. The gut should be soaked in ether for twenty-four hours, and be immersed for twenty-four hours in a 4 per cent. solution of chromic acid in water. The gut is then dried in a hot-air sterilizer and is disinfected by one of the several methods. The cumol method is satisfactory.

*How to Tie Catgut.*—Catgut is tied in a reef knot (square knot) and distinct ends are left on cutting. The second knot, if pulled too tightly, may break the ligature. Moist catgut is slippery and is hard to tie. If a large vessel is tied by catgut, a third knot should be used and the ends cut close to the knot. In tying a vessel in the brain or omentum be sure the gut is not wiry; if it is it will tear the vessel and permit renewed bleeding. Wiry gut must be dipped in salt solution for a moment just before using. Really strong catgut can be tied in a surgeon's knot.

*Kangaroo-tendon and Its Preparation.*—This material is said to be obtained

[1] James E. Moore, in "Phila. Med. Jour.," June 22, 1898.

from the tail of the great kangaroo. It is hard to believe that kangaroos are sufficiently cheap and plentiful to furnish us with it in quantity. It is certainly a tendonous material. It is especially useful for buried sutures in hernia operations; it will be absorbed in the tissues, but only after a long time (sixty to seventy days). Kangaroo-tendon is not grossly infected as is catgut. The material is obtained from a recently killed animal and is promptly dried in the sun. This suture material was introduced by Dr. Henry O. Marcy. It can be prepared in the same manner as the chromicized catgut, and it ought always to be chromicized.

The following method of preparation is recommended by Charles Truax ("Mechanics of Surgery"): Soak the dried tendon until it becomes supple in a 1:1000 solution of corrosive sublimate. Separate the material into individual tendons, place them lengthwise between two towels; dry them; make them aseptic by soaking in a solution of formalin, as we would do with catgut (see above). After washing out the formalin chromicize the tendon by placing it in a fresh 5 per cent. solution of carbolic acid containing 1:4000 parts of chromic acid. When the tendons become "dark golden brown" in color, they are removed from the chromic acid solution, dried between sterile towels, and placed for keeping in 10 per cent. carbolized oil. When wanted, they are removed from the oil, and wiped with a sterile towel saturated with bichlorid solution (1:1000). Kangaroo-tendon is tied in a reef knot. It must be tied firmly, else the knot will slip.

*Silk.*—This material can be used for both ligatures and sutures; many sizes should be kept on hand. Silk is very strong, soft, extremely supple, and does not swell or irritate the tissue. It can be tied into very firm knots. Ordinary surgical silk is a form of twisted silk—that is, several or many strands are twisted into one. Cable twist or Tait's silk is very strong and is used for tying large pedicles. Braided silk is extremely strong and is made by plaiting together several strands of twisted silk. Floss silk is "a straight fiber slightly twisted" (Truax). Silk is usually tied in a reef knot, but occasionally in a surgeon's knot. White silk may be used, or black silk, which is more easily visible. Silk becomes encapsulated in the tissues. It is not absorbed at all or only after a very long time. It is not a good material for buried sutures, as in the long run it may form a sinus. Fine silk ligatures do not cause sinuses.

*Preparation of Silk.*—Sutures of silk should be boiled for half an hour before using in a 1 per cent. solution of carbonate of sodium. Some surgeons keep the silk after boiling in sublimated alcohol (1 : 1000) or carbolic solution (5 per cent.), but it is better to prepare it just before using. A convenient method of preparation is to wind the silk on a glass spool, place the spool in a large test-tube, close the mouth of the tube with jeweler's cotton, introduce the tube into a steam sterilizer, and subject it to a pressure of 10 pounds for twenty minutes, repeating the process the next day. These tubes are carried in wooden boxes sealed with rubber corks.

*Horsehair and Its Preparation.*—This is used for effecting very neat approximation when only light sutures are required; for instance, in wounds of the face. Its chief use is for capillary drainage. It is prepared by washing and then boiling for fifteen minutes in a 4 per cent. solution of carbonate of sodium. It is kept until needed in sublimated alcohol (1 : 1000).

*Silkworm-gut and Its Preparation.*—This material contains fewer bacteria than catgut and does not swell when introduced into a wound. It is strong, solid, smooth, non-irritating, can be drawn through the tissues with slight force, and does not tend to cut the tissue as does a metallic suture. The designation silkworm-gut is a misnomer; the material is not gut at all, but is obtained from the silk-producing glands. Italy supplies most of the gut used by fishermen, but the gut used by the surgeon comes chiefly from Murcia

in Spain. When the silkworms are just ready to spin they are placed in vinegar and water for a number of hours and are thus killed. Each worm is opened and the silk-producing glands are clearly exposed and each gland is drawn by its ends into a single thread. The threads are dried in the air and assume a reddish color (M. J. Triollet, in "Bulletin des Sciences Pharmacologiques," 1905, No. 5. Quoted in "Lancet," Feb. 3, 1906). "This crude silkworm-gut is sold to the manufacturer and further treated. It is first boiled in alkaline water to remove fat and blood and is then dried in the sun, being protected from dust. It is next polished by means of slightly oiled pumice stone. The gut is then bleached with sulphurous acid and rubbed vigorously with chamois leather to remove dust and sulphur" ("Lancet," Feb. 3, 1906). It is a very valuable material, but is not used for ligatures, as it cannot be tied as firmly as catgut and because when left buried in the tissues the sharp ends may stick and irritate and a point of least resistance may be created. Silkworm-gut is prepared by placing it in ether for forty-eight hours and in a solution of corrosive sublimate (1 : 1000) for one hour, or it can be boiled in plain water for half an hour. It is carried in a long tube filled with alcohol. A few minutes before using the gut is placed in carbolic acid and alcohol (one-third of the solution is a 3 per cent. solution of acid, two-thirds of it is alcohol). Silkworm-gut is tied by the surgeon's knot.

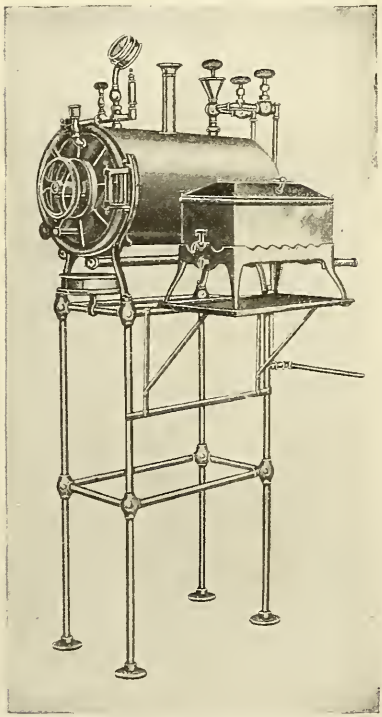


Fig. 37.—Small steam-pressure sterilizer and instrument boiler (Fowler).

*Celluloid Thread and Its Preparation.*—This material is warmly advocated by Pagenstecher. He calls it celluloid yarn, and prepares it from English gray linen thread. I have used it with much satisfaction. It is strong, smooth, flexible, and the knot holds firmly; it can be sterilized by any method used for raw silk, and sterilization by dry heat actually increases its strength. Its one disadvantage is that it absorbs about 40 per cent. of fluid, but it does not soften. The celluloid is added after the thread has been boiled in a 1 per cent. solution of carbonate of soda, wiped on or wrapped in a sterile towel, and dried in hot air or steam. It is then dipped in a solution of celluloid heated in a hot-air sterilizer, and is packed in sterile boxes (Schlutius, in "Pacific Med. Journal," Jan., 1900; Keen and Rosenberger, in "Phila. Med. Journal," May 10, 1900). Celluloid thread can be used for sutures or ligatures.

*Silver wire* is prepared by boiling. It is a very useful suture material, as it can be thoroughly sterilized and has a mild inhibitory effect on the growth of bacteria. Some surgeons use it for buried sutures, but many are opposed to using it thus on the ground that it is apt to lead to sinus formation. *Copper*, *brass*, and *bronze* have a very distinct inhibitory effect on bacteria (C. L. Green, in the "Practitioner," March, 1907), and wire made of any one of these metals is useful. Gold, tin, platinum, magnesium, aluminum, and nickel are devoid of inhibitory power. If iron oxidizes freely it has decided inhibitory

power; if it is so coated that it cannot oxidize it has no inhibitory power. Copper is more powerfully inhibitory than any other metal (C. L. Green, in the "Practitioner," March, 1907). I have used copper wire and brass screws in bone and have used wire of aluminum bronze for various purposes.

Most wounds are closed by interrupted sutures of silkworm-gut, but silk, catgut, chromic catgut, or silver wire can be used. The old continuous suture (glovers' stitch) is rarely used except as a buried suture. An admirable closure can be effected by Halsted's subcuticular stitch, and scarcely any scar results (see page 57). Marcy's buried tendon sutures are much used, especially in hernia operations and in various other operations upon the abdomen.

**Dressings** are made of cheese-cloth. In order to make *antiseptic gauze* the cheese-cloth is boiled in a solution of carbonate of sodium, rinsed out, and dried; it is then soaked for twenty-four hours in a solution containing 1 part of corrosive sublimate, 2 parts of table salt, and 500 parts of water. It is placed in clean jars with glass lids, and it may be kept moist or dry.

*Sterilized* or *aseptic gauze* is prepared by boiling in carbonate of sodium solution, etc., as described under Antiseptic Gauze. The gauze is then

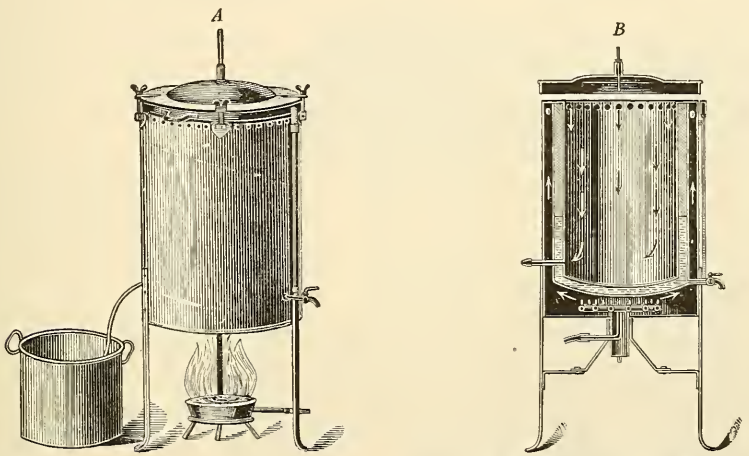



Fig. 38.—Lautenschläger's steam sterilizer for dressings: *A*, Exterior view; *B*, cross-section.

wrapped in a towel and is placed in a steam sterilizer (Figs. 37 and 38) for an hour. It is kept until wanted in sterile glass jars with glass lids.

*Sterile absorbent cotton* is prepared in the same manner as gauze. Cotton is useful as a dressing to supplement gauze, being placed on the outside of the gauze. It absorbs quantities of serum, but will take up very little pus.

*Iodoform gauze* is very useful for packing in the brain and abdomen, for packing abscesses and tuberculous areas, and for dressing foul wounds. It is prepared as follows: Make an emulsion composed of equal parts by weight of iodoform, glycerin, and alcohol, and add corrosive sublimate in the proportion of 1 part to 1000 of the mixture. This mixture stands for three days. Take moist bichlorid gauze, saturate it with the emulsion, let it drip for a time, and keep it in sterilized and covered glass jars (Johnston).

Lister's *cyanid gauze* (double cyanid of zinc and mercury) is not certainly antiseptic, and must be dipped into a corrosive sublimate solution (1 : 2000) before using. All forms of gauze can be bought ready prepared from reliable firms.

Some surgeons place *silver foil* upon a wound before applying the gauze (Halsted, page 32). Very small wounds in which drainage is not employed may

be dressed by laying a film of aseptic absorbent cotton over the wound and applying, by means of a clean camel's-hair brush, *iodoform collodion* (48 gr. of iodoform to 1 oz. of collodion). Among other materials sometimes used for dressing wounds the following should be mentioned: Wood wool, absorbent wool, moose pappe, oakum, jute, peat, and sawdust.

**Protectives.**—A protective is a material placed directly upon wounds to shield them from irritation and infection. The commonly used protectives are Lister's oiled silk protective, gutta-percha tissue, rubber dam, waxed paper, paraffin paper, mackintosh, Cargile membrane, and silver foil. Undoubtedly, many antiseptic agents destroy young cells and in this way hinder repair. The same is true of certain rough dressings.

R. T. Morris showed us that gauze and particularly cotton are injurious to a healing wound. A non-irritant protective laid directly upon a wound may be useful by saving new cells from injury by an irritant germicide and from being pulled away at each change of dressings.

Among the best protectives in common use are Lister's protective, gutta-percha tissue, silver foil, and Cargile membrane. Morris condemns gutta-percha tissue as irritant. He uses thin gold-beaters' skin made from the peritoneum of the ox, which material he calls Cargile membrane, after an Arkansas physician who introduced it into practice. The advantage of this material is that moisture cannot penetrate and new cells do not adhere. I have used it with satisfaction in many cases, but in wounds and ulcers prefer silver foil.

Silver foil, Lister's protective, or gutta-percha tissue is laid directly upon a wound, the dressing being placed above it. Silver foil comes in books and is sterilized by dry heat. Gutta-percha tissue should be sterilized by washing with soap and water, rinsing in sterile water, and soaking in a solution of corrosive sublimate. Lister's protective is employed to save the wound from the irritation of carbolized dressings.

*Impermeable Material Over Dressings.*—In the United States, if it is desired to place an impermeable material *over* a dressing, a rubber dam is usually employed. A rubber dam before being used should be washed with soap and water and soaked in a solution of corrosive sublimate.

The use of an impermeable material on the outside of the gauze dressing is not nearly so common as formerly. In an aseptic wound dry dressing uncovered by rubber is the most useful plan. When a dressing is covered by an impermeable material it becomes moist, acts as a poultice, and the discharges on the dressing may undergo decomposition.

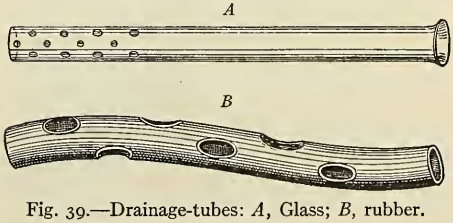


Fig. 39.—Drainage-tubes: *A*, Glass; *B*, rubber.

**Drainage** is used in all infected wounds, in most very large wounds, in wounds to which irritant antiseptics have been applied, in cases in which large abnormal cavities exist, in very fat people, and in individuals whose skin is so thin that we dare not apply firm pressure (see page 57). Drainage, when needed, is obtained by rubber or glass tubes (Fig. 39), by strands of horsehair, silkworm-gut or catgut, by pieces of gauze, and occasionally in the abdomen by Mikulicz's bag or tampon by which we obtain pressure to arrest hemorrhage and also secure drainage (Fig. 42). Gauze drainage is satisfactory for the removal of serum, but not pus. An objection to the gauze drain is the suffering caused by its removal. Before removal it should be thoroughly moistened and carefully separated from the wound edges to which it is apt to adhere. Sometimes it is

removed a little at a time. If pus is plentiful, especially if it is thick, rubber tubes should be used. The caliber of the tube must be sufficient to permit the pus to flow freely. Rubber drainage-tubes (Fig. 39, *B*) are rendered sterile by boiling in plain water. They are kept until wanted in a mercurial solution. This solution should be changed every few days, because the mercury is apt to be precipitated as sulphid. Glass tubes are sterilized by boiling. A bit of rubber tissue is sometimes used for drainage. The *cigarette drain* is useful in many cases. It drains serum well and is easily removed. It is made by folding up a piece of gauze and surrounding it, except at each extremity, with gutta-percha tissue. Gauze, catgut, etc., are known as capillary drains. When moist they drain serum excellently, but pus very badly or not at all. Pus requires tubular drainage. Drainage-tubes or strands are brought out at a portion of the wound which will be dependent when the patient is recumbent.

**Sponges.**—Marine sponges are never used to-day; instead we use gauze rolled into balls, the edges and ends being turned in.

**Pads and Packs.**—Ashton's gauze pads are very useful to push away structures during an abdominal operation and to temporarily pack a wound. Several layers of sterile gauze are taken. Each piece is about 6 in. long and 4 in. wide. A stitch is run round the margins. A piece of tape is sewed in one corner. During the operation the tape protrudes from the wound and is clamped with forceps. This plan saves the pad from being lost or forgotten in the abdomen. Long narrow pieces of gauze make the safest and best packs (*Halsted's packs*), as then a long end always protrudes from the wound during the operation.

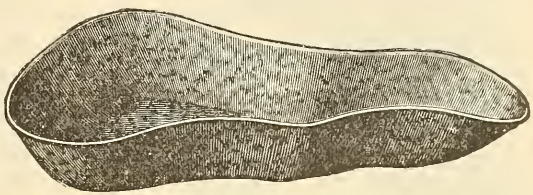


Fig. 40.—Smith's dressing basin.

**Change of Dressing.**—Dressing should not be changed unless indications call for change. To unnecessarily meddle with a wound is stupid and harmful. In many cases dressings are not renewed until the wound has healed. When a change of dressings is determined upon the surgeon should carefully sterilize his hands and forearms and should have at hand a warm solution of corrosive sublimate, normal salt solution, an irrigator, iodoform, iodoform gauze, scissors, forceps, basins (Figs. 40 and 41), etc. Dressings should be moistened before removal with salt solution or corrosive sublimate solution. If they stick to the part, a spray of hydrogen dioxid projected from an atomizer between the skin and dressings will soon loosen them. Dressings must be changed as soon as soaking with blood or wound-fluid is apparent. If the wound becomes uneasy and painful, or if constitutional symptoms of wound infection arise, the dressings must be removed to permit of inspection of the wound. A change of dressings must be effected with all of the aseptic care employed in a surgical operation. Dressings are not dispensed with until the wound is soundly healed.

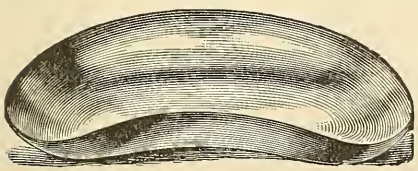


Fig. 41.—Plain dressing basin.

**Removal of Stitches.**—Buried stitches of animal material are not removed by the surgeon, but are gradually absorbed in the tissues. Buried stitches of silk or silver wire, which are used by some surgeons, although they are not absorbed in the tissues, may never require removal, but in some cases cause sinuses to form, and a sinus from a suture or ligature will not heal until the

suture or ligature is cast out or removed. Sutures of aluminum-bronze wire are absorbed after a long stay in the tissues.

If a catgut stitch is passed through the skin and tied externally the loop in the tissue is absorbed, but the knot and remainder of the loop is on the surface and is not absorbed, but remains adherent to the wound and the surgeon needs only to lift it off with forceps. Catgut is used as a material for cutaneous suturing in the operation of circumcision. When a skin wound is closed by unabsorbable sutures, as it usually is, the surgeon at the proper time takes forceps and scissors and removes the stitches. There is no day after an operation immutably fixed as the proper day to remove stitches. Stitches may usually come out from the sixth to the eighth day, although if there is much tension on the edges of the wound they are allowed to remain several days longer. In large wounds half of the stitches are taken out at one time, the remainder being allowed to remain for a couple of days longer. When a stitch begins to cut, it is doing no good, and it should be removed, no matter how short a time it has been in place. If it is allowed to remain it will cut into the wound, make a stitch-abscess, and cause an irregular suture-line. In order to remove a stitch pick up an end distal from the knot with forceps, lift it lightly, cut one side of the suture close to the skin by scissors, and remove it by pulling in the direction of the side on which the suture was cut (Fig. 43).

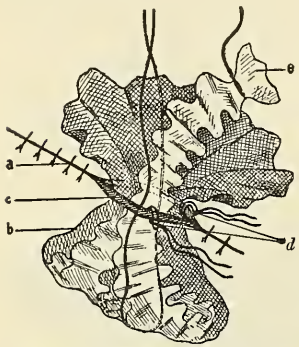



Fig. 42.—Mikulicz's bag: *a*, Abdominal sutures; *b*, gauze bag; *c*, abdominal wound; *d*, loops in the abdominal wall; *e*, gauze strip.

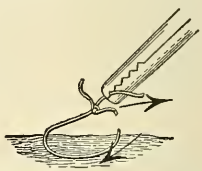


Fig. 43.—Method of extraction of a suture (Esmarch and Kowalzig).

**Bandages.**—For retaining dressings upon wounds unbleached muslin bandages may be used, but in most cases gauze bandages are employed. Gauze bandages may be applied when dry or wet; normally, they are applied when dry. Gauze bandages soaked in corrosive sublimate solution are antiseptic, do not seal the dressing, hence do not act like rubber dam; can be applied firmly, evenly and rapidly, and are very comfortable.

## III. INFLAMMATION

**Definition.**—When the tissues are injured they react or respond, and this reaction or response is known as inflammation. The process of inflammation was defined by the late Sir John Burdon-Sanderson as "the succession of changes which occur in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality." Professor Adami, in his article upon inflammation in Allbutt's "System of Medicine," points out that this definition really includes too much. He alludes to the hemorrhage which occurs in the liver after a traumatism, and the subsequent changes in the extravasated corpuscles, and points out that these changes are not inflammatory phenomena. This definition, however, includes all inflammatory conditions, is largely employed, is very useful, indicates the cause, and, as Burdon-Sanderson says, makes clear that inflammation is a process and not a state (Adami). Adami's definition is as follows: "The series of changes constituting the local manifestation of the attempt at repair

of actual or referred injury to a part, or, briefly, the local attempt at repair of actual or referred injury." The changes alluded to in Burdon-Sanderson's definition comprise (1) changes in the vessels and the circulation, (2) departure of fluids and solids from the vessels, and (3) changes in the perivascular tissues.

**Vascular and circulatory changes** were formerly thought to be absolutely essential to inflammation in both vascular and non-vascular tissues. In the former they occur in the inflamed tissues; in the latter (cornea and cartilage) they are manifest in neighboring tissues from which the non-vascular area derives its nutritive material. As a matter of fact, in inflammation vascular changes are almost always present; but in a rather trivial corneal inflammation the episcleral vessels may not dilate, and the only white corpuscles which gather in the damaged area are those which come from the lymph-spaces of the cornea. Inflammation in any tissue will not be accompanied by vascular dilatation unless the process reaches a certain stage of severity.

Fig. 44.—Normal vessels and blood-stream.

Fig. 45.—Dilatation of the vessels in inflammation.

**Active Hyperemia.**—When an irritant is applied to tissue there may be a momentary arterial contraction due to irritation of the nerves, but this contraction is transitory, and is not an inflammatory phenomenon. The first vascular phenomenon is dilatation of all the vessels—capillaries, venules, and arterioles—appearing first and being most pronounced in the small arteries. As a result of the dilatation there are increased rapidity of circulation and increased determination of blood to the part, and the area of hyperemia becomes warmer than is normal. This condition of increased circulatory activity is known as "active hyperemia" (Fig. 45).

Active hyperemia is an increase in the amount of moving blood in a part. Passive hyperemia is an increase in the amount of blood in a part, but not of moving blood, as passive hyperemia or congestion is due to venous obstruction, and the blood is stagnated. Diminution in the amount of blood in a part is *ischemia*. Local anemia is the complete cutting off of the blood-supply of a part.

In active hyperemia more blood goes to the part and more blood passes through it, an increased amount of venous blood comes from the hyperemic

area, the venous tension is increased, and the veins may even pulsate. The capillaries, which under ordinary circumstances contain but few blood-cells (Fig. 44), become filled with corpuscles (Fig. 45), and even the smallest capillaries pulsate. The blood in the veins adjacent to the area of inflammation is of a much lighter red than in health. Many capillaries which were invisible under normal conditions become visible when active hyperemia exists. The capillaries contain no muscle-fiber, and hence these tubes cannot actively contract, except so far as the caliber of the tubes is altered by the contraction or expansion of the endothelial cells of the capillary wall. Contraction and dilatation of the capillaries depend chiefly on the amount of blood sent to or retained in them. In active hyperemia the increased amount of blood sent to the part causes capillary dilatation. As a result of the dilatation the endothelial cells become thinner than before, the cells in consequence of irritation lose some of their power to restrain exudation, and some observers assert that openings are formed between the cells or that previously existing openings enlarge (page 82). Fluid elements rarely leave the blood-vessels during active hyperemia, but they occasionally do. The wheals of urticaria are thus formed (Warren). Active hyperemia is often the first stage of an inflammation, but it is not of necessity followed by other inflammatory changes, and it can be caused by nerve section or nerve stimulation.

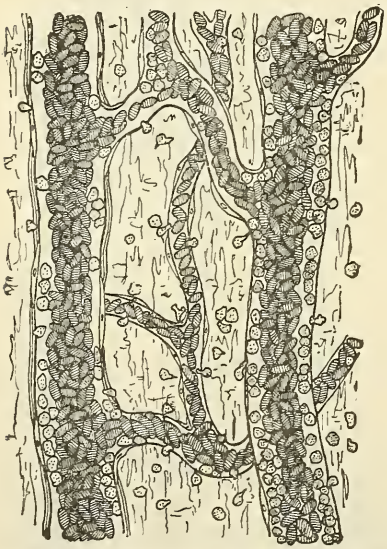


Fig. 46.—Retardation of blood and migration of white corpuscles in inflammation.

The duration of active hyperemia is variable. If the irritation was brief, the hyperemia is very transitory. In some cases dilatation with accelerated circulation is scarcely more than momentary, giving way almost immediately to dilatation with retardation. If the irritation is prolonged, hyperemia may last some time before giving way to retardation. In the web of a frog's foot, if an irritant is applied, hyperemia lasts from one-half hour to two hours before it is replaced by retardation.

*Clinical Signs of Active Hyperemia.*—A hyperemic part, if on or near the surface, is red in color, imparts a sense of heat to the examining hand, the color quickly disappears on pressure and quickly returns when pressure is released. In a congested part the temperature is diminished, the surface is purple, the congested veins are visible, there are edema and a sensation of coldness and numbness. When congestion is purely local the lividity disappears quickly when pressure is applied and returns quickly when pressure is removed. When due to disease of the heart or lungs, lividity disappears and returns slowly. When a local congestion is about to give way to gangrene the lividity disappears very slowly on pressure and crawls back very slowly when pressure is released.

**Retardation.**—After active hyperemia has existed for a variable time the blood-current begins to lessen in velocity, until it becomes more tardy than in health. This is known as "retardation of the circulation." Retardation is first noted in the venules, next in the capillaries, and last in the arterioles; but arterial pulsation continues. The red cells take the center of the blood-stream, which is known as the axial current. The white corpuscles settle out

of the central stream, separate from the red cells, and float lazily along near the vessel wall, and they are accompanied by many third corpuscles. The white cells show a strong tendency to adhere to the venule walls, and, as a result, accumulate against the inside of, and stick to, these walls and to one another, until the venules are entirely lined with layers of *leukocytes* (Fig. 46). The third corpuscles act in a similar manner and take the peripheral current. In the capillaries some leukocytes gather, but not so many. In the arterioles they adhere during cardiac dilatations, but are swept away by the force of the heart's contractions. Retardation is believed to be chiefly due to paresis of the muscular walls of the arterioles. This causation seems probable when we recall Lord Lister's experiments upon the pigment-cells of the frog's foot. Lister proved that inflammation paralyzes the pigment-cells, and concluded that dilatation at the focus of an inflammation is due to the paralyzing action of an irritant. Dilatation at a distance from the focus is a reflex phenomenon (W. Watson Cheyne). When the vessels are weakened or paralyzed the contractions of the arterioles are feeble or absent, and the blood is no longer urged forward by arterial power. The endothelial cells of the small vessels enlarge distinctly during retardation and develop a condition of stickiness,which leads the white cells to adhere to them, and thus increases resistance to the current of blood and adds to retardation. Fluids pass through the wall of a vessel in this condition more readily than through a healthy vessel, and white corpuscles leave the vessel in large numbers.

**Oscillation and Stagnation.**—By the accumulation of leukocytes the blood-stream is progressively narrowed and the axial current is impeded. The red blood-cells begin to stick to one another, forming aggregations like *rouleaux* of coin, which masses increase the difficulty the axial current has to contend with, until progressive movement ceases and the contents of the vessels sway to and fro with each heart-beat. This is the stage of *oscillation*. In a short time oscillation ceases and the vessels are filled with blood which does not move, and the vessel walls become irregular in outline or even pouched. This stage is known as "*stasis*" or "*stagnation*." Stasis is chiefly due to paralysis and damage of the vessel walls. Migration ceases when stasis takes place. If stasis persists, coagulation occurs, because the vessel walls have been so injured by the irritant as to be practically dead material, and they are no longer able to prevent clotting of their contents. Finally, in persisting stasis the vessel walls rupture or are entirely destroyed.

**Résumé of the Vascular Changes of Inflammation.**—We can sum up the vascular changes of inflammation by stating that they consist in a dilatation of the small vessels and a primary acceleration, a secondary retardation, and a subsequent stagnation of the blood-current, exudation of blood-liquor, adhesion of leukocytes to the walls of veins and capillaries, migration of leukocytes, the aggregation of the red blood-cells into intravascular masses, and coagulation of the material remaining in the vessel.

**Exudation of Fluids.**—It is to be remembered that in the process of nutrition blood-liquor and also white cells pass into the tissues through the walls of veins and capillaries, and during this process certain other materials are passing from the tissues into the vessels. Hence, a diffusible irritant in the vessels may pass into the tissues and a diffusible irritant in the tissues may pass into the vessels. Whenever retardation of the circulation arises there is an increase in the amount of plasma which passes out of the vessels, but in inflammation the exudation into the lymph-spaces is vastly greater in amount and is different in composition. In a slight inflammation, and in the early stage of any inflammation, there is an increase in the fluid exudate, and we speak of the condition as "*serous inflammation*." This fluid is really not serum, but is liquor sanguinis. We find true serum in passive congestion, not in active

inflammation. The fluid of a serous exudation contains very few white cells, and hence little or no fibrin can form in it, and coagulation does not take place in the perivascular tissues; and if the inflammation goes no further, the exudate is absorbed by the lymphatics. A blister is an example of serous inflammation. If the inflammation continues to intensify, the exudation is altered in character—it becomes thicker, turbid and very coagulable, and exhibits a greatly increased bactericidal power. It contains many white cells and fibrin elements, and coagulates in the tissues, because some of the leukocytes break up and set free fibrin ferment, and fibrin ferment causes the union of calcium and fibrinogen and the formation of fibrin. This fluid exudate is known as "*lymph*," or *plastic exudation*, and when it is present we speak of the condition as "*plastic inflammation*." Lymph can be seen in the anterior chamber of the eye in cases of plastic iritis. Coagulated fibrin in a recent wound causes the edges to adhere or glazes the raw surface. In inflammation of a mucous surface it may appear as a false membrane. In inflammation of serous surfaces it may glue the surfaces together and lessen motion, the fibrinous masses which effect the gluing being called fibrinous or plastic adhesions. Such adhesions within the abdomen may seal a perforation, may cover a raw spot, or may encompass an area of infection and prevent fatal diffusion. Further, fibrin surrounds and entangles bacteria and retards their diffusion. Pyogenic cocci lessen, retard, or prevent fibrin formation or destroy fibrin previously formed. Fibrinous adhesions may, of course, do harm. They may retard or prevent the absorption of exudate; they may narrow and obstruct important structures (bowel, urethra, larynx); they may bind up and cripple an important viscus (liver, heart, or brain). Fibrinous adhesions may be succeeded by dense contracting and constricting bands of fibrous tissue. The lymphatics endeavor to absorb the fluid exudate in inflammation, but become occluded by coagulation, and the area they drain becomes swollen, hard, and "brawny." The slighter the inflammation, the less albuminous is the fluid; the more intense the inflammation, the more albuminous is the fluid. The focus of a severe inflammation feels brawny because of coagulation of a highly albuminous exudate; the periphery of an inflammation is soft and edematous because of the presence there of thin and non-coagulated exudate. Inflammatory lymph contains proteins and other substances. "Of these the more important are ferments, the results of proteolysis (notably fibrin and its precursors and peptones), and in many cases mucin, together with bactericidal substances, and, where bacteria are present, the products of their growth."[1] The amount of the exudation varies with the violence of the irritation, the nature of the irritant, the general condition of the organism, and the state of the tissues which are involved. In dense tissue (bone, periosteum, etc.) the exudation is scanty. In loose tissue (subcutaneous tissue) it is profuse. Profuse exudation may take place into a joint, the pleural sac, the peritoneal cavity, or the pericardium. In such cases the exudation is profuse because the serous membrane has a thin covering of endothelium, contains quantities of vessels, and the vessels receive but a thin covering and obtain but a scant support toward the cavity.

Does the plasma leave the vessels as a simple filtrate? Some maintain that it does. Heidenhain and others claim that it does not, and believe that the endothelial cells play an active part in the process. Heidenhain likens exudation to secretion, because some materials from the plasma pass out and others do not. Adami is inclined to agree with Heidenhain, that the endothelium plays "not a passive but an active rôle." Are there spaces between the endothelial cells of the capillary? It was long taught positively that there are no open spaces between the endothelial cells of the vessel wall and that

[1] Adami, in Allbutt's "System of Medicine."

these cells are held close together by a cement substance. It is now believed by some observers that spaces exist between the protoplasmic strands which hold the cells together, these spaces being closed when the vessel is contracted and open when the vessel is dilated. When these spaces are open fluid passes, and through these doorways leukocytes emerge.

Fig. 47.—Stages of the migration of a single white blood-corpuscle through the wall of a vein (Caton).

**Migration and Diapedesis.**—Even early in an inflammation a number of white corpuscles pass through the vessel walls; but when the inflammation is well established, large numbers, and when it is severe vast hordes, pass into the perivascular tissues. This process is known as "migration" (Figs. 46 and 47). The leukocytes throw out protoplasmic arms, insert themselves between the cells of the walls of the vessel, and pull themselves through by their power of ameboid movement (Fig. 48). Some observers claim that they do not pass through existing open doors, but force openings which close after them. This is readily accomplished, because the vessel wall is itself damaged, weakened, and convoluted. Others claim that stomata exist between the endothelial cells, the vessel wall being porous like a filter (see page 82). The escape of leukocytes takes place chiefly from the venules, though some migrate through the capillaries and even the arterioles (Fig. 46).

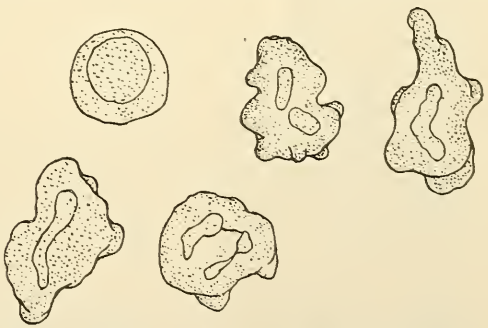


Fig. 48.—Ameboid movements of a leukocyte (Warner).

The leukocytes are influenced to move toward the damaged tissue by the attractive force known as *positive "chemiotaxis,"* a force which draws them toward invading bacteria, to regions of irritation, and to areas of tissue death. Leukocytes may move from very virulent organisms, influenced by what is known as *negative "chemiotaxis."* The migration of a leukocyte requires but a short time. Figure 47 shows the migration of a white blood-cell through a vein wall, the process requiring one hour and fifty minutes. In very acute inflammations red corpuscles also pass into the tissues. Red corpuscles are not capable of ameboid movements, and if they do escape from the vessels the process is passive on their part and not active. This passive escape happens because the capillary walls have been destroyed or because stomata have been greatly enlarged by vascular dilatation. If red corpuscles do pass into the

exudate, as happens in pneumonia, the inflammation is a very severe one, and is called a hemorrhagic inflammation. The escape of corpuscles by a passive process is known as "diapedesis," in contradistinction to the escape of leukocytes by active ameboid movements, a process known as "migration." The white corpuscles usually greatly increase in number in the blood of a person who has an acute inflammation, and the blood-making organs, such as the spleen and lymphatic glands, are often enlarged. An increase of white corpuscles in the blood of an individual is called *leukocytosis* (see page 92).

**Blood-plaques.**—*Blood-plates, blood-plaques,* or *third corpuscles,* may be discovered in freshly drawn blood, but unless they are present in unusual numbers they will rarely be seen in specimens prepared in the usual way. The third corpuscles can be seen by a high-power microscope in the moving blood of the web of a frog's foot. In blood outside of the body they are destroyed as soon as coagulation begins, and in order to see them coagulation must be prevented. Some observers maintain that the third corpuscles are the real fibrin-formers. The blood-plaques, or third corpuscles, are found to be present in increased numbers in inflammation. In health their usual proportion to red cells is as 1 to 20. They are especially numerous at the height of fever processes and during convalescence from an extensive abscess.

**Changes in the Perivascular Tissues.**—The cells of the perivascular tissue are phagocytes, and when stimulated they enlarge, become more actively phagocytic, and undergo reproduction. The liquor sanguinis which exudes during an acute inflammation coagulates unless prevented by virulent bacteria. It has often been asserted that exudation is Nature's method of supplying nutriment to the cells of the damaged region. Adami points out the apparently contradictory observation that the amount of exudate is in direct proportion to the rapidity of cell destruction, but, nevertheless, concludes that exudation stands in close relation to cell proliferation.[1] From whatever cause, tissue-cells multiply, and this process is known as "*cell proliferation.*"

When a tissue is injured it inflames, and, as Adami points out, the reaction we call inflammation is an attempt to repair injury.

Irritation may lead to degeneration and death of cells; it may lead to growth and multiplication. In many cases both processes are active in the acute stage, the cells at the focus of the inflammation undergoing degeneration and destruction, and those at the boundary undergoing growth and proliferation.

If tissue-cells have been seriously damaged they perish, and new cells are required to replace them. The inflammatory process has led to exudation of plasma and migration of leukocytes into the perivascular tissues. The connective-tissue cells multiply and produce young cells, which are known as "*fibroblasts,*" and which eat up many leukocytes. Early in an inflammation polynuclear leukocytes preponderate, later mononuclear phagocytic cells predominate (Opie). The leukocytes contain two enzymes. One is derived from bone-marrow and digests protein in an alkaline medium; the other is derived from lymph-glands and digests protein in an acid medium (Opie). The migrated leukocytes in part surround the inflamed region and retard diffusion of the process. Many enter the diseased area and attack bacteria. Some undergo degenerative changes and liberate fibrin ferment which makes the exudate clot. Some move out of the inflamed area, each one carrying within it tissue débris or a dead bacterium, and many are eaten up by the fibroblasts. There is no real proof that leukocytes proliferate and help directly to form new tissue. This mass of young cells, taking origin from the fixed cells, has been called *embryonic tissue,* because of a fancied resemblance to the cells of the

[1] Adami, in Allbutt's "System of Medicine."

embryo. John Hunter called it *juvenile tissue.* It has also been called *indifferent tissue,* because of the belief that it could be converted indifferently into various tissue according to circumstances. It is also spoken of as *inflammatory new formation.* The cells of embryonic tissue are called *fibroblasts* because they form fibrous tissue.

An exudation may be absorbed by the lymphatics. It may be converted into pus if infected with pyogenic bacteria, or be replaced by cells from the proliferation of fixed tissue-cells, the cellular mass being subsequently vascularized by the extension into it of capillary loops derived from adjacent capillaries. When embryonic tissue is filled with blood-vessels—that is to say, when it is vascularized—it is called *granulation tissue.* Granulation tissue is finally converted into fibrous tissue. The above complicated processes, vascular and perivascular, are not accidents nor haphazard freaks, but are Nature's efforts to bring about a cure.

Dilatation is due to the direct effect of the irritant upon the muscle or its nerve-elements. Retardation and stasis are due to paralysis of the vessel wall, which paralysis causes resistance to the passage of the blood-stream and adhesion of the leukocytes to the vessel wall. The blood-liquor exudes and the leukocytes migrate. Often these efforts of Nature succeed. Acceleration of the circulation may succeed in washing away an irritant from the vessel wall. By bringing quantities of blood to the part copious exudation of plasma is rendered certain. The exudation may wash and remove irritants from the tissues, and the germicidal blood-liquor may destroy bacteria in the damaged area. The migration of corpuscles may prove of great service. The leukocytes surround an area of infection and tend to limit its spread. Leukocytes have phagocytic properties, and energetically attack and often destroy bacteria, and they furnish enzymes which may digest proteins and antitoxins, which antagonize and may neutralize the poisons produced by micro-organisms. Leukocytes aid in forming fibrin. Fibrin formation is of service by helping immobilization and by hindering the spread of bacteria. Leukocytes also aid in separating dead tissue from living, and they remove tissue débris from the area of inflammation. The multiplication of the fixed connective-tissue cells leads to the formation of fibroblasts, and fibroblasts are converted into fibrous tissue, which effects permanent repair (these changes will be alluded to again in the section on Repair).

Nature may fail in her efforts. For instance, an enormous exudate increases stasis and may cause such tension that gangrene results.

**Inflammation in Non-vascular Tissue.**—A type of non-vascular tissue is the cornea, and the cornea can inflame. The healthy cornea contains no blood-vessels. It is formed of many layers of fibers, each layer running parallel with the corneal surface and forming angles with the fibers of the adjacent layers. Between the layers are communicating lymph-spaces containing connective-tissue cells known as corneal corpuscles. It obtains its nourishment in part from the vessels of the conjunctiva, but chiefly from the vessels of the ciliary body and sclera. When the cornea inflames the episcleral, conjunctival, and ciliary vessels usually dilate and pour out exudate, and the fluid exudate and the leukocytes enter into the corneal lymph-spaces. The exudate coagulates and cell multiplication ensues as in any other inflammation. In mild inflammations the vessels about the cornea may not dilate. Leukocytes, from the lymph-spaces, reach the seat of injury in small numbers, and the fixed cells multiply. De Nancrede points out that in trivial inflammation, which injures but does not destroy the epithelium, leukocytes may not go to the seat of inflammation, the only change being enlargement and multiplication of corneal corpuscles. If new formation takes place, a permanent opacity mars the cornea as a consequence.

Cartilage has no blood-vessels except in regions where growth is very active or where ossification is taking place. Cartilage has no spaces, like the cornea, for a free circulation of lymph. In man canals have not been demonstrated and it is thought that fibrils conduct nutritive fluids, the nutritive plasma flowing between the cells, but there is no direct connection with blood-vessels. The plasma is furnished by the vessels at the margin of the perichondrium. Cartilage can inflame, and an inflammation of this structure is slow in evolution and of long duration. When inflammation occurs the cartilage cells enlarge and their nuclei proliferate, the intercellular substance softens and cartilage cells may be cast off. After a long time vessels may invade the inflamed cartilage and fibrous tissue forms from the perichondrium, but in some cases a loss of substance is not repaired.

**Inflammation of Mucous Membrane.**—It may be catarrhal, suppurative, croupous, or diphtheritic. In a *catarrhal inflammation* the increased blood-supply causes an excessive flow of mucus. The submucous tissues present the ordinary changes of inflammation and quantities of epithelial cells are cast off from the surface. Fibrous tissues may form in the submucous tissue and thus cause permanent thickening (strictures, etc.).

*Suppurative inflammation* is usually preceded by catarrhal inflammation. In this condition there is a discharge of mucopurulent fluid and ulcers are apt to form. A trivial loss of substance permits of regeneration, but a considerable loss is repaired by fibrous tissue, which by its bulk and by contracting may interfere greatly with the functional usefulness of an organ or a canal.

A *croupous inflammation* is one in which quantities of epithelial cells are cast off the surface and there forms upon the surface a highly fibrinous exudate (false membrane).

In *diphtheritic inflammation* the mucous membrane is destroyed and the false membrane invades the submucous tissue. Diphtheritic inflammation is due to a specific bacillus.

**Classification of Inflammations.**—The various forms of inflammations are—(1) *Simple* or *common*, that which is due to any ordinary traumatic, chemical, thermal, or actinic cause, and not to bacteria. An instance of simple inflammation is traumatic periostitis or sun dermatitis. It does not tend particularly to spread. Often the cause of a simple inflammation is momentary in action; (2) *infective* or *specific*, that which is due to micro-organisms, as the streptococcus of erysipelas. An unsuccessful attempt has been made to charge all inflammations to bacteria. It is true that bacteria can generally be found in inflammatory areas, but that they are the only causes of inflammation is accepted by few. Infective inflammations often tend to spread widely; (3) *traumatic*, which is due to a blow or an injury; (4) *idiopathic*, which is without an ascertainable cause. There is certainly a cause, even if it cannot be pointed out, and the term "idiopathic" means that we do not know the cause; (5) *acute*, which is rapid in course and violent in action; (6) *chronic*, which follows a prolonged course; (7) *subacute*, which is intermediate in violence and duration between acute and chronic; (8) *sthenic*, characterized by high action; it occurs in strong young subjects; (9) *asthenic* or *adynamic*, occurring in the old, the debilitated, and the broken down. In such an inflammation there is no certain limitation of the inflammation by leukocytes, and there is an indisposition on the part of the tissue-cells to form fibroblasts; (10) *parenchymatous*, affecting the "parenchyma," or active cells of an organ; (11) *interstitial*, affecting the connective-tissue stroma of an organ; (12) *serous*, characterized by profuse non-coagulating exudation (as in pleuritis) or by marked inflammatory edema; (13) *plastic*, *adhesive*, or *fibrinous*, characterized by an exudation which glues together adjacent surfaces, as in peritonitis; (14) *purulent*, *phlegmonous*, or *suppurative*, when pyogenic cocci are present

and multiply; (15) *hemorrhagic*, when the exudate contains many red blood-cells, as in strangulated hernia and in the pustules of black small-pox; (16) *croupous*, when an inflammation produces upon the surface of a tissue a fibrinous exudate which cannot be organized into tissue, and which is due to the action of micro-organisms. An exudate of this character was called by the older surgeons "*aplastic lymph*." It occurs most usually on mucous membrane; (17) *diphtheritic*, which differs from croupous in the fact that the false membrane is in the tissue rather than upon it; (18) *gangrenous*, an inflammation resulting in death of the part, the gangrene being due to the tension of the exudate or the virulence of the poison; (19) *healthy*, when the tendency is to repair; (20) *unhealthy*, when the tendency is to destruction; (21) *latent*, an inflammation which for some time does not announce itself by any obvious symptoms, as the inflammation of Peyer's patches in typhoid fever; (22) *contagious*, when its own secretions can propagate it; (23) *dry*, without exudation; (24) *hypostatic*, arising in a region of passive congestion (as a bed-sore); (25) *malignant*, due to a malignant growth; (26) *catarrhal*, affecting a mucous membrane; (27) *neuropathic*, due to impairment of the trophic functions of the nervous system, as in perforating ulcer; and (28) *sympathetic* or *reflex*, due to disease or injury of a distant part, as when orchitis follows mumps.

**Extension of Inflammation.**—Inflammation extends by continuity of structure, by contiguity of structure, by the blood, and by the lymphatics. Extension by continuity is seen in phlebitis. Extension by contiguity is seen when a cutaneous inflammation advances and attacks deeper structures. Extension by the blood is seen in the formation of the small-pox exanthem. Extension by the lymphatics is witnessed in a bubo following chancroid.

**Terminations of Inflammation.**—Inflammation may be followed by a return of the tissues to health, and this return may take place by delitescence, by resolution, or by new growth. By *delitescence* is meant abrupt termination at an early stage, as when quinsy is aborted by the administration of quinin and morphin, and the production of a sweat; *resolution* means the gradual disappearance of the symptoms when inflammation has passed through its regular stages; and *new growth* means that an inflammation has lasted a considerable time, with ample blood-supply and without suppuration, and has gone on to the formation of fibroblasts, granulation tissue, and fibrous tissue. Inflammation may be followed by death of the inflamed part or necrosis. Death of the part may be due to suppuration, ulceration, or gangrene.

The **causes of inflammation** are—*predisposing*, or those residing in the tissues, and rendering them liable to inflame; and *exciting*, or those which directly awake the process into activity. The first may be thought of as furnishing inflammable material; the second may be regarded as sparks of fire.

*Predisposing causes* are those which impair the general vigor, injure the blood, weaken the tissues, or lower nutritive activities. Among these causes are shock, hemorrhage, nervous irritation, gout, rheumatism, diabetes, Bright's disease, alcoholism, and syphilis. Plethora renders a person liable to sthenic inflammations (those characterized by high action). Tissue debility renders one prone to adynamic or asthenic inflammations. Nerve injury predisposes to inflammation, either from damage to trophic nerves and consequent failure in tissue nutrition and resistance or because analgesia exists and irritants which reach the region are not recognized and are allowed to remain. For instance, if the conjunctiva is in a condition of analgesia, the presence of foreign bodies is not noticed and destructive inflammation may result from their non-removal.

After removal of the Gasserian ganglion the cornea is devoid of sensation, the flow of tears is lessened, dust gathers in the eye, and if not removed by irrigation or kept out by a shield, inflammation and disastrous ulceration will ensue.

*Exciting Causes.*—The exciting causes of inflammation are—*traumatic*, as blows and mechanical irritation; *chemical*, as the stings of insects, the rubefacient effects of mustard, venom of serpents, products of bacteria, ivy poison, etc.; *thermal*, heat and cold; *specific*, the micro-organisms, causing, for instance, tuberculous peritonitis or erysipelas; and *nervous*, nerve stimulation certainly being capable of producing hyperemia and sometimes even inflammation. Inflammation due to nerve stimulation is seen in herpes zoster and in the swollen and discolored skin over an inflamed joint (Adami). Inflammation may also be induced by electric currents, by the *x*-rays, by radium rays, and by the actinic rays of sunlight and of electric light.

Some writers insist that every inflammation is due to the action of micro-organisms, but this statement lacks proof. They maintain that inflammation is a destructive microbic process which cannot bring about repair, and that repair begins only when inflammation ends. As Adami points out, the advocates of this view argue that swelling, pain, and discoloration point to the existence of inflammation; that repair can take place when these phenomena are absent, hence inflammation is not present when repair begins. As a matter of fact, swelling, discoloration, and pain are phenomena often but not invariably associated with inflammation; and in inflammation one or all of these phenomena may be absent. Because these signs are not discovered is no proof that inflammation does not exist. I believe that inflammation is not always due to microbes and is not always a destructive process, but may be from the start conservative and reparative. It is the reaction of the tissue to injury and is the first step on the road to repair.[1]

**Symptoms of Acute Inflammation.**—Inflammation, if at all severe, announces its presence by symptoms which are both *local* and *constitutional*.

**Local Symptoms of Acute Inflammation.**—The most prominent local symptoms were known centuries ago to the famous Roman, Celsus, who stated them as "*rubor, calor cum tumore et dolore*"—redness and heat with swelling and pain. As set forth to-day, the local symptoms are: (1) heat; (2) pain; (3) discoloration; (4) swelling; (5) disordered function; and (6) muscular rigidity, which is noted in inflammation of certain regions and structures.

*Heat* is due to the passage of an increased quantity of blood through the damaged area and to the arrival at the surface of the body of warm blood from internal parts. Although an inflamed part may be, and usually is, warmer than the surrounding parts, its temperature is never greater than the temperature of the blood. This increase of heat is especially noticeable when we, for instance, touch an arm affected with erysipelas and contrast the sensation obtained with that obtained by placing the hand on the sound arm. The diseased arm feels much warmer to the examining hand than does the sound arm, but its temperature is not above the general body temperature. An extremity in health, as is well known, shows on the surface a temperature below that of the blood; in an inflamed state the temperature may nearly equal that of the blood. Heat is always present in inflammation of a superficial part. The surgeon examines for heat by placing his hand upon the suspected area and then placing the same hand upon a corresponding portion of the opposite side of the patient in order to note the contrast. If great accuracy is desired, a surface thermometer is used.

*Pain* is a constant and conspicuous symptom. It is due to stretching of or pressure upon nerves from exudate; to irritation of nerves; or to inflam-

[1] See Adami's masterly article in Allbutt's "System of Medicine."

mation of the nerves themselves, producing cellular changes. Pain is associated with *tenderness* (pain on pressure), it is aggravated by motion and by a dependent position of the part, and it varies in degree and in character. In serous membranes it is acute and lancinating, like dagger-thrusts; in connective tissue it is acute and throbbing; in large organs it is dull and heavy; in the bone it is gnawing or boring; in the skin and mucous membrane it is itching, burning, smarting, or stinging; in the urethra it is scalding; in the testicle it is sickening or nauseating; in the teeth it is throbbing; and in inflammation under dense fascia it is pulsatile. Pain in inflammation after presenting itself in one form may change in character. If a pain becomes markedly throbbing, suppuration may be anticipated. Pain does not always occur only at the seat of trouble, but may be felt also at some distant point. Usually there is also pain at the seat of disease. Sometimes no pain is complained of in that region. I have seen pain in the right sciatic region dependent upon a chronically inflamed appendix, no complaint having been made of pain in the abdomen. This is known as a "*sympathetic*" or *referred pain*, and is due to the fact that the area to which pain is referred receives its nerve-supply from the same spinal segment as does the inflamed area; in other words, there is a nervous communication between the inflamed part and a distant area. In many cases of sympathetic pain a nerve-trunk refers the sense of pain to its peripheral distribution, but sometimes pain is referred to an adjacent nerve, a distant nerve, or even, perhaps, to a nerve on the opposite side of the body. Tenderness, however, is detected at the seat of trouble, whether or not it exists at the seat of referred pain.

*Pain of hepatitis* is often felt in the right shoulder. Pain at the point of the right shoulder or in the shoulder-blade is felt also in gall-stones, cholecystitis, and in cancer of the liver. The pain arises in filaments of the pneumogastric from the hepatic plexus.

*Pain of coxalgia* is often felt on the inside of the knee, because the obturator nerve, which sends a branch to the ligamentum teres, also sends a branch to the interior and to the inner side of the knee-joint.

*Inflammation of an eye* with increased tension causes browache. *Inflammation of the anus, uterus, tubes,* or *ovaries* may cause sacral backache. *Pain of rectal inflammation* may be referred to the back of the sacrum, down the thighs, to the penis and to the perineum. I have seen pain in the heel as a symptom of rectal cancer. *Pain of inflammation of the sacro-iliac joint* may be referred to the sciatic nerve and its branches. *Inflammation of the prostate and neck of the bladder* causes pain in the head of the penis, and often pain in the lower abdomen and loin. *Inflammation of a testicle or epididymis* cause pain in the groin, and often also in the abdomen, back, and thighs. *Renal calculus and pyelitis* cause pain in and retraction of the testicle, and pain in the loin, groin, or thigh.

If the covering of an organ is involved, pain becomes more violent; for instance, hepatitis becomes much more painful when the perihepatic structures are attacked. Inflammation without pain is known as "latent" (as the inflammation of Peyer's patches in typhoid). The sudden disappearance of inflammatory pain, when not due to the administration of opiates, suggests the possibility of gangrene, because analgesia exists in gangrene. The characteristics of inflammatory pain are that it comes on gradually, has a fixed seat, is continuous, is attended by other inflammatory symptoms, and is increased by motion, by pressure, and by a dependent position of the part. If there be no tenderness in a part, the source of the pain is not local inflammation; but tenderness may exist when there is no local inflammation, as in an area to which pain is referred from a distant part. Pain of an inflammation which does not involve a nerve does not correspond to an exact nervous dis-

tribution. If pain corresponds exactly to the area of a nerve's distribution, the cause of it is acting on the nerve-trunk or on its roots. If the cutaneous surface is involved, the lightest touch causes pain. The surface may be extremely hyperesthetic even when it is not inflamed, the condition resulting from deep-seated or distant inflammation. Areas of hyperesthesia of the skin of the abdomen are noted in various visceral inflammations. Such hyperesthesia is due to referred impressions. If touching the skin produces no pain, but deep pressure does produce it, the deeper structures are the source. Pain in muscle and ligament is developed by motion; in muscle, by contraction, but not by passive movements with the muscle relaxed; in ligament pain is developed by active or passive movements which stretch the ligament. If, for example, a man with a stiff neck has pain on the right side of the back of his neck on voluntarily turning his face toward the left shoulder, but is without pain when his face is turned by the surgeon, who, conversely, induces pain by turning the patient's face far to the right, this condition indicates the trouble to be muscular. If, however, no pain arises on turning the face to the right, but it is manifest on turning the face actively or passively to the left, the pain is in those ligaments which stretch when the face is turned to the left.[1] In inflammation of the synovial membrane even gentle passive motion in any direction causes pain.

The pain of colic differs from that of inflammation. It is sudden in onset, intermits, recurs in paroxysms, and is relieved by pressure. The pain of inflammation is gradual in onset, is continuous, and is made worse by pressure. The pain of neuralgia is often preceded by cutaneous anesthesia of the skin of the part, is very paroxysmal, comes on suddenly, darts through recognized nerve-areas, the attack lasts some hours, and is apt to recur at a certain hour. It presents no general tenderness, as does inflammation, but there may be several points which are acutely sensitive to pressure (Valleix's *points douloureux*). The tender spots of Valleix are met with in *inveterate* neuralgia, and occur at points where nerves "pass from a deeper to a more superficial level, and particularly where they emerge from bony canals or pierce fibrous fasciæ."[2]

Pain is often of great value by calling attention to parts diseased; but it may be a terrible evil, racking the organism and even causing death. If pain continues long, it becomes in itself formidable: it prevents sleep, it destroys appetite, and it deteriorates the mind, and one of the surgeon's highest duties is to relieve it. The *physiognomy or expression of physical pain* presents the following characteristics: Heavy fulness about the eyes, dropping of the angles of the mouth, and the aspect of fatigue. The victim of pain may be restless, but in severe inflammation he is apt to assume some fixed posture. He may be anemic. There may be widespread tremor, muscular twitches, or muscular rigidity. The absence of the physiognomy of pain in a person who complains of great agony is a strong indication that the patient exaggerates the gravity of his sufferings or deliberately deceives.

*Discoloration* arises from determination of blood to the part; hence the more vascular the tissue, the greater the discoloration. A non-vascular tissue presents no discoloration, though we usually find discoloration adjacent in the zone of blood-vessels which furnish the tissue with nutriment. Discoloration in vascular tissue is most intense at the focus or center of the inflammation. Discoloration varies in tint and in character according to the tissue implicated and the nature of the inflammation. It may be circumscribed or diffuse. Arborescent redness means a distribution in dendritic lines. Linear discoloration signifies redness running in straight lines, as in phlebitis. Punctiform discoloration occurs in points, and is due to vascular rupture. Maculiform redness resembles an ecchymosis or blotch. Dusky discoloration points to suppuration.

[1] "Surgical Diagnosis," by A. Pearce Gould.
[2] Anstie, "Neuralgia and Diseases which Resemble It."

Inflammation of the throat and skin produces scarlet discoloration; inflammation of the sclerotic coat of the eye and of the fibrous coat of muscle produces lilac or bluish discoloration; inflammation of the iris produces brick-dust, grayish, or brown discoloration; erysipelas causes a yellowish-red discoloration; secondary syphilis causes a copper-hued discoloration; and tonsillitis causes a livid discoloration. A tuberculous ulcer is of a purple color on the edge. Gangrene is shown by a black discoloration. A scorbutic ulcer is surrounded by an area of violet color.

Redness as a sign of inflammation must be permanent and joined with other symptoms. Redness due to inflammation disappears on pressure, but returns when the pressure has been removed. If redness is due to staining of the surface by dye, pigmentation, or extravasation of blood, pressure will not blanch the spot. If on taking off pressure the redness of inflammation rapidly returns, the circulation is active; if, on the contrary, it very slowly reappears, the circulation is very sluggish and gangrene is threatened. Subcutaneous hemorrhage gives rise to a purple-red color which does not fade when subjected to pressure. Stains of the surface by dyes fail to disappear on pressure, are distributed over a considerable surface, show a hue which is uniform throughout, are obviously superficial, are not associated with other signs of inflammation, and can be washed away.

A. Pearce Gould, in his excellent little work upon "Surgical Diagnosis," tells us that the color of a hyperemic surface may furnish important information. Lividity may mean failure of the heart and lungs, or simply venous congestion in the part. In lividity from obstruction of the lungs or heart the color slowly returns after pressure has driven it out. In lividity due to local congestion the color quickly returns when pressure is released and the dilated veins are often distinctly visible. Of course, in a local trouble, when the circulation becomes impaired to such a degree that gangrene is threatened, the lividity fades very slowly on pressure and reappears very slowly on the release of pressure.

*Swelling* or *tumefaction* is due in small part to vascular distention, but chiefly to effusion and cell multiplication. The more loose cellular material a part contains, the more it swells; hence the eyelids, scrotum, vulva, tonsils, glottis, and conjunctivæ swell very greatly when inflamed. A swelling is soft or edematous when due to uncoagulated effusion; is brawny and doughy when due to coagulated effusion; is hard and elastic when produced by proliferating cells. Swelling may do good by unloading the vessels and acting like a blister or local bleeding, or it may do great harm by pressing upon the vessels and cutting off the blood-supply. Swelling of the conjunctiva, or chemosis, may cause sloughing of the cornea, and swelling of the prepuce may cause gangrene. A swelling may do harm by obstructing an aperture, as in edema of the glottis, when the larynx becomes blocked; or by compression of a normal channel, as in the swelling of the perineum when the urethra is compressed. The cutaneous surface over a swollen area may become covered with blisters or blebs. This condition is noted particularly after burns and fractures.

*Disordered function* is always present in inflammation. It may be manifested by *increased tenderness* or sensibility; a slight touch, it may be, producing torturing pain. This condition is called *hyperesthesia*. Parts almost or entirely destitute of feeling when healthy (as tendons, ligaments, and bones) become highly sensitive when inflamed. It may be manifested by *increased irritability*. In dysentery the colon repeatedly contracts and expels its contents; the stomach does likewise in gastritis; and the bladder acts similarly in cystitis. Spasmodic twitching of the eyelids occurs in conjunctivitis, and twitching of the muscles of a limb after fracture and amputation.

*Impairment of Special Function.*—In inflammation of the eye, when an attempt is made to look at objects the lids close spasmodically, and even a little light causes great pain and lacrimation (photophobia). In inflammation of the ear noises cause great suffering, and even when in a quiet room the patient has subjective buzzing and roaring in his ears (tinnitus aurium). In coryza the sense of smell, in glossitis the sense of taste, in dermatitis the sense of touch, and in laryngitis the voice may be lost. In inflammation of the brain the mind is disordered; in arthritis the joints can scarcely be moved; and in myositis it is difficult and painful to employ the muscles.

*Derangement of Secretions.*—In dermatitis the sweat is not thrown off; in hepatitis bile is not properly secreted; and in nephritis urea is not satisfactorily removed. The secretions may undergo important changes of composition. The sputum in pneumonia is rusty, and dysentery causes a discharge of bloody mucus.

*Derangement of Absorbents.*—In the height of an inflammation the absorbents are blocked and clogged by coagulated exudate, and they cannot perform their offices.

*Muscular rigidity* is sometimes an important sign of inflammation. If a joint is inflamed, the muscles which move the joint are rigid and the joint is more or less immobile. In inflammation of the peritoneum the abdominal muscles are rigid, and the respirations become shallow, frequent, and thoracic. In pleuritis the intercostal muscles of the inflamed side become rigid and the respiratory excursion of the chest is limited. Rigidity serves to lessen motion, prevent pain, protect the part, and so gives physiological rest.

**Constitutional Symptoms of Acute Inflammation.**—The chief constitutional symptoms of acute inflammation are elevated temperature and leukocytosis. Constitutional symptoms may be absent, and often are in moderate or limited inflammations; but in severe, extensive, or infective inflammations the symptom group known as *fever* is certain to exist. This is known as *symptomatic*, or *inflammatory fever*, and it arises in non-septic cases from the absorption of aseptic pyrogenous exudate, and in microbic inflammations from the absorption of pyrogenous toxic products of bacterial action. In young and robust individuals an acute non-microbic inflammation causes a fever characterized by full, strong pulse, flushed face, coated tongue, dry skin, nausea, constipation, and possible acute delirium (the *sthenic type* of the older authors). In broken-down and exhausted individuals an ordinary inflammation, and in any individual a bacterial inflammation, may cause a fever with typhoid symptoms (the *typhoid*, *asthenic*, or *adynamic type*). Fibrin ferment is obtained from the white corpuscles; it is liberated as the corpuscles break up in the exudate, and acting on the liquor sanguinis cause the union of calcium and fibrinogen and the formation of fibrin. The absorption of fibrin ferment many believe causes aseptic fever (see page 129). Inflammatory blood contains an increased amount of albumin and salts. If a person with inflammatory fever is bled, the blood coagulates rapidly, the clot sinks, and there is found on the surface a cup-shaped coat, made up of liquor sanguinis and white cells, known as the *"buffy coat"*; but this is not really a sign of inflammation, and occurs normally in the blood of the horse. The buffy coat forms when blood contains a great number of leukocytes, because these leukocytes sink more slowly than do the red corpuscles. Cupping occurs because the white corpuscles sink more slowly by the side of the tube than far from the sides.

*Leukocytosis.*—In many inflammatory and infectious diseases leukocytosis is noted. It probably indicates an attempt on the part of the organism to protect itself from noxious materials. Leukocytosis is usually much more marked if pus exists than if the exudation is serous or fibrinous.

"The degree of leukocytosis may be considered a general index to the in-

tensity of the infection and to the strength of the individual's resisting powers in reacting against it. It follows, therefore, that intense infections occurring in individuals whose resisting powers are strong, produce a decided increase; but the presence of an infection of like intensity in one whose resisting powers are greatly crippled fails to cause leukocytosis, for in such an instance the organism is so overpowered by the effects of the morbid process that it is incapable of reacting" ("Clinical Hematology," by J. C. DaCosta, Jr.). We see from the above that gangrene or any other virulent infection may be accompanied by a low leukocyte count, and when pus is surrounded by a thick wall the leukocytes may be normal or nearly normal in number. An increased proportion of polymorphonuclear leukocytes strongly suggests body reaction against infection, and an increase of eosinophiles aids us in recognizing deep-seated pus when the leukocyte count is normal or but slightly increased.

The introduction of salt solution into the peritoneal cavity leads to the gathering of numbers of white cells, and the resistance of the serous membrane to infection is increased. Horse-serum that has been boiled is said by Petit to be a valuable material to draw polynuclear leukocytes to a part ("Med. Record," June 22, 1907). It has been injected into the peritoneal cavity the evening before an operation (30 c.c.); it has been poured into the cavity at the termination of an operation; the gauze used for drainage after an appendicitis operation has been soaked in it.

There is no fixed number of leukocytes which causes us to affirm the presence or absence of gangrene or pus. Most serious inflammations show marked inflammatory leukocytosis and an increase in the relative proportion of polynuclear cells. Typhoid shows no leukocytosis and even a mixed infection shows comparatively little increase of polynuclear cells. The same is true of tuberculosis. The same man should make all the counts on one patient; at least five hundred cells should be counted and several examinations ought to be made.

**Chronic Inflammation.**—This condition results from the action on the tissues of some mild but long-acting irritant. It progresses slowly and does not produce symptoms of severity either in the part or the body at large.

*Causes.*—Blood diseases, as rheumatism and gout; infective diseases, as tuberculosis and syphilis; retained pus in an ill-drained abscess; blocking of the duct of a gland; the retention of a foreign body in a part; the flow of an irritant secretion (as saliva from a fistula); repeated identical traumatisms of an occupation, etc. W. Watson Cheyne tells us that chronic inflammation is not due to the ordinary pyogenic organisms (see Cheyne's article on Treves's "System of Surgery").

*Tissue Changes.*—These changes are practically the same as in acute inflammation, but take place far less rapidly. Vascular dilatation, exudation, and leukocytic migration are often trivial. Cell proliferation is always conspicuously marked. It is maintained by Cheyne and others that typical granulation tissue does not form, the tissues of the part being replaced directly by fibrous tissue. The amount of fibrous tissue produced is relatively very great. This tissue may cause permanent thickening, or may contract and thus diminish the size of a part. Contraction is very considerable in cirrhosis of the liver and in interstitial nephritis.

*Symptoms.*—Pain varying in intensity and character; tenderness; great swelling, which in some cases is followed by shrinking, and is usually indurated or brawny. As a matter of fact, great swelling is the most usual symptom. Sometimes there is a trivial amount of heat. There is rarely discoloration unless the skin is itself inflamed, but usually the surface veins are distinctly, and sometimes they are greatly, distended. There are no constitutional symptoms attributable purely to the inflammation. If there are such symp-

toms, they are due to the disease which induced the inflammation or to interference with the function of an organ because of the fibrous mass. (For the treatment of chronic inflammation see articles upon special regions and particular structures.)

**Treatment of Acute Inflammation.**—The first rule in treating an inflammation must be to remove the exciting cause. If this cause is a splinter in the part, take out the splinter; if it is a foreign body in the eye, remove the foreign body; if urine is extravasated, open and drain; take off pressure from a corn; pull out an ingrown nail; and remove microbes from an infected area by draining, irrigating, and perhaps by applying antiseptics. The rule, remove the cause, applies to a chronic as well as to an acute inflammation. If the cause of an inflammation was momentary in action (as a blow), we cannot remove it, for it has already ceased to exist. After removing the cause, endeavor to bring about a cure by local and constitutional treatment.

**Local Treatment of Inflammation.**—It must be remembered that the division of inflammation into stages is natural, and not artificial, and that a remedy which does good in one stage may do harm in another. Certain agents are suited to all stages of an acute inflammation, namely, *rest* and *elevation*. In many inflammatory conditions nature seeks to immobilize, protect, and rest the part by increasing the tension of adjacent muscles. By this muscular rigidity inflamed joints are fixed and rested. Rigidity of the intercostal muscles in pleuritis limits chest motion and pain; rigidity of the abdominal muscles in peritonitis limits abdominal movements and lessens suffering.

*Rest.*—Physiological rest is of infinite importance, and is always indicated in acute inflammation. In the exercise of function blood is taken to a part and an existing inflammation is aggravated. Further, as Billroth has pointed out, rest prevents the dissemination of infection, because motion exposes fresh surfaces to inoculation and breaks down protective barriers of leukocytes. Its principles were first thoroughly studied by Hilton.[1] Baron Larrey, the celebrated military surgeon of the Napoleonic Empire, anticipated many modern views on this subject. He insisted on the necessity of rest in the treatment of wounds; he believed that rest permitted Nature to perform her work unhampered; he was accustomed to leave a "first dressing," if properly applied, undisturbed for several or even for many days. He believed it advisable to associate with rest well adjusted and judicious compression made by bandages, especially flannel bandages. (The author, on Baron Larrey, in "Johns Hopkins Hospital Bulletin," July, 1906.) The means of securing rest differ with the structure or the part diseased. When rest is used, do not employ it too long. *Rest in bed* diminishes the amount of blood sent to an inflamed part and lessens the force of the circulation; hence it antagonizes stasis. It has been shown that the heart beats at least fifteen times per minute less when the patient is recumbent than when he is erect. The saving of strength and the benefit of the local condition are thus seen to be enormous. In fact, the heart saves at least twenty-one thousand beats a day. In every severe inflammation insist on the patient going to bed.

In *cerebral concussion* rest must be secured by quiet, by darkness, by the avoidance of stimulants and meat, by the application of ice to the head, and by the use of purgatives to prevent reflex disturbance and the circulation of poisons in the blood. In *inflamed joints* rest must be obtained by proper position, associated in many cases with the adjustment of splints or plaster of Paris, or the employment of extension.

In *pleuritis* partial rest can be secured by strapping the affected side with adhesive plaster, or by using a bandage or a binder to limit respiratory movements. In *fractures* Nature procures rest by her splints—the *callus*—and

[1] "Lectures upon Rest and Pain."

the surgeon procures rest by his splints—firm dressings or extension. In *cancer of the rectum* and *intractable rectitis* a colostomy secures rest for the inflamed and damaged bowel. In *enteritis* opium gives rest to the bowel by stopping peristalsis. In *cystitis* rest is obtained by the administration of opium and belladonna, which paralyze the muscular fibers of the bladder. The use of the catheter gives rest to the bladder by removing urine. A cystostomy allows complete rest by permitting the bladder to suspend its function as a reservoir of urine. In cystitis from *vesical calculus* rest is obtained by incising the bladder, removing the stone, and draining, or by crushing and evacuating the stone. In *inflamed mucous membrane* rest from the contact of irritants is secured by touching the membrane with silver nitrate, which forms a protective coat of coagulated albumin. Opening an *abscess* gives its walls rest from tension. In *inflammations of the eye* light must be excluded to obtain complete rest, but tolerably satisfactory rest is given in some cases by the use of glasses of a peacock-blue tint. In *aneurysm* the operation of ligation cuts off the blood-current and gives rest to the sac. In *hernia* the operation gives rest from pressure. Instances of the value of rest could be multiplied indefinitely.

*Relaxation* is in reality a form of rest, and consists in placing the part in an easy position. In *synovitis of the knee* semiflexion of the knee-joint lessens the pain. In *muscular inflammation* relaxation relieves the pain.

*Elevation* partly restores circulatory equilibrium. A *felon* is less painful when the hand is held up in a sling than when it is dependent. A *congestive headache* is worse during recumbency. A *gouty inflammation* in the great toe is more painful with the foot lowered than when it is raised. A *toothache* becomes worse on lying down.

Certain agents are suited to the stage of vascular engorgement, increased arterial tension, and beginning effusion. These agents are—(1) local bleeding or depletion; (2) cold.

**Local Bleeding.**—Local bleeding, or *depletion*, is the abstraction of blood from the inflamed area. This abstraction relieves circulatory retardation and causes the blood to move rapidly onward. The corpuscles clinging to the vessel walls are washed away, the capillaries shrink to their natural size, and the exudate is absorbed. In other words, local blood-letting increases the rate of the circulation, though not its force.

The *methods of bleeding locally* are—(*a*) puncture; (*b*) scarification; (*c*) leeching; (*d*) cupping.

*Puncture* is recommended in inflammation, not only because it abstracts blood locally, but also because it gives an exit to effusion under fibrous membranes. It is very useful in relieving tension—for instance, in epididymitis. It is performed with a tenotome and with aseptic precautions. If numerous punctures are made, the procedure is termed "multiple puncture." This is very useful when applied to the inflamed area around a leg ulcer. The late Prof. Joseph Pancoast was very fond of employing multiple punctures, designating the operation "the antiphlogistic touch of the therapeutic knife."

*Scarification or Incision.*—By means of scarification we bleed locally, evacuate exudate, and relieve tension. One cut or many cuts may be made, and these cuts may be deep or may not go entirely through the skin, according to circumstances. Multiple incisions are useful when applied to inflamed ulcers, tissues in danger of gangrene, and to almost any condition of great tension. Scarification is of notable value when edema of the glottis exists. Free incision is of great benefit in periostitis and in threatened gangrene. In osteomyelitis the medullary canal must be promptly opened.

*Leeching.*—Leeches must not be applied to a region plentifully endowed with loose cellular tissue, as great swelling and discoloration are sure to ensue. These regions are the prepuce, labia majora, scrotum, and eyelids. Leeches

should never be applied to the face (because of the scar), near specific sores or inflammations, nor over a superficial artery, vein, or nerve. A leech is best applied at the periphery of an inflammation and between an inflammation and the heart. To leech at the inflammatory focus only aggravates the trouble. Before applying leeches, wash the part and shave it if hairy. Place the leech in a test-tube or an inverted wine-glass, inserting the tail or thick end first, and invert the tube so that the leech's head will come in contact with the prepared skin. The leech is restrained in the tube until it "takes hold" and begins to feed, when the tube is removed. If the leeches will not bite, smear the part with milk or a little blood. Never pull off a leech; let it drop off. It will usually drop off when full, but if it refuses to do so, sprinkle it with salt. After removing a leech, employ warm fomentations if continued bleeding is desired. Sometimes the bleeding persists or recurs. Weill and Mouriquand ("Press. Médicale," Paris, No. 1, 1911) report 6 cases of severe bleeding. Bleeding can usually be arrested by styptic cotton and pressure. In some rare cases the bleeding continues in spite of pressure. This is due to the fact that the tissue contains a considerable quantity of a material secreted from the throat of the leech, which material prevents coagulation of blood. In such a case excise the bite and the area of tissue adjacent to it, and suture the wound. Leeching leaves permanent triangular scars. The Swedish leech, which is preferred to the American, draws from 2 to 4 drams of blood. After a leech has been removed, if we desire to use it again, place it in salt water. This causes it to vomit the blood which it has taken up. Leeching has both a constitutional and a local effect. It is at present used comparatively rarely, but it is employed by some practitioners over the spermatic cord in epididymitis, on the temple in ocular inflammation, and over the right iliac region to relieve pain in mild cases of appendicitis.

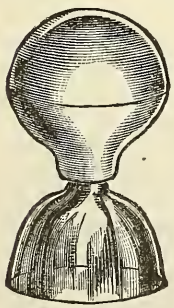


Fig. 49.—Rubber bulb cupper.

*Cupping.*—Dry cups deviate blood from a deeply placed inflamed area to the surface. Wet cups actually remove blood.

*Dry cups* are applied without first incising the skin. One or more may be applied. A special instrument is sold in the shops for the performance of dry cupping. It consists of a glass bell, with a globular and hollow top of rubber (Fig. 49). The rubber is emptied of air by squeezing. The glass bulb, the edges of which have been greased, is pushed upon the skin, and the compression is relaxed upon the rubber bulb. A partial vacuum is created, and an area of skin and subcutaneous tissue full of blood rises into the glass bell.

Cupping can be easily performed by means of a tumbler. The edge of the glass is greased; a bit of blotting-paper wet with alcohol is placed in the bottom of the tumbler and lighted. After a brief period the glass is inverted and placed upon the skin, which has been dampened with warm water. As the air in the glass cools the tissues rise into the partial vacuum.

*Wet cups* draw blood, and the skin should be cleansed before they are applied. In wet cupping apply a cup for a moment, remove it, incise or puncture the skin, and replace the cup to draw the requisite amount of blood. Incisions may be made by an ordinary scalpel, a lancet, or a scarificator, a cup being then applied. An excellent scarificator is shown in Fig. 50. In this instrument concealed blades are thrown out by touching a spring. Baron Heurteloup devised an instrument (Fig. 51) in which the incision is made by a scarificator. The blood is drawn out by a pump, the tube being placed upon the cut area and the withdrawal of the piston creating a vacuum. This instrument is known as the "artificial leech." After scarification and the application

of the cup, the partial vacuum draws blood into the cup; when the wounds cease to bleed the cup is removed, and if further bleeding is thought desirable, the clots are wiped away and the cup is again applied, and after its removal warm fomentations are used. Wet cupping is of value in pleuritis, pericarditis, and nephritis.

**Cold** is a very powerful and useful agent if used judiciously and applied at the proper time. It is valuable because of its reflex effect upon the vessels of the inflamed area rather than because of direct action upon the cells of a part. It should only be used early in the case, that is, before stasis occurs. It is not to be used in the later stages of inflammation, for it will then only aggravate the existing state; in fact, when there is considerable exudation cold does actual harm.

Cold acts by constricting the vessels of a hyperemic area, thus lessening the amount of blood sent to the part, and preventing the evolution of the process into the stage of stasis and exudation. Further, it prevents the migration of leukocytes, retards cell-proliferation, relieves pain and tension, and lowers temperature. If cold is too intense, if it is kept too long applied, if it is used too late in an inflammation, if it is used upon an old or feeble patient, or if it is employed when there is much exudation or a condition of tissue strangulation, it does actual harm. It lessens the nutritive activity of cells, constricts

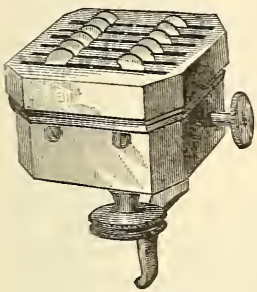


Fig. 50.—Scarificator.

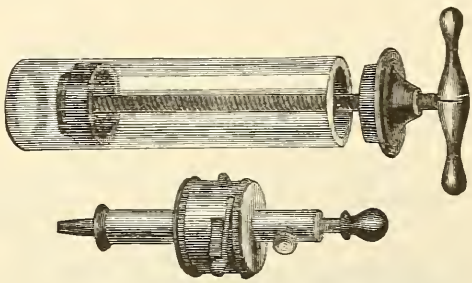


Fig. 51.—Heurteloup's artificial leech.

the lymph-spaces and channels, increases existing stasis, hence lowers the vitality of the tissues, and may cause gangrene. If the parts are constricted, as in strangulated hernia, or if they are compressed by a large exudate or fed by diseased blood-vessels, or if the patient is old or exhausted, cold is particularly apt to cause gangrene. Cold should not be used in a bacterial inflammation. In such an inflammation it is desirable that quantities of active leukocytes should come to the part. These phagocytes destroy bacteria and circumscribe the inflammatory focus. Cold keeps much blood and hence many leukocytes out of the part, lessens the ameboid activity, and prevents migration of the leukocytes which do succeed in arriving. Hence, cold actually favors the spread of a microbic process. Furthermore, it lessens leukocytosis and thus lessens the protective reaction of the tissues. De Nancrede, in his "Principles of Surgery," points out that in an inflammation stasis soon arises at the focus of the inflammation, and there is an area of stasis surrounded by a zone of hyperemia. Cold benefits the hyperemic zone, but aggravates the stasis. De Nancrede cautions us as follows: "Judgment is, therefore, requisite to decide whether the evil at the focus will not outweigh the good exerted at the periphery."[1] De Nancrede further points out that cold must not be used intermittently; but if employed at all, must be continuously applied. If cold is applied intermittently, there will be a reaction whenever it is removed, and this reac-

[1] "Principles of Surgery."

tion causes increased hyperemia. Hence, cold must be "continued in action to prevent reaction." If during the employment of cold the skin becomes purple and congested and the circulation feeble, at once discontinue the use of it, as its continuance will be dangerous.

Cold may be used as wet cold or as dry cold.

*Wet cold* is easily applied, but it is much more depressing than dry cold, is likely to produce discomfort, macerates the skin, and may lead to the formation of excoriations, etc. A part can be subjected to wet cold by the application of evaporating fluids or the use of a siphon. When wet cold is used inspect the part at frequent intervals, and discontinue the treatment if evidences of stasis become positive. Evaporating fluids are extensively employed. If such a fluid is used, never cover the part with a thick dressing. If this should be done, the fluid will not evaporate with sufficient rapidity to produce cold. A piece of thin muslin or flannel should be moistened with the fluid and laid upon the part, and be kept constantly moist by the application from time to time of small quantities of the liquid. Lead-water and laudanum is used extensively, and probably owes its chief value to the fact that it produces cold on evaporation. Lead-water and laudanum is composed of 1 oz. of laudanum, 1 oz. of liquor plumbi subacetatis, 1 pint of water. Liquor plumbi subacetatis dilutus may be used without laudanum. It is thought that the addition of laudanum tends to allay pain. Coplin demonstrated by a series of laboratory experiments that lead-water and laudanum is a germicide. A solution of ammonium chlorid may be used in the strength of 1 oz. of the drug to 2 quarts of water. If ammonium chlorid is used for more than a short period of time, it is prone to cause the formation of blisters, which are irritable and painful. Cheyne and Burghard use the following formula: ½ oz. of ammonium chlorid, 1 oz. of alcohol, and 7 oz. of water. Plain spring-water, iced water, or a mixture of alcohol and water may be used. The *siphon* is occasionally used. If there is a wound, the fluid which comes in contact with it must be aseptic or antiseptic. We may use sterile water, sterile salt solution, a solution of boric acid, or a solution of acetate of aluminum. In conjunctivitis, cold is applied to the eye by means of linen or muslin soaked in iced water, laid upon the closed lids, and changed frequently.

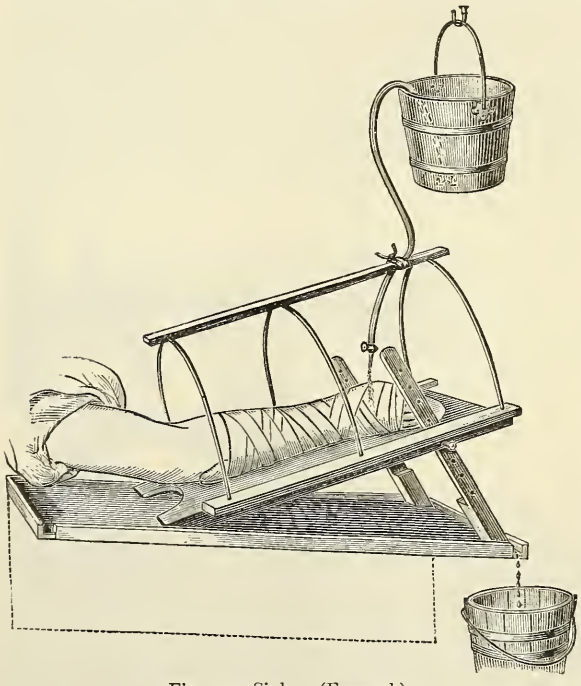


Fig. 52.—Siphon (Esmarch).

To apply wet cold by means of a siphon, the part is covered with one layer of wet linen or muslin, and is laid upon a rubber sheet folded like a trough and

emptying into a bucket. A vessel filled with cold water is placed upon a higher level than the bed. A wet lamp-wick is now taken, one end is inserted into the water of the vessel, and the other end is laid upon the part. Capillary action and gravity combine to keep the part moist. A rubber tube may be used instead of a wick. If a tube is employed, tie it in a knot or clamp it so that the fluid is delivered drop by drop (Fig. 52). Ordinary water or iced water can be used. If the water be too warm, it can be reduced to about 45° F. by adding 1 part of alcohol to every 4 parts of water. A mixture of 5 parts of nitrate of potassium, 5 parts of chlorid of ammonium, and 16 parts of water produces great cold.

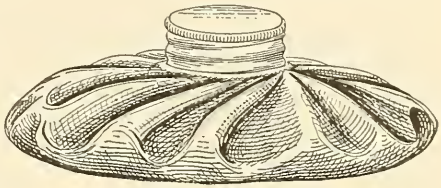


Fig. 53.—Ice-bag (W. E. Ashton).

*Dry cold* is more manageable and more generally useful than wet cold. It is applied by means of a rubber bag or a bladder filled with ground or finely cracked ice, several folds of flannel being first laid over the part. The flannel collects the moisture from the "sweating" bag and thus prevents maceration of the skin. Further, it saves the tissue from being subjected to too much direct cold and enables us to obtain the beneficial reflex effect. The ice-bag of India-rubber is widely used. We can venture to apply by means of the ice-bag a greater degree of cold than it is proper to apply by the use of fluids, as dry cold is not so likely to induce gangrene as is moist cold. If there is much tenderness, the weight of an ice-bag causes pain, and it is best to suspend it from a frame, so that it lightly touches the part. The frame is the same as is used to keep the bedclothes from contact with a fractured leg, and can be easily made from barrel-hoops. During the time an ice-bag is being used the part must be inspected at brief intervals to see that the circulation is not unduly depressed. The ice-bag is frequently used in joint inflammation, in intracerebral inflammation, in epididymitis, in acute myelitis, and by many in the earliest stage of appendicitis (see page 1016, where the author expresses his disapproval of such a method of treatment). If a joint is sprained, the immediate application of an ice-bag is of great service. A part can be encircled with a rubber tube through which iced water is made to flow (Fig. 54). Even when this apparatus is used the part should first be wrapped in flannel. Leiter's tubes, which are tubes of lead made to fit various regions and which carry a stream of cold water, can also be used. A piece of flannel must be placed between the tube and the skin. The temperature of these tubes can be lowered to any desired degree by lowering the temperature of the circulating fluid. Cheyne and Burghard wisely caution us to use a fluid at a temperature not

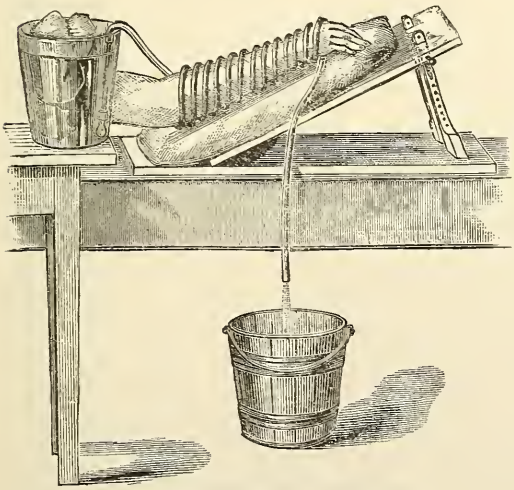


Fig. 54.—The Esmarch cooling coil.

under 50° or 60° F., to inspect the part every three or four hours, and not to employ the tubes longer than twenty-four hours.

*Heat* is employed by some early in an inflammation. It is rarely beneficial at this stage, except when applied by a hot-air apparatus for the treatment of an injured joint. It is true that a degree of heat which does not actually destroy the tissues will contract the vessels as does cold; but this degree of heat will not be borne by the patient unless but a limited portion of a superficial part is involved.

Certain agents are suited to the stage of fully developed inflammation, when there is a great deal of swelling due to effusion and cell-proliferation. The indication in this stage is to abate swelling by promoting absorption. This is accomplished by (1) compression; (2) local use of astringents and sorbefacients; (3) the douche; (4) massage; (5) heat.

**Compression** is especially beneficial in fully developed or in chronic inflammation, but it will do good even in the early stages. Compression is of great usefulness; it supports the vessels and causes them to drink up effusion, and it strongly rouses the absorbents. This agent is valuable in most external inflammations with marked swelling, and is particularly beneficial in chronic inflammation. In *erysipelas* of an extremity the part should be elevated and the extremity bandaged from the periphery to the body. In *ulcers*, especially those with hard and blue edges, the use of Martin's elastic bandage or of straps of adhesive plaster gives decided relief. In *chronic inflammation of a joint* elastic compression is of great value. In *epididymitis*, after the acute stage, the testicle may be strapped with adhesive plaster. In *lymphadenitis* compression by a weight or by a bandage is very generally employed. In *fractures* compression not only antagonizes spasm, but also combats the swelling and pain of inflammation. Compression must be judicious; it must never be forcible, and it must not be applied to a limb without including the distal portion of the extremity (never, for instance, strongly compress the elbow without including the hand, nor the palm without bandaging the fingers). Injudicious compression causes severe pain and great edema, and may produce gangrene.

**Astringents and Sorbefacients.**—Astringents may have direct value in inflammation of the skin, but it is not likely that they have any effect on deep-seated inflammation. When used in evaporating lotions in an earlier stage of inflammation the cold does good rather than the drug. Lead-water and laudanum is extensively employed and it is thought to somewhat allay inflammatory pain. The mixture certainly gives comfort in cutaneous erysipelas. It is very doubtful if lead-water is of any service at any stage of a deep-seated inflammation or in any fully developed inflammation. If used after the first stage it must not be applied as an evaporating lotion, because cold will do harm. Pieces of lint are soaked in the fluid and placed upon the part, and a bandage is applied. The wet lint which has been placed upon the part is covered with oiled silk or a rubber-dam before the bandage is applied. If used in the latter manner, the body-heat is retained in the part. If greater heat is required, a hot-water bag can be placed outside of the bandage. Lead-water, though germicidal, is seldom used in treating wounds, and hot lead-water should not be applied to an area of cutaneous inflammation, as heat increases congestion and does harm to a cutaneous inflammation.

*Saturated watery solution of Epsom salt* is of real value in inflammation. It is applied as a wet compress covered with rubber-dam. It is moistened every two or three hours and renewed in twenty-four hours, the skin being washed at the time of renewal. In many cases it allays pain and abates swelling. Its use was suggested by Tucker ("Jour. Experimental Med.," May 25, 1907).

*Tincture of iodin* is astringent, sorbefacient, counterirritant, and germicidal. It must not be used pure. For application to adults it should be diluted with an equal amount of alcohol, and for children with 3 parts of alcohol. In using iodin, paint it upon the part with a camels'-hair brush and fan it dry, applying one or more coats. The repeated application of iodin to the skin is of great benefit in inflammation of the glands, muscles, tendons, joints, and periosteum. Iodin is apt, after a time, to vesicate, and must not be used in full strength, because it is irritant. It is of special value in chronic inflammation. In deep-seated inflammation it acts as a counterirritant.

*Nitrate of silver* is a non-irritating astringent of considerable value in inflammation of mucous membranes. It forms a protective coat of coagulated albumin, and is much used in treating the throat, mouth, and genital organs. In urethral inflammation a protein compound of silver known as protargol may be used.

*Ichthyol* is a drug of decided efficiency in reducing inflammatory swelling. It is usually employed in ointments, the strength being from 25 to 50 per cent. It is best exhibited with lanolin. When rubbed in over inflamed glands, joints, and lymphatic enlargements, it is of great value. In children a 25 per cent., and in adults a 50 per cent., ointment should be rubbed in thoroughly twice a day. In inflammatory skin disease, synovitis, thecitis, frost-bite, bubo, chilblain, and in many other conditions, acute or chronic, the use of ichthyol is indicated. The odor of ichthyol is highly disagreeable, and when ordered for a refined person it had better be deodorized. For this purpose Hare uses oil of citronella, 20 minims to 1 oz. of ointment.

*Mercurials.*—Blue ointment, pure or diluted to various strengths, is extremely valuable. It is spread upon lint and kept applied over areas of fully developed inflammation. It is especially useful in acutely or chronically inflamed joints, glands, tendons, etc. Blue ointment is strongly irritant, and will soon blister or excoriate a tender skin. It is very beneficial in periostitis, and is employed by many surgeons in chronic inflammations.

The **douche** consists of a stream of water falling upon a part from a height. The water may be poured from a receptacle or may run through a tube, and may be either hot or cold. Alternating hot and cold streams are very popular in inflammations of joints and tendons, especially in chronic inflammation. This mode of application is known as the "Scotch douche." It restores the tone of the blood-vessels and plasma-channels and promotes the absorption of inflammatory exudate. If the part is very tender, the water should be squeezed upon it from sponges. In a sprain of a joint, after a time, when thickening has occurred, pour upon the part daily, from a height, first a pitcherful of very warm water, then a pitcherful of very cold water; then dry the part and use friction with a hand greased with cosmolin. Hot vaginal douches are generally employed in pelvic inflammations.

**Massage** is a procedure not employed frequently enough. It is very useful in some acute inflammations, though in these it must be gentle. It is of great service in the treatment of sprains of joints and fractures of bones. It is influential for good in chronic inflammations at the period when rest is abandoned. It acts by promoting the movements of tissue-fluids (blood, lymph, and areolar fluid), stimulating the absorbents, strengthening local nervous control, and thus improving nutrition. Passive motion in joints acts as massage.

**Heat** may be used continuously or intermittently, and may be either moist or dry. A considerable degree of heat will act like cold and contract the vessels. The degree necessary to cause vascular contraction would not destroy the tissue, but would produce discomfort, which discomfort would become unbearable during the continuance of the application. Therefore,

heat is rarely used in the earliest stage of an acute inflammation. It is hard to state exactly when heat should be substituted for cold. Certainly when retardation and stasis are manifest it is to be preferred. Moderate heat should be used when inflammation is not very superficial. In a cutaneous inflammation heat usually does harm, because it increases the congestion of an inflamed superficial part. In deep-seated inflammations heat to the surface acts as a revulsive or counterirritant. Thus a poultice to the chest may do good in the first stage of pneumonia, and cauterization of the skin near a joint may benefit an acute synovitis. The use of heat for purposes of counterirritation will be discussed under the head of Counterirritants. A moderate degree of heat applied over a fully developed and not too superficial inflamed area dilates the vessels, especially the veins, of the skin and superficial tissues. Thus circulation is re-established in an area filled with stagnant blood or blood which is scarcely moving and the inflamed region is drained, fluid exudate is absorbed, tension is lessened, the lymph-spaces and vessels distend, and lymphatic absorption becomes active. The application of heat increases the ameboid activity and the migratory tendency of the leukocytes, phagocytes gather in great numbers and surround an area of infection, and those which have taken up bacteria or tissue débris hurry away. Heat also, in all probability, causes antibodies to escape from the leukocytes and enter the blood-serum. Furthermore, heat favors leukocytosis. Thus, we see, that heat tends to help the building of protective barriers about an area of infection and aids the protective reactions of the body. Heat notably lessens the pain of inflammation. It is often used purely to relieve pain.

The *forms of heat* are—(1) fomentations; (2) poultices; (3) water-bath; (4) dry heat.

*Fomentation* is the application to the skin of a piece of flannel containing a hot liquid. A basin is warmed and over the top of the basin a towel is placed. A piece of flannel folded in two or three thicknesses is laid upon the towel and boiling water is poured upon it. By twisting the towel the water is squeezed out of the flannel. Great care must be taken to squeeze the water thoroughly out of the flannel, otherwise the skin may be scalded. The hot flannel is laid upon the skin over the disordered part. A rubber-dam larger than the flannel is placed over it, a mass of cotton is laid upon the rubber-dam, and a bandage is applied. The fomentation must be changed within an hour unless a hot-water bag has been placed outside the bandage, in which case it need not be changed for two hours or more. The flannel which is dipped into the hot liquid is known as a "stupe." The turpentine stupe is made by wringing out the flannel as above and then putting upon it from 10 to 20 drops of turpentine. Instead of fomenting the part, steam may be thrown upon it. Fomentations are used chiefly for their reflex influence over deep congestions or inflammations. The liquid of a fomentation may, if desired, contain corrosive sublimate, carbolic acid, or other agents. A fomentation containing an antiseptic is known as an antiseptic fomentation. An *antiseptic fomentation* or, as it is often called, an *antiseptic poultice* is made and applied as follows: Gauze is used instead of flannel, and is laid upon the towel over the basin as previously described. A very warm solution of corrosive sublimate (1:1000) of concentrated boric acid solution, or a 2 per cent. solution of acetate of aluminum, is poured upon the gauze, the material is partly wrung out, placed upon the part, covered with a rubber-dam, and upon it a hot-water bag is placed. Fomentations are very useful in relieving pain in any stage of an inflammation and act also as counterirritants. Fomentations are used in preference to ordinary poultices if there is any probability of a surgical operation becoming necessary, because skin to which an old-fashioned poultice has been applied cannot be satisfactorily sterilized. The antiseptic fomentation is of great service in

removing sloughs from foul wounds and ulcers. It is the only form of poultice which is admissible when the skin is broken.

*Poultice or Cataplasm.*—A poultice is a soft mass applied to a part to bring heat and moisture to bear upon it. Poultices can be made of ground flaxseed, of slippery-elm bark, of arrowroot, starch, bread and milk, potatoes, turnips, etc. The poultice should be placed upon the part and be covered outside with oiled silk, a rubber-dam, or waxed paper. A mass of cotton is applied outside of the rubber and the poultice is held in place by a bandage or binder. It can be kept very warm for a considerable period by placing upon it a bag filled with hot water. If a hot-water bag is not employed, a poultice should be changed every two hours. Spongiopilin, when moistened with hot water, is a good substitute poultice. Lint soaked with hot water and covered with some impermeable material does very well. A poultice containing opium is known as a "sedative" poultice. About 2 gr. of opium to the ounce of poultice-mass may relieve pain. Flaxseed is a vegetable material, adheres to the skin, enters the mouths of glands and follicles, undergoes decay, can be removed only with great difficulty, and is a very objectionable material to use if there is any breach of surface continuity or if it is possible that an incision will be required. The preparation of an antiseptic *poultice* or fomentation is described above. Poultices must not be kept on the part too long, as they will cause vesication, especially in adynamic conditions. If a poultice is causing vesication, remove it and do not replace it, or replace it after sprinkling the part and the poultice with powdered oxid of zinc. If suppuration exists or is seriously threatened, do not waste time by using poultices, but incise at once. Incision may prevent suppuration by relieving tension, affording drainage, and permitting the local use of antiseptics. If pus exists, it cannot be evacuated too soon. To use poultices and delay incision is often productive of irreparable harm. After incision of a purulent focus it is common practice to apply an antiseptic fomentation in order to draw quantities of leukocytes to the part and thus limit the spread of infection and stimulate granulation.

*Hot-water Bath.*—The continuous warm bath is now rarely employed except in burns and cases of phagedena, when it often proves curative. In these cases an antiseptic agent may be dissolved in the water. Continuous immersion in a warm bath is regarded favorably by some surgeons for the treatment of sloughing wounds and large purulent areas. The immersion of a part from time to time in water as hot as can be tolerated is useful in fully developed and in chronic inflammation. Such immersion benefits an inflamed joint, lessening the pain, swelling, and stiffness.

*Dry heat* is applied by a metallic object dipped in hot water and laid upon the part; by Leiter's tubes, through which hot water flows; by the hot-water bag or by the hot-air apparatus. Some surgeons use the hot-water bag in cases of mild appendicitis in order to favor the limitation of the area of infection. The hot-water bag is often soothing and beneficial when laid upon an inflamed joint, or on the perineum or the hypogastric region in cystitis. A bag of hot sand, a hot brick, or a bottle or can of hot water may be used instead of the water-bag. The hot-air apparatus is of very great service in the treatment of chronic inflammation, and particularly of inflamed joints (*vide* dry hot-air apparatus).

*Treatment When Suppuration is Threatened.*—When suppuration is threatened, ordinarily hot fomentations or antiseptic fomentations must be used, and the part must be kept at rest. As previously explained, the flaxseed poultice is inadmissible. When suppuration is threatened, the use of heat causes the collection of multitudes of leukocytes, which tend to limit the area of infection and destroy bacteria. Even when suppuration is not prevented, heat aids in the rapid breaking down of the diseased tissue at the focus of

the inflammation and causes hordes of leukocytes to gather and encompass the suppurating tissue, and these leukocytes prevent the spread of the infection.

In most cases, when suppuration is obviously inevitable or seriously threatened, a *free incision* will be of greatest benefit.

**Irritants and Counterirritants in Inflammation.**—*Irritants* attract an increased supply of blood to the part whereon they are applied, and are used for their local effects. *Counterirritants* are used to affect by reflex influence some distant part. In chronic inflammation irritants may do good by promoting the blood-supply, thus favoring the removal of exudates (liniment for rheumatism and synovitis, and nitrate of silver for ulcers). Counterirritants are powerful pain-relievers when used over an inflamed structure; they bring blood to the surface, and are thought by many writers to cause anemia of internal parts, the site and area of anemia depending on the site, the area, and the duration of the surface irritation. Some recent studies seem to suggest that counterirritation produces hyperemia of the superficial part, compensatory anemia of surrounding regions, and anemic edema of the subcutaneous tissue and muscles (W. Wecksberg, "Zeit. f. klin. Med.," Bd. xxxvii, H. 3 u. 4). Nancrede dissents from the statement that counterirritants cause anemia of internal parts; and he maintains that they irritate deeper parts and cause more external blood to be taken to them. He claims that a blister applied to the chest produces a hyperemic area in the pleura, and refers to Furneaux Jordan's opinion that direct irritation to the surface over a joint adds to synovial hyperemia, and that consequently in joint inflammation counterirritants should be applied above and below a joint, but not directly over it. As a matter of fact, we know clinically that powerful counterirritation directly over an inflamed superficial joint is occasionally followed by an aggravation of the trouble, and that in pericarditis blistering directly over the pericardium may, as pointed out by Brunton, make the condition worse. Counterirritants not only relieve pain in the earlier stages of inflammation, but they also promote absorption of exudate in the later stages, and are particularly valuable in chronic inflammations. Great benefit is obtained by blistering old thickened ulcers, and by painting the chest with iodin to relieve pleuritic effusion. Frictions, besides their pressure effects, act as counterirritants. Frictions may relieve skin pain, and are associated with the application of stimulating liniments in the treatment of stiff joints. A mustard plaster is a valuable counterirritant in an acute deeply seated inflammation. Tincture of iodin is extensively used in chronic inflammation.

There is no more efficient method of relieving pleural effusion than by the application of a succession of blisters. Blisters are also used in the treatment of inflamed joints, pericarditis, pneumonic consolidation of the lung, acute and chronic rheumatism, etc.; and are applied back of the ears or at the nape of the neck in congestive coma or meningitis. A blister can be produced in a few minutes by soaking a bit of lint in chloroform, and after applying it to the surface, covering it with oiled silk or with a watch-glass. Equal parts of lard and ammonia will blister in five minutes. It is easier to blister with cantharidal collodion or blistering paper. Before applying a blister, shave the part if it be hairy; then grease the plaster with olive oil and apply it. Blistering plaster is left in place six hours in the case of an adult, but only two hours in the case of an old person or a child; the plaster is then removed, and if a blister has not formed, the part must be poulticed for a few hours. When a blister is obtained, open it with a needle which has been dipped in boiling water. If the surgeon wishes the blister to heal, it should be covered with a piece of lint smeared with cosmolin or with zinc ointment. If it is to be kept open for a time, cut away the stratum corneum and dress with cosmolin, each ounce of which contains 6 drops of nitric acid.

Pustulation can be effected with tartar-emetic ointment or with Vienna paste. Tartar-emetic ointment was formerly used on the scalp in meningitis. Vienna paste consists of 5 parts of caustic potash and 6 parts of lime made into a paste with alcohol. It is applied for five minutes, and is then washed off with vinegar.

The actual cautery is the most powerful of counterirritants. It is chiefly used in chronic inflammation of joints, bone, nerves, and the spinal cord. The application is, of course, very painful, and it is best to give an anesthetic before using the cautery. The Paquelin cautery is the instrument used. This is a hollow platinum point which, after being heated in the flame of an alcohol lamp, is kept hot by forcing through it the vapor of gasolene (Fig. 226). The point is used at a white heat. One area or several may be seared. The cautery is drawn lightly two or three times over each spot we wish to burn. The object is to destroy only the superficial layers of the skin. After the cauterization is completed, lint wet with iced water is applied for several hours to allay pain, and then hot antiseptic fomentations are used until the slough separates.

If the wish is to prevent healing after separation of the slough, dress the sore with cosmolin, each ounce of which contains 6 drops of nitric acid. It is not wise to cauterize deeply directly over a superficial joint.

**Constitutional Treatment of Inflammation.**—Certain remedies are used in inflammation for their general or constitutional effects; these remedies are—(1) general bleeding; (2) arterial sedatives; (3) cathartics; (4) diaphoretics; (5) diuretics; (6) anodynes; (7) antipyretics; (8) emetics; (9) mercury and iodids; (10) stimulants; (11) tonics.

**General Bleeding, Venesection, or Phlebotomy.**—Venesection is suited to the early stages of an acute inflammation in a young and robust subject. The indication for its employment is increased arterial tension, as shown by a strong, full, rapid, and incompressible pulse in a vigorous young patient. General blood-letting diminishes blood-pressure and increases the speed of the blood-current, thus amends stasis, causes the absorption of exudate, and the washing of adherent white corpuscles from the vessel wall; furthermore, it reduces the whole amount of body blood and thus forces a greater rapidity of circulation, decreases the amount of fibrin and albumin, lowers the temperature, arrests cell-proliferation, and stops effusion.

This procedure was in former days so highly esteemed that it settled into a routine formula to be applied to every condition from yellow fever to dislocation. The terrible mortality of the cholera epidemics from 1830 to 1835 led practitioners to question the belief that bleeding was a general panacea, and from this doubt there was born in the next generation violent opposition to blood-letting in any disease. Like most reactions, opposition has gone too far, the pendulum of condemnation has swung beyond the line of truth and sense, and thus is universally neglected or broadly condemned a powerful and valuable resource. Many physicians of long experience have never seen a person bled; its performance is not demonstrated in most schools, and but few patients and families will permit it to be done; but when properly used it is occasionally beneficial. It is applicable, however, only to the young, strong, and robust, and not to the old, weak, or feeble. It is used for violent acute inflammations of important organs or tissues, and not for low inflammations or for slight affections of unimportant parts. It is used in the early, but not in the late, stages of an inflammation. It is used when the pulse is frequent, full, hard, and incompressible, but not when it is slow, small, soft, compressible, and irregular. It is used when the face is flushed, but not when it is pallid. It is not used in fat persons, drunkards, very nervous people, or the sufferers from adynamic, septic, or epidemic diseases. It is of value in some few cases of congestion of the lungs, pneumonitis, pleuritis, meningitis,

prostatitis, cystitis, and other acute inflammatory conditions. It is particularly valuable in any subject when uremia exists, or when there is distention of the right side of the heart. The method of bleeding is described on page 462.

After bleeding the patient should be put on arterial sedatives, diuretics, diaphoretics, anodynes, and, if necessary, purgatives. A favorite mixture of Prof. S. D. Gross was the antimonial and saline, 40 gr. of Epsom salt, $\frac{1}{10}$ gr. of tartar emetic, 2 drops of tincture of aconite, and 1 dram of sweet spirits of niter, in enough ginger syrup and water to make $\frac{1}{2}$ oz.; given every four hours.

**Arterial Sedatives.**—Drugs of this character are of great use before stasis is pronounced; but if used after stasis is established they will increase it. If stasis exists it may be relieved by blood-letting, local or general, and then arterial sedatives can be given. Either local bleeding or venesection abolishes stasis and lowers tension, and arterial sedatives maintain the effect and hold the ground which is gained. The arterial sedatives employed are aconite, veratrum viride, gelsemium, and tartar emetic. These sedatives lessen the force and the frequency of the heart-beats, and thus slow and soften the pulse, and are suited to a robust person with an acute inflammation, but are not suited to a weak individual in an adynamic state.

*Aconite* is given in small doses, never in large amounts. One drop of the tincture in a little water is given every half hour until its effect is manifest on the pulse, when it may be given every two or three hours. Large doses of aconite produce pronounced depression, and are dangerous. Aconite lowers the temperature, slows the pulse, and produces diaphoresis.

*Veratrum viride* is a powerful agent to slow the pulse and to lower blood-pressure; it produces moisture of the skin, and often nausea. It is given in 1-drop doses of the tincture every half hour until its physiologic effects are manifested, when the period between doses is extended to two or three hours. Ten drops of laudanum given a quarter of an hour before each dose of veratrum viride will prevent nausea.

*Gelsemium* is an arterial sedative. It is given in doses of 5 to 10 drops of the tincture every three or four hours.

*Tartar emetic* lowers arterial tension and lessens the pulse-rate. This drug is not generally employed; if it is used with the greatest care it is no better than some other agents, and if it is not so used it will cause dangerous depression. The dose is from $\frac{1}{20}$ to $\frac{1}{10}$ gr., given in water every three hours until the physiological effects are manifest.

**Cathartics.**—Purgation is of great value in inflammation. By it putrid material is removed from the intestine, fluid containing poisonous elements is drawn from the blood, and the liability to infection of the tissues is lessened. Purgation is a powerful aid in removing serous effusions and other exudates. The administration of purgatives is, of course, not to be a routine procedure in inflammatory states. The bowels may be acting so freely that no cathartic is required. Treatment in an inflammation should be inaugurated, if constipation exists, by giving a cathartic. The tongue affords important indications as to the necessity for purgation. Castor oil can be given in capsules, or in the froth of beer, or the juice of half a lemon is squeezed into a tumbler, 1 oz. of oil poured in, and the rest of the lemon is squeezed on top, thus making a not unpalatable mixture. Aloin, podophyllum, the salines, and calomel in 3- or 5-gr. doses, followed by a saline, have their advocates. In threatened peritonitis the salines are used by some surgeons, a teaspoonful of Epsom salt and a teaspoonful of Rochelle salt being given hourly until a movement occurs. In this condition, however, purging may prove disastrous. In the course of inflammation, from time to time, if there be constipation, a coated tongue, and foulness of the breath, there should be ordered 1 gr. of calomel with 24 gr. of bicarbonate of sodium, made into twelve powders, one being given

every hour; if the bowels are not moved by the time the powders are all taken, a saline should be given. If a violent purgative effect is desired, as in meningitis, croton oil or elaterium may be ordered. If constipation is persistent, give fluidextract of cascara sagrada daily (20 to 40 drops), or a pill at night containing $\frac{1}{4}$ gr. of extract of belladonna, $\frac{1}{4}$ gr. of extract of nux vomica, $\frac{1}{10}$ gr. of aloin, $\frac{1}{4}$ gr. of extract of physostigma, and $\frac{1}{2}$ drop of oil of cajuput. Enemas or clysters may be used in some cases. A very useful enema is composed of 1 fl.oz. of oil of turpentine, 1½ fl.oz. of olive oil, ½ fl.oz. of mucilage of acacia, in 10 fl.oz. of water. Soap and turpentine is very satisfactory. Soapsuds and vinegar in equal parts make a serviceable clyster. A combination of oil of turpentine, castor oil, the yolk of an egg, and water can be used. Asafetida, 30 gr. to the yolk of 1 egg, makes a good enema to amend flatulence. An ounce of alum in 1 quart of water is valuable for the same purpose.

**Diaphoretics.**—These agents are very useful. A profuse sweat removes much toxic material from the blood and in the beginning of an acute inflammation, such as tonsillitis, may abort the disease. Dover's powder is commonly used, but pilocarpin is preferred by some. Camphor in doses of from 1 to 5 gr. is diaphoretic, and so are antimony and ipecac. Acetate and citrate of ammonium, opium, alcohol, hot drinks, heat to the surface (baths, hot bricks, hot-water bags), serpentaria, and guaiac are diaphoretic agents.

**Diuretics** are useful in fevers when the urine is scanty and high-colored, and are valuable aids in removing serous effusions and other exudates. Among the diuretics may be mentioned calomel in repeated large doses, cocain, alcohol, infusion of digitalis, the nitrites, squill, turpentine, copaiba, and cantharides. The liquor potassæ and the acetate of potassium are the best agents to increase the solids in the urine. The liquor potassii citratis in doses of 1 to 4 fl.dr. is efficient. Large drafts of water wash out the kidneys. If the heart is weak, citrate of caffein is a good stimulant diuretic, and hot coffee is very serviceable in promoting the secretion of urine. The injection of hot salt solution into the rectum and under the skin favors diuresis, and the intravenous infusion of salt solution is a very powerful diuretic (see page 465). The application of heat to the loins promotes the secretion of urine. Sodiotheobromin salicylate (diuretin) is an uncertain but often valuable diuretic, in doses of 10 gr., every two or three hours.

**Anodynes and Hypnotics.**—Drugs may be required to allay pain or procure sleep. Dover's powder, besides being diaphoretic, is anodyne. Opium acts well after bleeding or purgation. If it causes nausea, it should be preceded one hour by the administration of 30 gr. of bromid of potassium. Opium is given by the mouth or by the rectum. Morphin is given by the mouth or hypodermatically. Opium is used when there is pain, but its use is not to be long persisted in if it can be avoided. It is given in doses measured purely by the necessities of the case. If opium disagrees, try the combination of morphin with atropin. After an operation antipyrin or phenacetin will often quiet pain and secure sleep. When a person feels "so tired he can't sleep," alcohol in the form of whisky or brandy must be given. Sleeplessness not due to pain is met by chloral, trional, veronal, the bromids, or sulphonal. Chloral is dangerous in conditions of weak heart or exhaustion. Bromids must be given in large doses to be efficient. Sulphonal must be given about four or five hours before sleep is expected, in doses of from 10 to 20 gr. in hot milk or hot mint-water. Trional is safe and very satisfactory. It is given in doses of 15 to 25 gr. in hot water.

**Antipyretics.**—Arterial sedatives, diaphoretics, and purgatives lower temperature, and have previously been alluded to (see page 106). There are two great classes of febrifuges—those which lessen heat-production and those which increase heat-elimination. In the first group we find quinin, salicylic acid and the salicylates, kairin, alcohol, antimony, aconite, digitalis, cupping,

and bleeding. In the second group we find alcohol, nitrous ether, antipyrin, acetanilid, phenacetin, opium, ipecac, cold to the surface, and cold drinks. In surgical inflammations it is rarely necessary to employ heroic means to lower temperature. Quinin is but a feeble antipyretic for non-malarial fevers, and that it shall be one at all requires a dose of 20 gr. or more. Salicylic acid is not advisable unless there is hyperpyrexia or unless the patient has acute rheumatism. If 30 or 40 minims of guaiacol are painted on the skin of the abdomen, it will cause a notable but brief drop in a febrile temperature. After a short period of lowered temperature there is commonly a chill and a rapid rise. Cardiac depression may arise after giving an antipyretic, and such an agent as antipyrin is dangerous to the weak and adynamic. In truth, all of the coal-tar derivatives are dangerous when used as antipyretics. As a matter of fact, fever is a condition in which the animal organism is endeavoring to oxidize and render inert certain poisonous materials, and antipyretic drugs lessen oxidation and actually make the patient worse. It is a suggestive fact that bacteria are said to multiply more rapidly when kept at about the normal body temperature than when kept at fever heat (102° F. or more). The mere discomfort of fever may be much mitigated by antipyretic drugs, but the fever process is not benefited by them. No attempt should be made to lower temperature by cold or antipyretic drugs, unless with the high temperature there are the nervous phenomena of hyperpyrexia.

**Emetics** may do good when the patient suffers from a parched, coated tongue, a dry and hot skin, nausea, and gastric oppression, but it is very rarely in these days that we employ them.

**Mercury and the Iodids.**—Mercury is an alterative, that is, an agent which favorably affects body nutrition without causing any recognizable change in the fluids or the solids of the body. Mercury lessens blood plasticity, hinders the exudation of liquor sanguinis—thus furnishing less food to the cells in the perivascular tissues—and retards cell-proliferation. Further, by a stimulant action on the absorbents it promotes the breaking up of an existing inflammatory exudation, and hence limits damage from excess of new formation. The time at which mercury is best given is when violent symptoms have abated, the guides being a reduced temperature and a moist skin. Mercury is often given in conjunction with the local use of sorbefacients (ichthyol or mercurial ointment). When possible, the administration of mercury is associated with compression of the inflamed part. Mercury is sometimes given until the gums are slightly touched, but it is not given to the point of salivation. When the breath becomes offensive and the gums tender on snapping the teeth, or when griping and diarrhea begin, the dose should be reduced or the drug should be stopped (see Ptyalism). In iritis mercury is used to get rid of the plastic effusion which is causing pupillary fixation and opacity. In keratitis the gums should be touched *slightly*. In orchitis, after the subsidence of the acute symptoms, mercury should be employed. In pericarditis, meningitis, and in many chronic and lingering, and in all syphilitic inflammations, this drug can be used.

Some persons will be salivated by very minute doses of mercury, either because of idiosyncrasy or previous saturation. Others can take enormous doses without any appreciable constitutional effect. The action of mercurials can be favored by a combination with ipecac or with tartar emetic.

In giving mercury, if a prompt effect is desired, give 3 gr. of calomel every three hours until a metallic taste is noted in the mouth. If the case is not so urgent, gray powder is a good combination. Children are given calomel and sugar or mercury and chalk. If it is desired to give the drug for some time, corrosive sublimate is a suitable form, and small doses will actually increase the number of red blood-corpuscles. Corrosive sublimate is to be given alone

or combined only with iodid of potassium. The green iodid of mercury is a drug suitable for prolonged administration. During a prolonged course of mercury it will often be necessary to give at the same time a little opium to prevent diarrhea and griping. A rapid effect can be obtained by rubbing daily with a gloved hand 1 dr. of the oleate of mercury or ½ dr. of the ointment into the groins, the axillæ, or the inside of the thighs. Suppositories of mercurial ointment induce rapid ptyalism. Hypodermatic injections of corrosive sublimate or gray oil may be used, and must be thrown deeply into the muscles of the buttock or back. Old people, those who are exhausted, anemic, and broken down, and the tuberculous bear mercury badly. If it be given to them at all, it must only be in small amounts and for a brief time.

*Alkaline iodids* are useful in removing the products of inflammation; they can be given for a long time, and admirably supplement mercurials. Iodid of potassium can be prescribed in combination with corrosive sublimate as follows:

R. Hydrarg. chlor. corros. ..... gr. ij;
Potass. iodidi ..... ℈v et ℈j;
Syr. sarsaparillæ comp. ..... q. s. ad f℥viij.—M.
Sig.—Two fluidrams in water, after meals.

Iodid of potassium, well diluted, is given on a full stomach; it is never given concentrated or before meals. A convenient mode of administration is to procure a concentrated solution of the iodid of potassium, remembering that every drop equals about 1 gr. of the drug, and give as many drops as may be desired in half a glass of water after meals. If the medicine disagrees, add to each dose, after it is put in water, 1 dr. of the aromatic spirit of ammonia. Extract of licorice is a good vehicle for the iodid. If the mixture in water disagrees, the drug should be given in milk. Capsules are satisfactory, but a drink of water should be taken just before and again just after taking a capsule, to protect the stomach from the concentrated drug. Iodid of sodium may agree when iodid of potassium does not. When the iodids disagree they produce *iodism*. The first indications of iodism are a bad taste in the mouth, running of the eyes and nose and sneezing, followed by a feeling of exhaustion, absolute loss of appetite, nausea, tremor, and skin eruptions (acne, hemorrhages, blebs, hydroa, etc.). If iodism occurs, stop the drug and give the patient Fowler's solution in increasing doses, laxatives, diuretic waters and also nutritious food, and stimulants if depression is great. Sometimes belladonna does good in obstinate cutaneous disorders induced by the iodids.

**Remedies Directed Against Special Morbid States.**—If inflammation is associated with rheumatism, gout, scurvy, syphilis, tuberculosis, or any other constitutional disease or predisposition, appropriate treatment should be instituted to control the disease or combat the predisposition, and at the same time the area of inflammation should be locally treated. Syphilis is treated by the internal use of mercury; in some cases the iodids are also given; scurvy, by vegetable juices and potash salts; rheumatism, by the alkalis or salicylates; gout, by colchicum or piperazin; tuberculosis, by the fats, tonics, and open-air life.

**Stimulants.**—The chief stimulants used are hot black coffee by the stomach or bowel; hot normal salt solution by the bowel, beneath the skin, or in a vein, alcohol by the mouth or rectum; and strychnin or atropin hypodermatically. The use of *alcoholic stimulants* is called for by conditions rather than by diseases, being indicated by the state of the patient rather than by the name of the malady. For a brief acute inflammation in a robust young person alcohol is not needed; but all who are weak or exhausted, be they young or old, all who are aged, those who are accustomed to alcoholic beverages, those

who have high temperature or failure of circulation, and those who labor under septic inflammations or adynamic processes require alcohol, and it should be given with a free hand. In an acute malady, a feeble, compressible, rapid, or irregular pulse, and great weakness of the first sound of the heart are indications that alcohol is required. Low, muttering delirium is a strong indication for stimulation. There is no *dose* of alcohol for these states; it is given for its effect. Two ounces of brandy or whisky may be needed in a day, or perhaps many ounces. If the breath of the patient smells strongly of the alcohol, he is getting too much. If delirium increases after each dose, alcohol is doing harm. Alcohol is contra-indicated in acute meningitis. In acute illness use whisky, brandy, champagne, or alcohol and water. During convalescence there may be used a little port, claret, or sherry wine, or malt liquor. These agents will promote appetite, digestion, and sleep.

*Strychnin* is a very valuable stimulant. It can be given in doses of $\frac{1}{30}$ to $\frac{1}{20}$ gr. three times a day, but after a few days seems to lose its stimulant effect.

*Atropin* is one of the best remedies for exhaustion of the vasomotor system. The dose is $\frac{1}{100}$ gr. hypodermatically.

**Tonics.**—The use of tonics is indicated during convalescence from acute and throughout the course of chronic inflammations. There may be used iron, quinin, and strychnin in the form of elixir; iron alone, as in the tincture of the chlorid; quinin in tonic doses (6 to 8 gr. daily); or Fowler's solution of arsenic. An excellent pill consists of—

R. Acid. arsenos ..... gr. j;
Strychnini ..... gr. ss;
Quinini ..... gr. xlviij;
Ferri reduct ..... gr. vj.—M.
Ft. in pil. No. xxiv.
Sig.—One after each meal.

Bitter tonics before meals improve the appetite. One of the best tonics is tincture of nux vomica in gradually increasing doses.

**Antiphlogistic Regimen.**—This term comprises the necessary directions relating to diet, ventilation, cleanliness, etc.

*Diet.*—When, in the early stages of an acute inflammation, the patient cannot eat, there must be administered a cathartic before food is given. Nausea is combated by calomel and soda, drop-doses of a 6 per cent. solution of cocain, iced champagne, iced brandy, chloroform-water, hot water, and counterirritation of the epigastric region. Sucking ice may check nausea, but it often makes the patient uncomfortable, because he sucks in air with the melted ice. Sucking ice does not quench thirst. When the process is depressive from the start, and in any case after the earliest stage, feeding is of vital moment. The great tissue waste calls for large quantities of nutritive material, but the impaired digestion demands that the food shall be easily assimilable; hence it is taken in liquid form, small quantities being frequently given. Albumin-water is an agreeable beverage of some nutritive value. Milk contains all the elements required by the body, and is the food of foods. If it disagrees, it should be mixed with lime-water, or to each dose an equal amount of Vichy or soda-water may be added. Peptonized milk is a valuable agent. Some people can take boiled milk who cannot take cold unboiled milk. Some patients, however, digest raw better than boiled milk. Peptonized milk has been to a great extent superseded by pancreatinized milk. It is given cold, either alone or mixed with a carbonated water. Koumiss is retained in some cases when the stomach rejects all other foods. This fermented milk is nutritious, stimulant, and very useful. One part of milk, 2 parts of cream, and 2 parts of lime-water make a nutritious and digestible mixture. Milk punch is largely used. Whey

may be used when plain milk cannot be taken. Eggs are highly nutritious, but are apt to disturb the stomach; they may be given as egg-nog, or simply soft-boiled, or the yolk can be beaten up in a cup of tea, or raw eggs may be given in sherry or brandy. When considerable nausea exists the yolk of an egg may be added to 1 oz. of lemon-juice and 2 dr. of sugar, the glass being filled with carbonated water. Beef-tea is certainly a stimulant, but it is not a food. It contains the excrementitious elements of the beef. It is prepared by cutting up 1 pound of lean beef, adding to it a quart of water, and then simmering, but not boiling, down to a pint, finally filtering and skimming the liquid. The dose is a wineglassful seasoned to taste. Beef juice is nutritious. It is prepared as follows: A thick and tender beefsteak is partly broiled over a hot fire, the outside is browned and the juice is retained within the meat. The steak is cut into pieces to fit a lemon squeezer or meat-press (the instrument having been warmed by previously dipping it in hot water). The juice is expressed by squeezing and may be given warm, seasoned with salt and pepper, or may be taken after it has been frozen. Fresh meat juice may be used plain or pancreatinized. The meat juices obtained in the shops have little nutritive value. Bouillon and beef extracts have slight nutritive value. Meat jellies (calf's foot being the one commonly used) have some though little nutritive value by producing a certain amount of energy which, were they not given, would of necessity be furnished by protein. Hence, meat jelly saves or spares protein (Bauer). Clam-juice and clam-broth are palatable and slightly nutritious. They are retained in many cases when any other food would be rejected. The broth is to be given hot and the juice either hot or cold. Coffee is a valuable stimulant in febrile conditions. When the stomach entirely rejects food day after day, nutritive enemata are given. There is dispute as to their value, because it is certain that the large intestine does not digest by juices of its own manufacture, and no protein matter can be absorbed without previous digestion. If undigested protein matter is introduced it undergoes putrefaction, causes irritation, and liberates toxins which are absorbed. It seems equally certain, however, that the large bowel does absorb water as part of its physiological duty and that it can absorb alcohol, saline fluid, grape-sugar, certain drugs, and perhaps digested albumin and fat. Undigested albumin and fat should never be given by enema. These materials should be pancreatinized before injection or should be mixed with pancreas and then injected, the peptones being formed in the bowel. Nutritive enemata are given at a temperature of 90° to 95° F. They should not be bulky (not over 7 or 8 oz.), because a large enema is usually quickly expelled. They should not be given oftener than three or perhaps four times a day, because too frequent administration irritates the rectum and enemas will not be retained by an irritated rectum. During the period that rectal enemata constitute the method of feeding the rectum should be washed out once a day by a high enema of warm salt solution; this cleansing enema is given one hour before a nutritive enema. A useful enema is Leube's meat and pancreas—3 oz. of pancreas and 8 oz. of meat are rubbed together by a pestle, tepid water is added, and the mixture is injected. It undergoes digestion in the large bowel. Bidwell's formula is as follows: 2 oz. of milk, 2 oz. of strong beef-tea, yolk of 1 egg, 1 dr. of pancreatic solution, prepared one hour before using and kept during the interim at a temperature of 100° F. Brandy can be added just before using. Enemata of salt solution greatly relieve thirst. When the sufferer feels able to eat a little, any good soup, strained and skimmed, should be ordered. As the patient gets better he may be fed on scraped meat, broth containing crumbs, tapioca with cream, custard, milk-toast, sweetbreads, chops, oysters, chicken, etc., until he gradually reaches ordinary diet.

The *temperature* should be taken at regular intervals, and the condition of the gastro-intestinal tract should be observed. The *urine* must be examined

at intervals, and the daily amount passed must be known. If insufficient urine is being passed, increase the amount of fluid, particularly of water, given by the mouth. If the urine is scanty and the patient is nauseated by drinking water, give enemata of hot saline fluid or employ hypodermoclysis. The *pulse* and *heart* must be frequently observed, and cardiac weakness must be combated by suitable stimulants.

*Ventilation and Cleanliness.*—The ventilation of the apartment is of the greatest importance. Every day the windows should be opened widely for a time, the patient, of course, being protected from chilling and kept out of a draft. When the windows are open the air of a room can be quickly changed by swinging the door to and fro. A constant access of fresh air must be secured, and the temperature kept as near as possible to 68° F. If high fever exists, the sick man must be cleaned and be sponged off with alcohol and water every day. It is important that the bed-clothing be clean and that the sheet be unwrinkled, as otherwise bed-sores may form.

**Treatment of Chronic Inflammation.**—The subject of chronic inflammation has been referred to previously. The local treatment comprises rest, relaxation, elevation, counterirritation, massage, passive movements, the douche, the application of sorbefacients, the use of compression, incision, and, perhaps, certain special methods, as the induction of passive hyperemia by Bier's method, or baking the part in a hot-air oven. The patient should be placed under proper hygienic and climatic conditions; the diet must be judiciously regulated; drugs are given symptomatically or to combat some constitutional tendency or disease (see articles upon Special Regions and Diseases).

**Bier's Hyperemic Treatment of Inflammation.**—Years ago Laennec asserted that cyanosis is antagonistic to tubercle. Rokitansky emphatically supported the contention that people with marked valvular disease of the heart are seldom attacked by pulmonary tuberculosis. Such lesions, of course, dam back the blood in the lungs. Farre and Travers point out the great liability to pulmonary tuberculosis of patients with anemic lungs because of stenosis of the pulmonary artery (Edward Adams, in "N. Y. Med. Jour.," February 26, 1910). The discovery of how to utilize this knowledge in treatment was made by Professor Bier, of Berlin. Bier believes that hyperemia in inflammation is a reaction on the part of the organism; that it is Nature's effort to remove an irritant and to supply increased nutritive material, hence that it is desirable and should not be combated by cold, but should be favored by every means in our power. Bier endeavors to increase the hyperemia of an inflamed region, or to produce hyperemia in an area of disease. He regards hyperemia as beneficial, stasis as harmful; hence he causes or increases hyperemia and combats stasis. Stasis lowers tissue resistance and may cause gangrene. The increase in the amount of moving blood in a part means an increase in the number of phagocytes and the amount of germicidal blood liquor. By the method of treatment recommended by Bier the surgeon induces venous, passive, or obstructive hyperemia by means of an elastic band or a cupping glass, active or arterial hyperemia by means of hot air.

**Obstructive Hyperemia by Means of the Elastic Bandage.**—The constrictor should be the soft broad bandage of an Esmarch apparatus. Figure 55 shows it applied around the arm. The bandage must not be so tight as to cut off the pulse at the wrist or to cause unpleasant sensations, pain, or very rapid distention of the subcutaneous veins (Meyer and Schmieden). When edematous swelling arises we may be unable to feel the pulse. Then our guide must be the sensations of the patient. If the treatment causes pain or increases existing pain, at once discontinue it (Waterhouse, "Brit. Med. Jour.," December 16, 1911). The part below the band should become bluish red and warm, but never white under the influence of the constriction. When the bandage is in

place an area of inflammation shows an increase of redness, heat and swelling, and a diminution of pain. If pain was not present before, the bandage must not produce it. Should it do so it is too tight. In chronic conditions the bandage is usually employed daily and for two, three, or four hours at a séance. When used for three hours or longer temporary edema may arise. In acute cases it is used for twelve, fifteen, or twenty hours a day. Prolonged application may make the skin sore unless a flannel bandage is applied before the band is used, unless the site of application is shifted daily, and unless the skin during each intermission is well rubbed with alcohol. The bandage is always applied well above the inflamed region, and that region is exposed free from dressings in order that its condition during treatment may be observed. If edema occurs, the band must be removed and the edema relieved by elevation and massage. If the inflammation is accompanied by marked edema, incisions are required. Treatment by the bandage may prevent pus formation. If pus forms it must, of course, be evacuated.

Fig. 55.—Shows elastic bandage in place around the arm, its ends tied with tapes which are attached to the bandage. This is the style of bandage usually found upon the market. If the bandage is to remain on for a number of hours, it is advisable to apply a strip of adhesive plaster, to guard against the tapes becoming undone. Note the engorgement of the subcutaneous veins of the forearm, showing the effect it is desired to produce by the bandage (Meyer and Schmieden).

**Obstructive Hyperemia by Means of the Cupping Glass.**—Endeavor to make the skin bluish red, but not white. Cupping glasses are used not only to treat areas of inflammation, but to aid in emptying sinuses and abscesses which have ruptured or have been incised. Figures 56–59 show cupping glasses.

I am satisfied from personal experience that Bier's method of treatment is of great value in acute inflammation as well as in chronic inflammation, and that it is not used as often as it should be. I have used it with success in several cases of ununited fracture. It is an improvement on the method of Thomas, of Liverpool, for inducing hyperemia about the ends of the fragments. He did it by repeated percussion with a mallet. Barker has recently advocated Bier's treatment for ununited fracture. I have never used it for purulent affections of large joints, a condition in which Bier finds it highly useful (Bier and Baetzner, in "Practitioner," Jan., 1912). He empties the joint with a trocar, washes it with carbolic solution, puts on dressing, but does not immobilize. Bier retains the band from twenty to twenty-two hours a day. As soon as acute symptoms subside he uses hot air. The reduction of the daily period of hyperemia is brought about gradually before substituting hot air. I have seen very gratifying results from the Bier treatment of gonorrheal joints. I believe that this treatment gives the best chance of cure without deformity and with

retained function. It is valuable for joint tuberculosis, although very numerous brief applications are necessary. It is beneficial for thecitis, areas of suppuration after incision, and chilblains. Waterhouse suggests its use to prevent

Fig. 56.—The simplest form of suction glass. The rubber bulb is attached directly to the glass. This glass is used in the treatment of furuncles of smaller size and sinuses (Meyer and Schmieden).

Fig. 57.—Illustrating an ordinary suction apparatus for the finger (felon, etc.) with a convexity at the lower surface, designed to receive the pus (Meyer and Schmieden).

suppuration in a crushed limb. The treatment is contra-indicated in spreading inflammation—for instance, erysipelas.

Bier claims that arterial hyperemia induced by hot air is particularly useful in chronic inflammation, as it favors the absorption of exudates and of

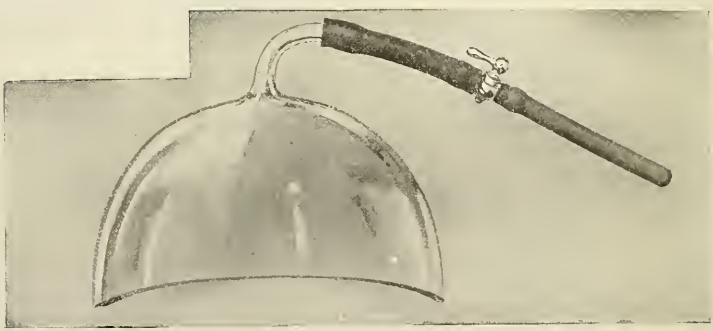


Fig. 58.—Shows glass of simpler configuration; a rubber tube connects glass with bulb; the same can be readily detached, thus rendering easy the sterilization of the glass by boiling. In the tube a three-way stop-cock is inserted. This cup is used for treating furuncles of larger dimensions, etc. (Meyer and Schmieden).

adhesions. It will hasten the separation of sequestra. Venous hyperemia produced by the elastic band or the cupping glass is claimed to be of great value in infections. It may abolish the infection, prevent suppuration, and hasten the process to a conclusion. It does certainly lessen pain and favor

absorption. The elastic band may be used upon an extremity, a testicle, the scrotum, and the head. In other regions cupping glasses are used, a partial vacuum being established in the glass by means of a pump or a rubber bulb.

**Sir Almroth Wright's Views Upon Inflammation and Its Treatment.**—Wright maintains that a free supply of blood and lymph is necessary for repair, that both the blood and lymph should contain a sufficiency of protective materials, and that it is essential that numerous active leukocytes enter the area of disease.

When there is a large serous effusion and few leukocytes, a condition met with often in tuberculous pleurisy, repair does not take place.

In abscess the leukocytes are dead and the material obtained from dead leukocytes retards healing. If the fluid exudate does not contain protective material the process extends.

Fig. 59.—Constructed for the treatment of the hand. A soft-rubber band wound around the cuff makes it fit air-tight around the arm (Meyer and Schmieden).

Repair is retarded by induration, or by the formation of a fistula or a sinus.

If there is a small amount of fluid exudate, there is little protective substance thrown into the inflamed part and repair is retarded.

If there be a large effusion and few leukocytes, cure is favored by removing the fluid. This is done in abscess and in serous effusion. If there is too little lymph in the part salt solution and citric acid should be used locally, and citric acid should be administered internally. The administration of proper bacterial vaccines will increase the protective qualities of the lymph. (See Wright, "The Pathology of Inflammation," and the résumé of his views in "Progressive Medicine," Sept. 1, 1908, p. 31.)

## IV. REPAIR

A damaged tissue reacts to the injury and Nature attempts to effect repair. It is held by many that inflammation is a destructive process and repair is a constructive process; that repair is constantly effected in an aseptic wound without many of the evidences of inflammation; that repair does not proceed from inflammation, but is retarded or prevented if inflammation occurs. As before stated, we agree with Adami, that inflammation is reaction to injury and the effort of Nature to repair the injury. As Adami points out, the attempt to repair may fail, the reaction to injury being excessive or not powerful enough; but even should the attempt fail, the conservative intention exists. "What is the development of cicatricial tissue but an attempt at repair? What other meaning can be ascribed to the increased bactericidal power of the inflammatory exudate as compared with that of ordinary lymph and blood-serum? Why do leukocytes accumulate in a region of injury? Why do some of them incorporate bacteria and irritant particles, and others bring about the destruction of these without necessarily

ingesting them? All these are means whereby irritants are antagonized or removed, and reparation and return to the normal sought after."[1]

Repair is favored by good general health, asepsis of the wound, coaptation of wound edges, and rest. It is retarded or prevented by infection, gaping of the wound, frequent or forcible motion, and impairment of the general health.

*Albuminuria* and *diabetes* particularly obstruct repair. R. T. Morris points out that sugar in the blood is hygroscopic, removes water from the tissues, and thus obstructs repair; and also that the wound fluids contain sugar and are good culture-media ("Med. News," June 29, 1901).

**Healing By First Intention.**—A wound may "heal by first intention." This mode of healing, which is known as "primary union," occurs without suppuration, and is observed in the healing of an aseptic wound. If infection occurs, primary union will not take place. The phrase "by first intention" comes down to us from the past. It was properly thought that Nature intends to repair a wound, and first intention signifies the first, best, the most desirable way in which it can be accomplished. In a small aseptic incision, in which no considerable vessels are cut, repair will take place very rapidly after the edges have been approximated and the wound dressed. In fact, the wound edges may be held firmly together in twenty-four hours. In such a wound a small amount of blood flows from the capillaries between the edges of the wound, and this blood clots. A trivial amount of exudate and some few migrated corpuscles pass into the clot and into the tissues. The fixed connective-tissue cells and the endothelial cells of the vessels multiply, and form embryonic or juvenile tissue. The cells are epitheloid cells and are known as fibroblasts. The fibroblasts eat up many of the leukocytes and multiply, so that the new cells from one side of the wound finally interlace with the new cells from the other side. Nearby capillaries become irregular in outline; at certain points bulging occurs, and at these points new capillaries develop, extend into the mass of fibroblasts, and join new capillaries of the opposite side. The reparative material is now said to be organized; it has become granulation tissue. The fibroblasts become spindle shaped and develop into interlacing fibers (Fig. 60). The tissue is now fibrous tissue; it contracts strongly, and finally most of the capillaries are obliterated by pressure. In such a slight wound the reaction to injury is chiefly noted in the cells of the part, and the vessels and leukocytes play but a small part in repair.

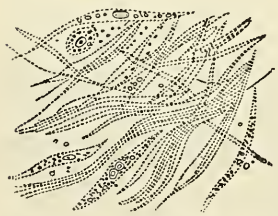


Fig. 60.—Cells developing into fibers (Bennett).

The exudation is so scanty that there is practically no swelling unless it arises from venous obstruction. The vessels are so slightly affected that there is no redness. The final step in healing is contraction of the fibrous tissue and the covering of the surface with epithelium, which springs from the epithelial cells upon the edges. This final process is called "cicatrization," and consists in the formation from fibroblasts of new fibrous tissue and the contraction of the new tissue. The "immediate union" of some writers never occurs. This term means the union of microscopic parts to their counterparts without any effort at repair. A first union is effected always by clotted blood and coagulated exudate, next by proliferating cells, and finally by fibrous tissue. A wound healing by first intention exhibits no evidence of inflammation. There is some slight tenderness, but no actual pain. A certain amount of swelling arises because of exudation of fluid from the blood, and the coagulation of this fluid makes the wound edges hard. Venous obstruction leads in some cases to a considerable fluid swelling. A wound may heal by first intention even though some

[1] Adami, in Allbutt's "System of Medicine."

bacteria are present, if the part has a good blood-supply and the patient is in good health. Active leukocytes and germicidal blood liquor may prevent infection. In a more extensive incised wound many vessels are cut. After oozing ceases the vessels are closed by clots continuous with the clot between the sides of the wound. An exudation of plasma from the blood-vessels and of lymph from the lymph-spaces takes place. Leukocytes in great numbers invade the wound edges and the exudate, and the exudate clots. Thus, an infection may be surrounded and limited. This mass of blood-clot, plasma-clot, and leukocytes used to be known as "coagulable lymph." The leukocytes actively eat up the clot, and by the end of the third day occupy the space formerly occupied by the clot. Embryonic tissue is formed by multiplication of the fixed connective-tissue cells and endothelial cells. These cells are called fibroblasts. They multiply and grow into the mass of leukocytes, eating up many of the leukocytes, and finally join the fibroblasts of the other side of the wound. Some leukocytes enter into the lymph-spaces. New capillaries form from the capillaries at the wound margins. By the end of the first week the fibroblasts begin to assume various outlines, sending out poles or branches or becoming spindle shaped. These spindle-shaped cells become fibers, and the fibers of the new tissue interlace and strongly contract. Thus the edges are pulled firmly together. Finally, new epithelium, derived from epithelium at the wound edges, forms and grows over the wound (Figs. 61–63). In order to obtain primary union the surgeon ought to cleanse the wound, and all his procedures must be thoroughly aseptic and bleeding should be carefully arrested. The parts are then accurately coaptated by sutures, aseptic or antiseptic dressings are applied, and special care is taken to secure *rest*. In a large wound special methods to secure drainage are required (page 76). In a small wound the spaces between the stitches give exit to the trivial quantity of wound fluid. The use of irritant germicides in a wound greatly increases the amount of discharge and renders necessary the introduction of material for drainage. Even a comparatively small wound requires drainage for the first twenty-four hours. During the first twenty-four hours after a large wound begins to heal by first intention the discharge of bloody serum is most plentiful, but after this period it becomes very scanty and soon ceases entirely, and can be much diminished in quantity on the first day by the application of pressure. Warren says that after a hip-joint amputation over a pint of bloody serum flows out during the first twenty-four hours. In an aseptic wound, as a rule, one-half of the stitches are removed on the sixth or seventh day and the remainder on the eighth day, but for two weeks more the wound should be rested and supported, as the new tissue is not very resistant to infection. *Aseptic fever* always arises when much exudation is poured out and it is slowly and imperfectly drained. Aseptic fever is due to the absorption of aseptic pyrogenous material (see page 129). If an incised wound becomes infected, the pyogenic organisms, by liquefying the intercellular substance, destroy the bond of union which is forming between the wound edges. As a consequence, the wound edges are soon widely separated by pus.

What used to be known as "*healing by blood-clot*" is healing by first intention. If there is a considerable gap between the edges of an aseptic wound, and the gap is filled with a blood-clot, healing goes on in the same manner as when the gap is narrow, although more corpuscles, more exudate, and more fibroblasts are required to effect repair.

**Healing By Second Intention.**—Healing of a wound in which there is a large cavity in the tissue or in which the edges have gaped apart is known as healing by granulation, or "healing by second intention." It is called healing by granulation because the granulations (areas of vascularized embryonic tissue) are visible. It is effected in the same manner as "healing by first

intention," the processes in the two cases being practically identical if pus is absent. As a matter of fact, in healing by granulation there is usually wound infection. As a result of infection intercellular substance is peptonized, many reparative cells are cast off, and repair can be effected only after the formation

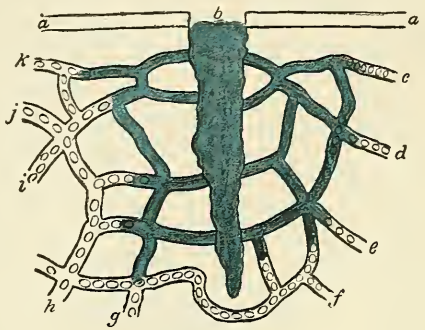



Fig. 61.

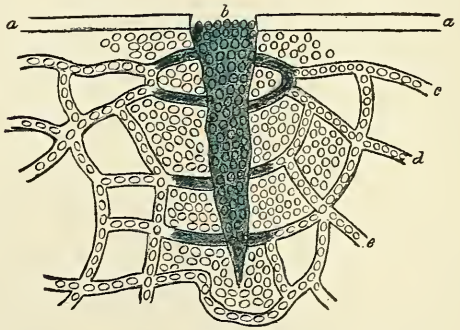



Fig. 62.

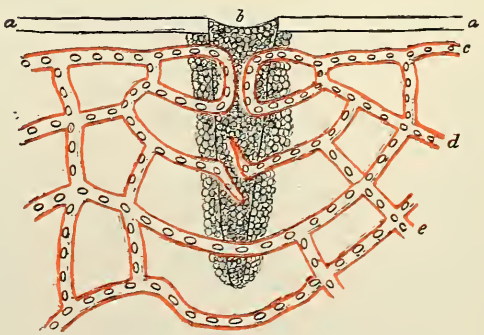



Fig. 63.

Figs. 61–63.—Healing by first intention: *a*, Skin; *b*, fibroblasts; *c*, *d*, *e*, capillaries. Fig. 61, Clot in the vessels continuous with clot between the edges of the wound. Fig. 62, Migration of leukocytes into the perivascular tissues and into the clot between the edges of the wound. Fig. 63, Formation of new capillaries (after Pick).

of enormous numbers of fibroblasts and the expenditure of considerable time. It requires much longer for an infected wound to heal than for an incised wound to be repaired, and an infected wound can heal only by granulation. A short time after the infliction of a wound the oozing ceases, because thrombi form in

the vessels and clot gathers in tissue-gaps and interstices. Exudation begins and leukocytes migrate into the exudate and into the walls of the wound. In an hour or two the surface of the wound becomes distinctly glazed or glistening, because of the formation and coagulation of fibrin. The exudation is at first thin and red, and it soon becomes so profuse as to wash away the discolored fibrin coat. Usually, in a few days the discharge becomes purulent. The connective-tissue cells, especially the endothelial cells of the vessels, proliferate and form fibroblasts, and the fibroblasts multiply to close the wound. From adjacent capillaries new capillaries form. This formation takes place as follows: A portion of a capillary thickens and a whip-like process comes off from the thickened part. This process fuses with a second filament budded from another or from the same capillary, or runs straight out as a terminal vessel. The filaments after a time are hollowed out from within, protoplasmic tubes are formed, and endothelial cells develop from the protoplasm. In some cases a tubular prolongation comes off from a capillary directly. Figures 63 and 64 show the formation of a capillary. In a wound healing by granula-

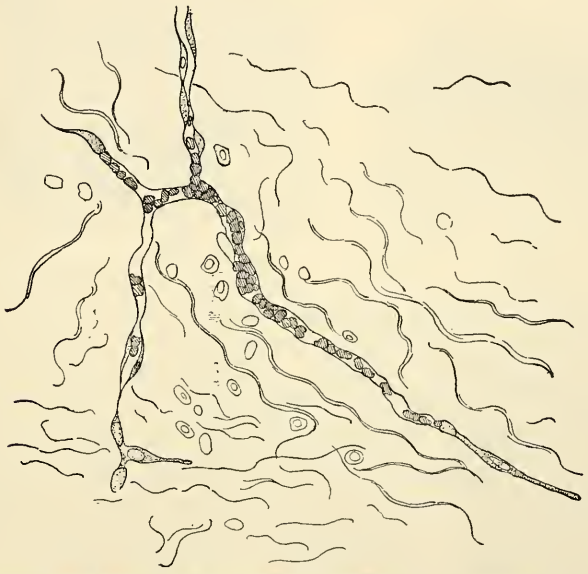


Fig. 64.—Development of a blood-vessel in mesentery of an embryo (Warren).

tion these newly formed capillaries run among the fibroblasts, and some of them run perpendicularly to the surface, or a loop forms and reaches the surface. The surface of a granulating wound is covered with migrated leukocytes, and directly under these are fibroblasts covering the new vascular strings or loops. Vascular strings or loops coated with fibroblasts are called granulations (Fig. 65 shows a granulating surface). When the discharge becomes purulent, many leukocytes and fibroblasts are destroyed, inflammation increases, exudation becomes profuse, and cellular multiplication widespread and rapid in order to make up for the cells lost by microbic action. Gradually the gap is filled. As it is being filled the older fibroblasts in the deeper layers of the edges and base of the wound are converted into *cicatricial*, *fibrous*, or *scar tissue* (Fig. 66). As the granulations rise to a higher level at the surface the area of fibrous tissue becomes broader at the base and margins, and this young fibrous tissue contracts. By contracting it draws the edges of the wound nearer together, and thus lessens the area of the surface which must be covered with epithelium.

When the granulations reach the level of the cutaneous surface the epithelial cells at the margin of the wound proliferate, and young epithelial cells, constituting a bluish or opalescent film, grow over the granulations. Epithelium comes only from epithelium. Granulations are never converted into epithelium. The epithelial covering comes only from the epithelium at the wound margins, unless there be epithelial remains in the wound; for instance, an undestroyed papilla, sweat-duct, or hair-follicle. The process of covering the surface with epithelium is known as *epidermization*. The epidermization of a large area always consumes considerable time and sometimes Nature fails to accomplish it. In such cases skin-grafting is employed (*q. v.*). Before, during, and for a time after epidermization the fibrous tissue of the walls and base of the wound contracts. Thus the wound margins are pulled and held nearer together, the gap to be bridged is diminished in size, the danger of tearing apart of the epithelial layer is lessened, many capillaries are destroyed by pressure, and

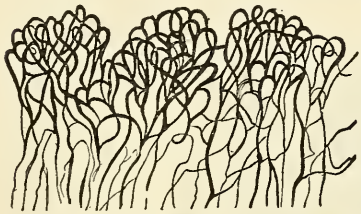


Fig. 65.—Blood-vessels in granulation (Gross).

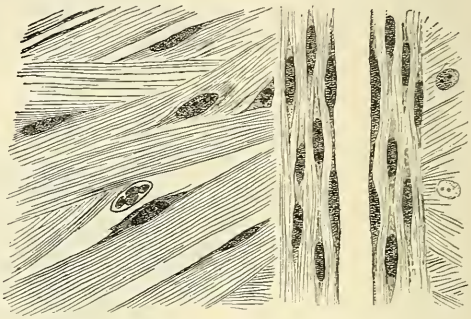


Fig. 66.—Cicatricial tissue; × 670 (Fowler).

the scar becomes firm, white, and puckered. *Cicatrization* consists in the conversion of immature connective tissue into mature fibrous tissue and in the contraction of the new fibrous tissue. Cicatrization is hurried in a healthy granulating wound by the application of an 8 per cent. ointment of scarlet red. It is kept in place only twenty-four hours. To keep it longer will irritate the wound edges. If infection is severe, destruction will exceed repair and healing will not occur. In such a case there is coagulation necrosis of granulation tissue, and the wound becomes covered with tissue remains (aplastic lymph). If granulations rise above the cutaneous level, healing will not take place, because the epithelium cannot then grow over the raw surface. A wound in this condition is said to possess *exuberant granulations*, or *proud flesh*. In some cases the granulations are *pale* from insufficient blood-supply, and in others *edematous* from venous congestion. Contraction of the fibrous tissue may be insufficient because there is adhesion to deep unyielding fascia or to periosteum. Excessive contraction is frequent after burns and often produces terrible deformity. The scars or cicatrices of burns contain much elastic tissue derived from cell protoplasm. Infected wounds and ulcers heal by second intention.

**Healing By Third Intention.**—This consists in the union of two granulating surfaces, the granulations of one side fusing with the granulations of the other side. It is seen in the union of collapsed abscess-walls. The surgeon occasionally seeks to obtain union of a wound several days old by third intention by approximating two granulating surfaces. If the surfaces are aseptic, he will often succeed. The procedure of approximation is known as *secondary suturing*. It is not unusual to pack a wound with iodoform gauze to control oozing. When this is done it is customary to pass the sutures, but not to tie them. After a few days the gauze is removed and the sutures are

tied. This plan renders healing much more rapid than would be possible by the process of healing by second intention.

**Cicatrices, or Scars.**—The newly formed connective tissue which constitutes a scar will be present in large amount if more granulations were formed than were really necessary for repair or if a considerable defect was repaired.

A recent scar contains fibrous tissue, many fibroblasts, and numerous blood-vessels, but no nerves, lymphatics, or elastic fibers. The skin above recent scars is usually red because of the numerous vessels beneath it and the layer of epidermis is well developed. In old scars fibroblasts have disappeared and fibrous tissue really constitutes the cicatrix. Some blood-vessels disappear and the diameters of those remaining are much reduced. These vascular changes result from contraction of the cicatrix. Delicate elastic fibers appear in old scars. They appear at the end of the second month in wounds healed by first intention, at the end of the third or fourth month in wounds healed by second intention, and they take origin directly from cell protoplasm and not from fibrous tissue (Minervini, in "Virchow's Archiv," vol. clxxv, No. 2). No genuine lymphatics exist in old scars, but occasionally nerve filaments are present. Some dermal papillæ are found after a time, but skin glands, skin muscle, and hair-follicles remain absent.

An old scar is smooth, whiter than the surrounding skin, somewhat creased or wrinkled, and deficient in tactile sense. The scar of a healed tuberculous ulcer is irregular, livid, and often actually corrugated. The scar of a healed syphilitic ulcer is at first coppery red and then glistening white and depressed. The scar of an old ulcer of the leg and of the skin about it is often darkened by pigmentation.

A cicatrix may be discolored by retained foreign bodies, for instance, grains of gunpowder.

During scar formation shreds of epidermis may be displaced and included in granulation tissue. Subsequently they are included in fibrous tissue, and may then give rise to transplantation (*implantation*) dermoids or to epithelial tumors (see page 379). A scar may be deformed, for instance, may be greatly depressed and adherent to underlying bone, and in certain situations such a scar will fix the jaws or any other joint. The *vicious cicatrix* is a great excess of scar tissue and results from delayed healing by second intention. Such cicatrices are particularly common after burns and tuberculous ulcerations. In some cases the scar is irregular and lumpy, in other cases it is thickened at certain parts and discolored and resembles keloid.

A cicatrix may block a natural orifice, as the mouth or nostril; may produce great deformity, for instance, the head may be drawn upon the chest or shoulder by a contracting scar in the neck, fingers may be grown together after a burn, or a hideous depression may exist on the forehead after an injury, or the face may be fearfully contorted by contracting cicatrices. A scar may produce great disability by blocking the jaws, obstructing the rectum or urethra, or fixing a joint or certain muscles of an extremity.

Most scars are insensitive, some are hypersensitive. The hypersensitive scars are usually thin and pale. The itching, burning, or tingling appreciated in a sensitive scar are located, as a rule, at the junction of sound skin and newly formed epidermis. Sometimes acute neuralgic pain exists in and about a scar due to pressure upon nerve filaments.

A scar may inflame or ulcerate, warts may spring from its cutaneous surface, keloid may arise from the fibrous tissue, carcinoma may come from the epithelial elements (Marjolin's ulcer), sarcoma from the connective-tissue elements.

**Healing of Subcutaneous Wounds.**—Blood fills the tissue gap and the blood clots. Plasma exudes and corpuscles migrate into the clot and the

tissue about it. The clot is eaten up by the leukocytes. The connective-tissue cells and the endothelial cells of the adjacent tissue proliferate and form fibroblasts, and fibroblasts multiply and replace the clot. The area of fibroblasts is vascularized by the formation of new capillaries, fibrous tissue forms and strongly contracts.

**Healing of Wounds In the Non-vascular Tissues.**—After a trivial injury of the cornea a few leukocytes gather from the lymph-spaces and a few of the fixed cells proliferate. When the cornea is more severely wounded, an increased flow of lymph occurs. The nerves are irritated, vessels adjacent to the cornea distend, and many leukocytes invade the lymph-spaces. The corneal corpuscles multiply and alter in shape. The product of the process may be transparent if fibrin is absorbed and leukocytes pass away, because proliferating corneal corpuscles form transparent tissue. The surface epithelium is replaced by proliferation of the deep layer of corneal epithelium. If the wound has penetrated the posterior portion of the cornea, it becomes filled by proliferating epithelium from the membrane of Descemet. In a severe injury of the cornea endothelial cells and corneal corpuscles proliferate, vessels grow in from the corneal margins toward the seat of inflammation, fibrous tissue forms, and permanent opacity results.

Repair in cartilage, when it occurs at all, is very slow and is accomplished in the same way as repair in the cornea. Any severe injury is repaired by white fibrous tissue, furnished by the cells of the perichondrium, and the scar is permanent.

**Cell-division.**—The multiplication of connective-tissue cells in repair may be by direct, but is usually by indirect, cell-division. *Direct cell-division* consists in division of the nucleus followed by division of the entire cell.

*Indirect cell-division*, or *karyokinesis*, takes place after remarkable changes in the nucleus. The membrane of the nucleus disappears; the nuclear network becomes first close and then more open; and the cell becomes round, if not so before. The network of the nucleus, now consisting of one long fiber, takes the shape of a rosette; next it takes a star form—the aster stage; two V's next form—the equatorial stage; an equatorial line appears and widens, and each one of the V's retreats toward a pole. Thus two new nuclei are formed, each polar V passing in inverse order through the previous changes of shape, and the protoplasm of the original cell collecting about each nucleus (Fig. 67).

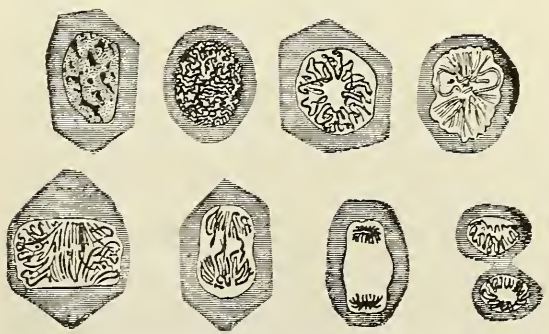


Fig. 67.—Forms assumed by a nucleus dividing (Green, from Flemming).

**Repair of Nerves.**—A nerve-fiber consists of a core known as the axis-cylinder, which is the essential element in function. About the axis-cylinder is an almost liquïd material, known as the medullary sheath or white substance of Schwann, or myelin. The myelin is surrounded by a firm sheath known as the neurilemma (sheath of Schwann, primitive sheath, neurilemma). On the inner surface of the sheath of Schwann, or between it and the white substance of Schwann, are nuclei which are supposed by some to be peripheral nerve-cells (neuroblasts). The neurilemma is absent in the brain and cord. The continuity of the white substance of Schwann is interrupted at frequent

intervals, and these breaks in the myelin are called nodes of Ranvier. Numbers of fibers of the kind just described, bound in bundles by connective tissue and surrounded by a fibrous sheath, constitute a nerve. It is known that a nerve may regenerate and completely regain function after division; that regeneration is strongly favored by suturing the ends together; and that if the ends of a divided nerve are more than 1 inch apart, regeneration will rarely take place unless they are sutured together. The method by which regeneration is affected has been much disputed and is still involved in uncertainty. If a nerve is divided, the peripheral segment at once loses its function and then undergoes degeneration (Wallerian degeneration). The degeneration begins within twenty-four to forty-eight hours and affects the entire peripheral segment. The axis-cylinder perishes, the myelin runs into globules and is absorbed, leaving an almost empty sheath; the nuclei of the inner surface of the neurilemma proliferate for a time, but cease to do so before the myelin is completely absorbed. The sheath shrinks and looks empty, but here and there are collected masses of proliferated nuclei and protoplasm. Degeneration takes place in days, but regeneration requires months. Regeneration takes place by the multiplication of pre-existing nerve-fibers, and not by the transformation of connective tissue into nerve structure. The ends of a divided nerve, it is true, become united by connective tissue formed by the proliferation of fibroblasts, but this connective tissue is only a bridge to carry nerve elements across the gap between the proximal and peripheral segments. The common view is that regeneration takes place as follows: The new axis-cylinder of the peripheral segment is a prolongation of the old axis-cylinder of the proximal segment, projected in the following manner: A fiber, which is at first devoid of myelin, is prolonged from a proximal axis-cylinder; it divides into many cylinders, which pierce the granulation tissue between the separated ends and enter into the empty sheaths of Schwann of the distal segment or insinuate themselves between these sheaths (Ranvier, Réclus, Senn). The above is the view entertained by those who teach that the new axis-cylinders come entirely and only from the prolongation of old axis-cylinders of the proximal segment, that the distal segment is passive in the process until "neurotised" (Vanlair), and that regeneration is impossible in the distal segment unless it is in approximation with the proximal segment or within easy reach of the prolongations of the axis-cylinders from above. Another view is that the axis-cylinders, myelin, and neurilemma are formed from cells which exist in the distal segment, and that juvenile axis-cylinders and medullary sheaths are formed in the peripheral portion and then effect a junction with like structures of the central segment. The last-mentioned view is advocated by Mayer and Eichhorst, Tizzoni, Cattani, and others, and Ballance and Stewart have published a most valuable monograph advocating it ("The Healing of Nerves"). The nuclei proliferate and form a mass of protoplasm within the old sheath, which protoplasm subsequently joins the proximal segment. Such a protoplasmic fiber has "conduction and irritability" (Raymond's "Human Physiology"), but there is as yet neither myelin nor axis-cylinder. "The fiber is responsive to mechanical stimuli, but not to induction shocks, which latter property returns only after the axis-cylinder is developed. The medullary substance later appears and forms a tube; and still later the axis-cylinder is formed, having its origin in the central end of the nerve" (Ibid.). The views of Ballance and Stewart may be set forth as follows: When a nerve-trunk is divided, the peripheral segment degenerates whether it has been sutured to the proximal segment or not, and the portion of the proximal segment near the wound also degenerates. The injury produces at once an effusion of blood, migration of leukocytes takes place into and about the wound at the proximal segment, but leukocytic invasion of the entire distal segment is noted. After three days

connective-tissue cells begin to replace the leukocytes, and after two weeks the excess of leukocytes is no longer observed, proliferated connective-tissue cells having taken their place (Ballance and Stewart, "Healing of Nerves," page 94). The proximal segment in the neighborhood of the wound and the entire distal segment are invaded by proliferating connective-tissue cells. The connective-tissue cells completely absorb the fatty myelin and axis-cylinders. The cells of the neurilemma actively multiply, and connective-tissue cells lying among chains of neurilemma cells become spindle-shaped and "the degenerated nerve-trunk therefore becomes hard, fibrous, and cirrhosed" (Ibid.).

In the proximal end of a divided nerve an "*end-bulb*" is formed. This was long supposed to be due to the prolongation of nerve-fibers from the central fibers and a turning backward because they could not cross the gap. As a matter of fact, the ends of the divided fibers curl up; on and in this scaffold-like arrangement new fibers are placed, they having been produced by the neurilemma cells which have taken on "neuroblastic function" (Ballance and Stewart). When a nerve has been sutured, the earliest signs of regeneration "occur at the end of three weeks" (Ibid.). Short lengths of new fibers are laid down within old neurilemma sheaths. The new axis-cylinder "is seen to consist in the deposition along one side of a spindle-shaped neurilemma cell, of a thin thread which grows in length until it projects beyond the limits of the parent cell and stretches on toward its next neighbor in the same longitudinal row" (Ibid.). The new medullary sheath is "laid down by a process of secretion" (Ibid.) along the sides of the neurilemma cells.

Ballance and Stewart go on to point out that if the central theory of regeneration is true, not a trace of regeneration could occur in the distal segment when the two segments have not been united by sutures, and yet such regeneration *does* occur, although slowly, the new axis-cylinders and medullary sheaths not attaining full size. "Evidently some stimulus afforded by the conduction of impulses is necessary in order to permit of their full development" (Ibid.). In the notable study quoted at such length are some experiments on the "conduct and fate of transplanted nerve." When the gap is wide between the two ends, a portion of fresh nerve-trunk may be inserted to bridge it. The transplanted piece degenerates; it is invaded by leukocytes, and proliferating connective-tissue cells, medullary sheaths, and axis-cylinders are destroyed, but regeneration may subsequently occur; "but when it does occur, it is not from the activity of the cells of the graft itself." Blood-vessels enter the degenerated graft at each end and they are accompanied by chains of neurilemma cells, which form axis-cylinders and medullary sheaths. The graft is merely a scaffold. Verga (quoted "Journal de Chirurgie," April, 1910) opposes the notion that the peripheral end plays any part in regeneration and advocates the view that all regeneration comes from the central end.

The studies of Ballance and Stewart persuade us that regeneration does occur in the distal part independently of the proximal part, although full development does not take place unless there is a junction with the central part. As to the exact method of regeneration we still feel somewhat uncertain. When we remember that the nerve-fibers of the spinal cord are devoid of neurilemma and that the cord can, to some extent at least, regenerate, we must conclude that regeneration can take place in the cord without the aid of neurilemma cells, and must infer that the same may be true in a nerve.

**Repair of the Spinal Cord and Brain.**—Can the spinal cord regenerate? Many observers have doubted it. But there is no doubt of the fact that sometimes, after the subsidence of an acute myelitis or after the relief of a pressure

which produced complete and prolonged paralysis, there is a return of functional power. It is usually assumed that restoration is possible in fibers which have not been hopelessly damaged, but is not possible in those which have been destroyed; but, as Gowers says, there are cases in which "we can scarcely believe that the axis-cylinders retain their continuity, although conducting capacity is ultimately restored." Clinical evidence indicates strongly that the pyramidal fibers may regenerate. Mills ("The Nervous System and Its Diseases") says: "Nerve-tracts in the spinal cord and brain have power to regenerate, but this is not so great as in the peripheral nerves, and yet even old cases of compression of the spinal cord may make great improvement after a long time, largely through the regeneration of the columns of the cord." Mills affirms that although nerve-cells sometimes appear to regenerate, the destruction in these cases was not complete. Recently I showed in my clinic a man who had had complete paraplegia with paralysis of the bladder and rectum for nineteen years. The condition was due to a bullet lodged within the spinal canal. Removal of the bullet was followed in a few weeks by restoration of control over the bladder and rectum. In a few months spastic paraplegia was substituted for flaccid paralysis.

When axis-cylinders have been destroyed in the cord and yet some power returns, we ask ourselves: Does this occur because new fibers have grown down from above? Gowers says that such a growth has been proved to occur in the lower animals, but has not as yet been demonstrated in man; although specimens have been described which strongly suggest such an occurrence in the human subject. That the cord can regenerate to some extent seems highly probable from the report of a case operated upon by my colleague, Professor Francis T. Stewart, of Philadelphia. He sutured a completely divided spinal cord and extraordinary restoration of function took place (Francis T. Stewart and Richard H. Harte, in "Phila. Med. Journal," June 7, 1902). This case is commented on at some length in the section of Injuries on the Spinal Cord. Another somewhat similar case was reported by George Ryerson Fowler in the "Annals of Surgery," Oct., 1905.

Many claim that a brain injury cannot be followed by repair with restoration of function; some think that complete regeneration can take place; others, that partial regeneration may occur. Vitzon and Tedeschi even believe that nerve-cells in the brain can regenerate. It seems probable that extensive injuries are not repaired, but slighter ones may be, new ganglion-cells and neuroglia being formed. Tedeschi describes the process of repair after a wound of the brain as follows: Degeneration occurs and a limited focus of necrosis forms and then the adjacent tissue shows evidences of repair. Capillaries form from the endothelial cells, glia tissue from the neuroglia, ganglion-cells present karyokinetic changes, and some nerve-fibers appear in the scar (Senn's "Principles of Surgery").

**Repair of Muscles.**—It has long been taught that the repair of muscle by muscle is impossible, and, as a matter of fact, it does not take place if the ends of a divided muscle are separated to the extent of an inch or more. When a muscle is divided transversely by a cut of considerable extent, the ends of the divided fibers retract and a wide space is left between them. Blood flows into the space between the ends, and also between individual fibers of the injured muscle and the blood-clots. Exudation of plasma occurs and migration of corpuscles takes place. Fibroblasts are produced by proliferation of connective-tissue cells and a mass of fibroblasts soon replaces the blood-clot. Granulation tissue is formed by vascularization of the mass of fibroblasts, and granulation tissue is converted into scar tissue, but not at all into muscle. After slight injuries a trivial amount of muscular regeneration does occur by the multiplication of living muscle-cells, but not by metamorphosis of fibroblasts.

Fibroblasts are incapable of transformation into muscular tissue. When the ends of a divided muscle are separated only to a very slight degree or when they have been brought together and sutured, some muscular regeneration occurs. After an injury a number of the muscular fibers always wither, perish, and are absorbed. The process of regeneration arises from the remaining fibers. The nuclei of the muscle-fiber proliferate and so do the nuclei of the perimysium. The muscle-cells are called myoblasts, and the nuclei of the perimysium are called sarcoblasts. About the juvenile muscle-cells a deposit of protoplasm takes place (Weber). The embryonal cells gradually become spindle-shaped, and muscular fiber is formed by cellular fusion or by elongation of individual cells.

The above remarks refer to striated muscle. Unstriated muscle-fibers are repaired solely by "indirect multiplication of their nuclei" (Senn).

If a muscle has been divided, it should be sutured. This process insures more rapid repair and secures a better functional result, and is followed by a much greater amount of muscular regeneration.

**Repair of Tendon.**—When a tendon is divided, the ends retract, and the sheath, as a rule, becomes filled with blood-clot. The blood-clot is rapidly removed, fibroblasts replacing it. This new tissue arises from the sheath, the cut ends of the tendon not participating in its formation. Granulation tissue is formed; this is converted into fibrous tissue, and after a time the fibrous tissue becomes true tendon. If no blood-clot forms in the sheath, the walls of this structure collapse and adhere, and the separated tendon-ends are held together by a flat fibrous band formed from the collapsed sheath (Warren's "Surgical Pathology").

**Repair of Bone.**—When a bone is broken a blood-clot quickly forms in the medullary cavity, between the broken ends and under and outside the periosteum. Leukocytes invade and destroy the clot. The cells outside the periosteum, the cells of the periosteum and of the medullary tissue, particularly the endothelial cells, proliferate and produce cells which are practically fibroblasts. The osteoblasts in the medullary tissue, and perhaps in the deeper layers of the periosteum, multiply and are distributed through the mass of fibroblasts. The osteoblasts may form bone directly or may form cartilage first. Some teach that fibroblasts can be converted into bone; others positively deny such a conversion. The point is not settled, but it is well to remember that in myositis ossificans a muscle is converted into bone, and hence that it is probable that fibroblasts, which are formed from periosteum and medullary tissue, are more prone to undergo such a development. During regeneration the bone ends soften and are partially absorbed by osteoclasts. Osteoclasts are large osteoblasts which have lost the power of bone production and furnish a secretion which dissolves osseous matter. The excess of callus is finally absorbed by osteoclasts. (For a more extended description see Repair of Fractures.) Sir William Macewan has recently denied that the periosteum plays a leading rôle in bone production ("Brit. Med. Jour.," June 22, 1907). He believes that the periosteum is a membrane to limit and control the osteoblasts and that new bone is formed purely from bone cells. There is much experimental evidence to confirm Sir William's assertion. If he is correct, a considerable osseous defect could not be filled up by new bone even if the periosteum were intact. As a clinical fact, we see this very thing occur. In a more recent article ("Lancet," August 3, 1912) Sir William claims that osteoblasts are the essential elements in repair, that they come from cartilage cells, that when growing bone is stripped of periosteum growth continues, that when bone is removed and periosteum is left no growth occurs, and that transplantation of spicules offers the best chance of repair because each fragment furnishes peripheral growth. During the last few years many surgeons have utilized bone

transplantation, for instance, transplanting a portion of a rib or the crest of the tibia of the same individual to fix an ununited fracture, to fill a bone gap the result of osteomyelitis, or to anchor vertebræ as in Albee's operation for vertebral caries. John B. Murphy in many respects disagrees with Macewan. (See "Practical Med. Series," vol. ii, 1912.) He believes that a bit of periosteum completely detached from the bone of a young individual and placed in the fat or muscle of the same individual may make new permanent bone. If one end of the strip is allowed to retain attachment to the bone, the transplanted end practically always makes new bone. Bone with or without periosteum transplanted in the same individual and placed in contact with growing bone unites to the living bone, acts as a scaffold for blood-vessels and osteogenetic cells, and is eventually absorbed.

Fig. 68.—Fracture one week: blood-clot containing fragment of bone (Warren).

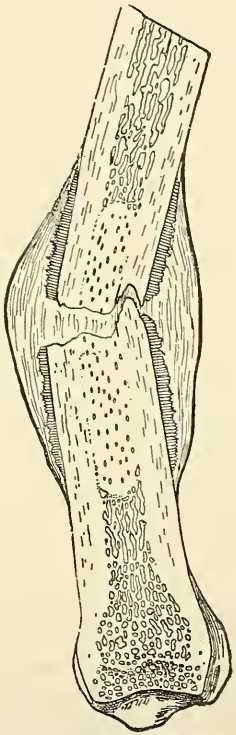


Fig. 69.—Callus of fracture (dog), four weeks: commencing ossification of external callus (Warren).

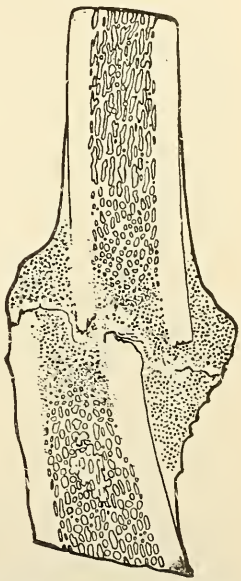


Fig. 70.—Femur of a child fifth week after fracture (Warren).

**Repair of Blood-vessels.**—If an artery is cut across and ligated, a clot forms within its lumen and about its divided end, and the circulation in the vessel at this point is permanently arrested. The proximal clot, it used to be thought, always reaches the first collateral branch. This statement was true before the days of asepsis; it is not always true now. Often a clot stops far short of the branch above. Exudation of plasma and migration of corpuscles take place from the vasa vasorum. The clot becomes filled with leukocytes, which gradually destroy it, and it plays no active part in repair. Fibroblasts form by the multiplication of the cells of the vessel wall and the clot is soon replaced by fibroblasts. The fibroblasts are converted into granulation tissue, granulation tissue becomes fibrous tissue, the fibrous tissue contracts, and the

artery is transformed into a fibrous cord. Warren insists that the muscle-cells of the middle coat play an active part in repair. Usually, when a ligature is applied to an artery in continuity, a deliberate attempt is made to rupture the internal and middle coats, in order to permit of contraction and retraction above and below the seat of ligature, and a turning inward of the inner coat. Such a sequence of events happens when an artery is completely divided across and not tied, and favors the rapid formation of a clot.

Ballance and Edmunds ("Ligation in Continuity") maintain that repair is obtained most rapidly when the artery is tied with two ligatures, the vessel at this point being deprived of blood, but the internal and middle coats being kept intact. Cell-proliferation forms a spindle-shaped mass of new cells and the lumen is obliterated at the seat of ligation by fibroblasts obtained from the fixed cells of the wall of the artery. Senn advocates the employment of two ligatures, not placed side by side as in the method of Ballance and Edmunds, but so applied as to include "a bloodless space about $\frac{1}{2}$ inch in length" ("Principles of Surgery").

When a lateral ligature is applied to a vein or when a small wound in a vein or artery is sutured, the circulation in the vessel is not completely cut off, a thrombus of small size is formed on the vessel walls, the fixed cells of the vessel wall proliferate, and a scar of fibrous tissue effects repair. A completely divided vein heals as does a completely divided artery. The clot after the aseptic application of a ligature to a vein may be of slight extent, but in some cases the proximal clot reaches the first collateral branch and in others goes far above it.

**Repair of Skin.**—The fibrous structure is repaired by fibrous tissue. Hair-follicles, sweat-glands, and sebaceous glands are not re-formed. The epithelial layer is regenerated by the proliferation of adjacent epithelial cells.

*Repair of Lymphatic Tissue.*—Lymphatic tissue can regenerate either from the fatty tissue, the divided ends of the lymph-ducts, or both structures.

*Repair of the Kidney and Testicle.*—These organs when damaged can undergo some regeneration.

*Repair of the Liver and Spleen.*—Each of these organs, after injury, is capable of considerable regeneration.

## V. SURGICAL FEVERS

The surgeon encounters fever as a result of an inflammation or an aseptic wound, in consequence of infection, as a result of poisoning by certain drugs, and in several maladies of the nervous system. It is important to remember that, while elevated temperature is generally taken as a gauge of the intensity of fever, it is not a certain index. There may be fever with subnormal temperature (as in the collapse of typhoid or pneumonia), and there may be elevated temperature without true fever (as in certain diseases of the nervous system). It is true, however, that elevation of temperature is almost always noted, and is usually accepted as the measure of the severity of the fever.

Elevated temperature is only a symptom. The elevated temperature of an infection may be regarded as evidence that the body is fighting the infection. An acute infection with a low or subnormal temperature is a far graver condition than an acute infection with a high temperature. The low temperature shows that the body is abandoning the contest; the subnormal temperature shows that it has abandoned it. Exham ("Brit. Med. Jour.," January 27, 1912) points out that the worst cases of pneumonia, peritonitis, and diphtheria are those with subnormal temperature, and that in some of the most malignant cases of scarlet fever the temperature does not rise much above

100° F. As Exham says: elevated temperature is a defence and subnormal temperature means that body resistance is at an end (see page 43). If in doubt as to the cause of fever, count the leukocytes; make a blood-culture, a Widal test, and a Wassermann test.

The *essential phenomena of fever*, according to Maclagan, are—(1) wasting of nitrogenous tissue; (2) increased consumption of water; (3) increased elimination of urea; (4) increased rapidity of circulation; (5) preternatural heat.

**Traumatic fevers** follow a traumatism and attend the healing or infection of a wound. The forms are—(1) benign traumatic fever; (2) malignant traumatic fever.

**Benign traumatic fever** is divided into two forms—the *aseptic* and the *septic*. There is but one form of aseptic fever, the postoperation rise. The septic benign fevers are surgical fever and suppurative fever. The malignant traumatic fevers are sapremia, septic infection, and pyemia. In this section we discuss only the benign fevers.

**Aseptic traumatic fever,** or the *postoperation rise*, often, but not always, appears after a thoroughly aseptic operation and after a simple fracture or a contusion. It is not preceded by a chill, by chilliness, or by a feeling of illness. It may appear during the evening of the day of operation or not until the next day, and reaches its highest point by the evening of the second day (100°–103° F.). This elevation is spoken of as the "postoperation rise" because it is usually encountered after an operation. Besides the elevated temperature there are no obvious symptoms; the patient feels well, sleeps well, and often wants to sit up; there are no rigors and there is no delirium. The wound is free from pain and appears entirely normal. Blood examination may show moderate leukocytosis. This fever is due to absorption of pyrogenous material from the wound area, the material being obtained from clot or inflammatory exudate, or from both. Many observers believe that the pyrogenous element is fibrin ferment, which is absorbed from disintegrating blood-clot and coagulating exudate. Warren thinks the fever is due to fibrin ferment, and "also to other substances slightly altered from their original composition during life." Some have asserted that the fever is due to nervous shock.

Schnitzler and Ewald have studied aseptic fever.[1] These observers maintain that aseptic fever can exist when no fibrin ferment is free in the blood, that fibrin ferment can be free in the blood when there is no fever, and, in consequence, that fibrin ferment is not the cause of the elevation of temperature. They rule out of consideration nervous shock as a cause, and assert that a combination of several factors is responsible, nucleins and albumoses which are set free by traumatism being looked upon as the most active causative agents. The presence of nuclein in the blood in aseptic fever is indicated by leukocytosis and by the increase of the alloxur bodies (including uric acid) in the urine. The capacity of nucleins and albumoses to cause elevated temperature is greater in the tuberculous than in the non-tuberculous, and we know clinically that a tuberculous patient is apt to exhibit a more violent postoperation rise than is a non-tuberculous subject. The diagnosis of aseptic traumatic fever is only to be made after a careful examination has assured the surgeon that there is no obscure or hidden area of infection.

In some cases aseptic fever may appear after an operation, and later be replaced by a septic fever. If the temperature remains elevated more than a day or so, if other symptoms appear, or if after the temperature has become normal it again rises, the wound should be examined at once, as trouble almost certainly exists.

[1] See "Archiv für klinische Medicin," Bd. liii, H. 3, 1896; also statement of their views in "Medical Record," Dec. 19, 1896.

**True traumatic, or genuine surgical fever,** is seen as a result of infected wounds in which there is decided inflammation, but no pus. The real cause is the presence of fermentative bacteria in the wound and the absorption of a moderate amount of their toxic products. The most active and commonly present organisms are those of putrefaction. Surgical fever ceases as soon as free discharge occurs, and the appearance of such a fever is an indication for instant drainage. The condition is ushered in two or three days after the operation by chilly sensations and general discomfort. The temperature rises pretty sharply, ascends with evening exacerbations and morning remissions, and reaches its height about the third or fourth day, when suppuration sets in; the temperature begins to drop when pus forms, if the pus has free exit, and reaches normal at the end of a week. (See Suppurative Fever.) The temperature may reach 104° F. or more, but rarely rises above 103° F. The patient has the general phenomena of fever, that is to say, thirst, anorexia, nausea, dry and coated tongue, constipation, pain in the back and legs, and headache. The urine is scanty and high colored. Blood examination usually shows decided leukocytosis. The wound is painful, tender, swollen, discolored, and often foul, and stitch-abscesses may form. Some or all of the stitches must be cut, the area should be asepticized, the wound edges separated by iodoform gauze, or the wound drained by a tube. The fact that this fever is apt to cease when discharge of pus begins led the older surgeons to hope for pus and to endeavor to cause it to form. A severe grade of surgical fever, such as arises when there is putrefaction in a large and ill-drained wound, is due to the absorption of a large quantity of the toxic products of putrefactive bacteria, and is known as sapremia (see p. 194).

**Suppurative Fever.**—This fever, which is due to the absorption of the toxins of pyogenic organisms, occurs after suppuration has begun, is found when the pus has not free exit, and is an intoxication rather than an infection, that is to say, toxins enter the blood, but no bacteria. It can follow or be associated with surgical fever, or may arise in cases in which surgical fever has not existed. Suppuration in a wound is indicated by a rapid rise of temperature—possibly by a chill. The temperature rises to a considerable height, shows morning remissions and evening exacerbations, and as it begins to fall toward morning sweating occurs. The patient is much exhausted and presents the phenomena of fever previously described. The skin about the wound becomes swollen, dusky in color, and edematous, pain becomes pulsatile, and much tenderness develops. Blood examination shows very marked leukocytosis. The wound must at once be drained and asepticized. In a chronic suppuration, such as occurs when there is pyogenic infection of a tuberculous area, there exists a fever with marked morning remissions and vesperal exacerbations, attended with drenching night-sweats, emaciation, diarrhea, and exhaustion. This is known as *hectic fever;* it is really a chronic suppurative fever. The treatment of hectic fever consists in the drainage and disinfection, if possible, the excision of the infected area, the employment of a nutritious diet, stimulants, tonics, remedies for the exhausting sweats, and free access of fresh air.

**Some Other Forms of Fever Seen by the Surgeon.—Fever of Tension.**—When there is great tension upon the stitches, the spots where the stitches perforate ulcerate and some fever arises. To relieve the fever of tension cut one or several stitches. This fever is in some cases surgical, and in some suppurative, according as to whether the infective organisms cause fermentation or suppuration.

**Fever of Iodoform Absorption.**—(See page 31.)

**Fever of Ptyalism, or Mercurial Fever.**—(See page 338.)

**Fever Due to Awakening of An Area of Pulmonary Tuberculosis.**—A quiet, non-progressive area of pulmonary tuberculosis may burst into activity

after an operation, and is particularly apt to if ether was administered. The surgeon must be watchful of this condition. Several times I have seen a person with signs suggesting bronchitis at the base of one lung develop a moderate and prolonged fever. Such a condition is not bronchitis because it is unilateral. The sputum shows pus cocci, but no tubercle bacilli. I formerly regarded it as tuberculosis, but it is always recovered from and is probably bronchopneumonia of one lobe of one lung.

**Fever of Morphinism.**—Sometimes a morphin habitué suffers from severe chills and intermittent fever of the quotidian or tertian type. The condition is usually thought to be malarial, a view which is strengthened by the common association with neuralgia; but quinin proves futile as a remedy and blood examination gives a negative result. If we have reason to suspect that the patient is using morphin, examine the urine for the drug and wash out the stomach and examine the washing. The latter test is of value even when morphin is used hypodermatically, because some of the drug is excreted into the stomach.

**Fever of Cocain-poisoning.**—(See Local Anesthesia.)

**Hepatic Fever.**—(See section on Liver and Gall-bladder.)

**Neurotic or Hysterical Fever.**—This remarkable condition is occasionally, though seldom, encountered. It is unusual for the temperature to rise above 101° F. Most of the reported cases of great hyperpyrexia are instances of simulation and fraud. That very great elevation can occur is shown by the case seen by Mr. J. W. Teale, which case was also observed by Sir Clifford Allbutt. The temperature rose again and again to 118° F. In such cases the temperature rises very rapidly, remains at its height but a short time, and then falls as rapidly as it arose. It may happen that elevated temperature is the sole evidence of illness, there being no wasting, thirst, or other febrile symptoms. Cold sponging rapidly lowers the temperature. Such elevated temperature may occur irregularly or be attained daily for months. As a rule, hysterical stigmata can be detected. Osler points out that cases of hysterical fever "with spurious local manifestations" are very deceptive. The case may resemble meningitis, peritonitis, or some other acute inflammatory condition; but the course of the supposed malady is found to be atypical and the symptoms are observed to be variable and often anomalous. There is no leukocytosis; frequently there is an apparent increase in red cells, because of vasomotor disturbance, a fall in hemoglobin, and an increased proportion of lymphocytes and eosinophiles ("Clinical Hematology," by J. C. DaCosta, Jr.). It is dangerous to make a diagnosis of neurotic fever; it must not be done unless all other possible causes have been excluded. Some supposed cases depend upon unrecognized tuberculosis, some on visceral syphilis, some on undiscovered malignant disease, some on toxin absorption from the alimentary canal.

An **emotional fever** sometimes occurs after accidents or operations. The patient may have a chill, and then develop violent headache, photophobia, and hysterical excitement, with elevated temperature. Inexplicable elevations of temperature may occur in neurasthenia.

**Malaria.**—It is wise to examine the blood in supposed septic fevers, for only by this means can malaria be excluded. It is more common to mistake sepsis for malaria than malaria for sepsis. In malaria the spleen is enlarged, the febrile attacks exhibit periodicity, neuralgias are common associates, and quinin cures the condition.

**Surgical Scarlet Fever.**—It is maintained by some writers (notably Sir Victor Horsley and Sir James Paget) that a child is rendered especially susceptible to scarlet fever by the shock of a surgical operation. Scarlet fever which develops after a wound, a burn, or an operation is spoken of as surgical scarlet fever. Warren quotes Thomas Smith as having had 10 cases of scarlet

fever in 43 operations of lithotomy in children. The puerperal state is supposed also to predispose to scarlet fever. It is not certain whether the poison enters by the wound, or whether shock and exhaustion predispose to ordinary scarlatina, or whether ordinary scarlatina was incubating before the accident or operation. Some surgeons hold that an attack of scarlet fever after an operation is a mere coincidence. Others maintain, and with great show of reason, that a red scarlatiniform eruption appearing after an operation rarely indicates genuine scarlet fever, but usually points to infection, as such eruptions are known occasionally to arise in septicemia. It *rarely* indicates scarlet fever, and yet it sometimes does. There is such a condition as surgical scarlet fever, as is proved by the facts that victims of the disease have been known to communicate it, and that it is often followed by "nephritis and usually by desquamation" (Holt's "Diseases of Infancy and Childhood").

Hoffa has discussed this subject elaborately. He concludes that four types of eruption can follow operation: (1) a vasomotor disturbance due to irritation of sensory nerves, and manifested by a transient urticaria or erythema; (2) a toxic erythema due to absorption of aseptic pyrogenous material from the injured area—the absorption of carbolic acid, iodoform, of corrosive sublimate, or the effect of ether; (3) an infectious rash, which is sometimes found in septicemia or pyemia, and is due to minute emboli composed of bacteria, which emboli lodge in the capillaries; (4) true scarlet fever, with the usual symptoms and complications, the micro-organisms having entered by way of the wound and the eruption often beginning at the wound edges (quoted in Warren's "Surgical Pathology"). Surgical scarlatina is aberrant. It develops rapidly, the period of incubation is extremely brief, and the throat may or may not be involved. Holt tells us that the rash is usually atypical and that "the general symptoms, particularly those relating to the nervous system," are "especially severe" ("Diseases of Infancy and Childhood"). The infection is believed to be due to a specific germ, but it has not been certainly identified. Streptococci have been found in the throat, skin, and the pus from secondary otitis media.

If surgical scarlet fever develops the wound should be drained and aseptic-ized, and, if the situation admits of it, dressed with hot antiseptic fomentations. The general treatment is the same as for ordinary scarlatina.

**Fever of Malignant Disease.**—Elevation of temperature may occur during the course of sarcoma or carcinoma. It is particularly apt to if growth is very rapid or if ulceration exists. Malignant growths of the liver are especially apt to cause elevation of temperature and leukocytosis.

Fever of malignant disease usually appears as irregular elevations of temperature of short duration. In some cases there is a continuous or remittent fever; in some an intermittent fever. Even the hectic type is met with.

**Urinary Fever and Urethral Fever.**—(See section on Disease of Genito-urinary Organs.)

**Syphilitic Fever.**—(See page 322.)

**Thyroid Fever.**—(See section on Thyroid Gland.)

## VI. SUPPURATION AND ABSCESS

Suppuration is a process in which damaged living tissues and inflammatory exudates are liquefied by the action of pyogenic organisms, and it is a common result of microbic inflammation. The organisms which are responsible are referred to on page 48. Staphylococci tend to produce local suppuration; streptococci tend to cause spreading suppuration. It is generally taught that pyogenic bacteria liquefy damaged tissues and exudates by peptonizing them,

the active agent in effecting the chemical change being poison furnished by the bacteria. There is some evidence that white corpuscles by disintegration set free enzymes, which dissolve or aid in dissolving albumin. Streptococci and staphylococci vary greatly in virulence, and the intensity and diffusion of a pyogenic infection depends upon the virulence and number of the bacteria and the level of vital resistance. Streptococci and staphylococci may both be present in one focus, and there may be secondary infection with bacteria of putrefaction or other bacteria. The pyogenic infection may be primary or it may be secondarily implanted in a disease area containing other micro-organisms. The pyogenic organisms are very irritant, and when deposited cause inflammation; inflammation leads to exudation, but the exudate cannot coagulate or coagulates but imperfectly, because it is peptonized by the ferment of the micro-organisms and also perhaps because albumin is dissolved by leukolysin from the white corpuscles. If an area of embryonic tissue is invaded by many pyogenic micro-organisms, it is promptly peptonized. The peptonizing action is upon the fibrinous elements of an exudate and upon the intercellular substance of embryonic or granulation tissue. Cells are separated from intercellular substance, and in consequence degenerate and die. Peptonized exudate or peptonized embryonic tissue is called pus. In suppurations induced by staphylococci a barrier of leukocytes is first formed around the region of irritation; this barrier is reinforced by fibroblasts, the pus is imprisoned, and rapid spreading and wide diffusion are prevented. In inflammations induced by streptococci the peptonizing action of the organisms is so great that no barrier of white blood-cells or of proliferating connective-tissue cells forms in time to imprison the micro-organisms, hence the suppuration spreads rapidly and widely. During the existence of a streptococcic infection some bacteria always enter the blood. Suppuration can be induced by the injection of pyogenic bacteria, by their entry through a wound, and by rubbing them into the skin. In some rare instances, especially when the diet has been putrid, they may enter through the blood and lodge at a point of least resistance. When a medullary canal suppurates after a chill to the surface or after a blow that does not cause a wound, we know that the bacteria must have arrived by means of the blood. Bacteria which reach a point of least resistance through the blood come from some atrium of infection which may be discoverable or which may not be found. The entry of pyogenic bacteria does not necessarily cause suppuration, as the healthy human body can destroy a considerable number, even if given in one "dose"; but a large number in a healthy, or even a small number in an unhealthy, body almost certainly leads to pus formation. The pus of all acute abscesses contains bacteria of suppuration, but the pus of tuberculous abscesses does not, unless there be a mixed infection; in other words, pure tuberculous pus is not pus at all.

Can suppuration be induced without the actual presence of bacteria? It is true that the injection of irritants can cause the formation of a thin fluid which contains no bacteria, but this non-bacterial fluid is not pus. A purulent fluid is formed by injecting cultures of pus cocci which have been rendered sterile by heat, the bacteria having been killed, a ferment contained in the bacterial cells being the active agent. Purulent material also results from the injection simply of the sterile products of the growth of pyogenic cocci. This purulent or sterile fluid is known as *spurious* or *aseptic pus*. An area of such aseptic suppuration does not tend to spread and the process concerns us but little as surgeons, except in cases of pyemia, in which thrombi containing toxins alone may occasionally induce limited secondary abscesses.

Impaired health or an area of lowered vitality predisposes to suppuration. Diabetes and albuminuria are common and influential predisposing causes, because in these diseases tissue resistance is always at a low ebb. In

amyloid disease resistance to pus cocci is greatly impaired. It is lessened in lithemia and in any condition of ill health. The lymphatic glands, medulla of bones, serous membranes, and connective tissue are especially prone to suppurate.

Pus may form within twenty-four hours after bacteria have been deposited, or it may not be formed for days. The older surgeons claimed that pus could do good by protecting granulations and separating disorganized tissue. It is now held that it is absolutely harmful by melting down sound tissue and poisoning the entire organism. Modern surgery has to a great degree abolished pus.

If pus stands for a time, it separates into two portions—(1) a watery portion, the liquor puris or pus-serum, containing peptone, fat, microbic products, osmazone, and salts, and not tending to coagulate; (2) a solid portion, or sediment composed of dead and living micro-organisms of suppuration, connective-tissue cells, often epithelial cells, perhaps red blood-cells, lymphocytes, pus-corpuscles (Fig. 71), débris of tissue, and shreds of dead tissue. The

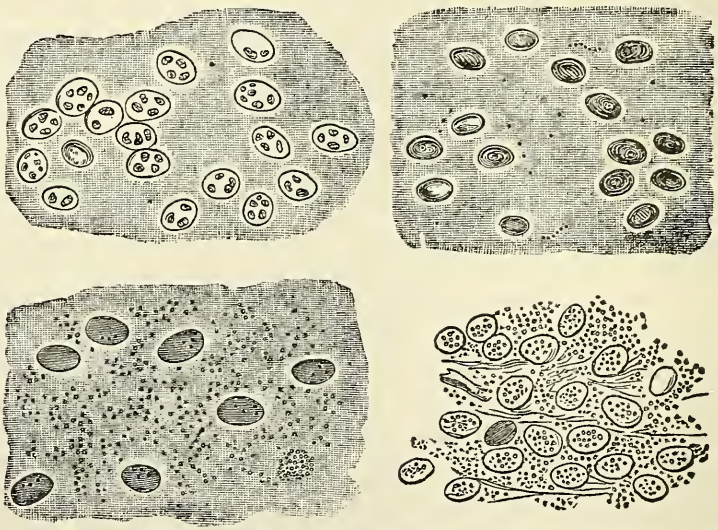


Fig. 71.—Fragmentation of nucleus in leukocytes undergoing transformation into pus-corpuscles (Senn).

pus-corpuscles are either polynuclear white blood-cells or altered connective-tissue cells containing many nuclei. Some of them are dead, some have ameboid movements, some are fatty, others are granular and contain more than one nucleus, and all are degenerating. A pus-cell is waste matter, and it cannot aid in repair. Very exceptionally pus disappears by absorption, by caseation, or by calcification.

**Pus in General.**—The color of pus is variable and depends upon the nature of the bacteria; the presence or absence of blood, fibrin, body secretions or body excretions (bile, urine, mucus, feces, etc.); and the existence or non-existence of putrefaction.

Its consistence varies. In some cases it is scarcely thicker than water, in others it is like cream, and in still others it is cheesy. Thick pus is opaque and of a greenish-yellow color, and thin pus has a distinct reddish or yellowish tinge. When freshly evacuated many varieties are almost or quite odorless, and are alkaline or slightly acid in reaction.

Some varieties possess a very offensive odor. Pus contaminated by the

bacteria of putrefaction is certain to have a foul odor. Pus which forms in the tonsil, in the brain, about the vermiform appendix, or around the rectum usually possesses an offensive odor.

**Forms of Pus.**—*Laudable*, or *healthy pus*, a name long in vogue, is a contradiction, no pus being healthy. In former days free suppuration after an operation was regarded as a favorable indication, and when it occurred the surgeon congratulated himself that surgical fever was at an end. At the present day suppuration after an operation is an evidence of previous infection, of lack of care, failure in our precautions, or of infection by the blood. The so-called *laudable pus* is seen coming from a healing ulcer, and is an opaque, yellowish-white, or a greenish fluid of the consistence of cream, without odor or with a very slight odor if it is not putrid, and having a specific gravity of about 1030.

*Malignant*, *watery*, or *ichorous pus* is a thin, watery, putrid fluid. It is pus filled with the organisms of putrefaction.

*Stinking pus* may be ichorous. Its odor may be due to the Bacterium coli commune. If this bacterium is the cause the pus is very foul, but not thin. Pus of this nature is met with in ischiorectal abscess and appendiceal abscess. The odor of stinking pus may be due to ordinary bacteria of putrefaction, in which case the pus is usually thin.

*Sanious pus* is a form of ichorous pus containing blood coloring-matter or blood. It is thin, of a reddish color, and very acrid, corroding the parts with which it comes in contact. It is found notably in caries and carcinoma.

*Concrete* or *fibrinous pus*, which contains flakes of fibrin or coagulated fibropurulent masses, is met with in serous cavities (joints, pleura, etc.). These masses also form in infective endocarditis.

*Red pus* signifies the presence of the Bacillus prodigiosus.

*Blue Pus.*—The color of blue pus is due to the Bacillus pyocyaneus.

*Orange Pus.*—The color of orange pus is due either to the action of Sarcina aurantiaca, or to the formation of crystals of hematoidin from the coloring-matter of red blood-cells which have been mingled with the pus. Pus of this color appears only in violent inflammations.

*Serous pus* is a thin, serous fluid, containing few flakes.

So-called *tuberculous*, *scrofulous*, or *curdy pus* is not pus at all, unless the tuberculous area has undergone pyogenic infection.

So-called *gummy pus* arises from the breaking down of a gumma which has outgrown its own blood-supply. It is not pus.

*Mucopus* is found in purulent catarrh—that is, in suppurative inflammation of an epithelial structure. It contains pus elements and epithelial cells.

*Caseous pus* comes from the fatty degeneration of pus-corpuscles or inflammatory exudations. It occurs especially in tuberculous processes. A caseous mass may calcify.

**Signs and Symptoms of Suppuration.**—Suppuration is announced by the intensification of all local inflammatory signs. The heat becomes more marked, the discoloration dusky, the swelling augments, the pain becomes throbbing or pulsatile, and the sense of tension is greatly increased. The skin at the focus of the inflammation after a time becomes adherent to the parts beneath, and fluctuation soon appears. This adhesion of the skin is a preparation for a natural opening, and is known as *pointing*. An important sign of pus beneath is edema of the skin. This is always observed in a superficial abscess, and is sometimes noticeable in empyema or pyothorax, in appendiceal abscess, and in perirenal suppuration. The above symptoms can be reinforced and their significance proved by the introduction of an aseptic tubular exploring needle and the discovery of pus. Irregular chills, high fever, drenching sweats, weakness, and a feeling of serious sickness are very significant of suppuration in an

important structure or of a large area. It must always be remembered that in some virulent pyogenic infections the human organism is so overwhelmed with toxins that, though the patient is desperately ill, the temperature is normal or even subnormal. This means that body resistance has abandoned the conflict. In abscess of the brain the temperature, after an initial rise, often becomes normal or subnormal.

**Diffuse Cellulitis or Phlegmonous Suppuration (Purulent Infiltration).**—This process may involve a small area or an entire limb, and is due to infection by the Streptococcus pyogenes (streptococcus of erysipelas), usually associated with mixed infection with other bacteria, particularly the bacteria of putrefaction. The streptococci are intensely virulent. Barriers of white corpuscles do not form early enough to restrain them, and tissues break down before cellular multiplication is able to encompass the bacteria. The bacteria disseminate through the lymph-spaces and lymph-vessels. The disease in severe cases produces enormous swelling, areas which feel boggy, a dusky red discoloration, and great burning pain. Gangrene of superficial areas is not unusual, due to thrombosis of vessels or coagulation necrosis from toxins. The discharges of the wound, if a wound exists, are apt to dry up, and the wound becomes foul, dry, and brown. The adjacent lymphatic glands are much enlarged. The disease is ushered in by a chill, which is followed by high oscillating temperature, due to suppurative fever, sapremia, or even septic infection or pyemia. Sweats are noted during periods of falling temperature. Diffuse suppuration tends to arise in an infected compound fracture after extravasation of urine and after the infliction of a wound upon a person broken down in health. It is not unusual after typhoid or scarlet fever, and is typical of phlegmonous erysipelas. The pus is sanious and offensive, and burrows widely in the subcutaneous tissue and intermuscular planes. This diffuse suppuration may widely separate muscles and even lay bare the bones. It is a very grave condition, and may cause death by exhaustion, septic intoxication, septic infection, pyemia, or hemorrhage from a large vessel which has been corroded. *Cellulitis* of a mild degree is due to attenuated streptococci or to staphylococci. An area of cellulitis may surround an infected wound or a stitch-abscess. Its spread is manifested by red lines of lymphangitis running up to the adjacent lymphatic glands. Light cases may not suppurate, the lymphatics carrying off the poison. Any case of cellulitis is, however, a menace, and any severe case is highly dangerous. (See Erysipelas.)

**Wooden, Woody, or Ligneous Phlegmon.**—This condition was fully described by Réclus in 1893. It is chronic inflammation of the cellular tissue and fascia, and is characterized by the production of quantities of fibrous tissue. It occurs in those over fifty years of age. The neck is the region usually involved. It begins with hard swelling of one side or of the front of the neck and for weeks is unaccompanied by any other sign. The swelling may be at first localized, but it spreads slowly and widely and finally comes to involve an extensive area, even perhaps the front of the neck and both sides from the jaw to the collar-bone. It may involve the cervical muscles and thus create rigidity, and it may compress the larynx and trachea and thus interfere with breathing. In most cases there is difficulty in swallowing. After weeks, or perhaps a month or two, the skin becomes edematous and red or rather of a violet hue. There is no fever and rarely pain. The significant facts are the gradually advancing hard swelling long unaccompanied by fever, pain, discoloration, or cutaneous edema. The condition is due to the deposition and multiplication of bacteria, which reach the tissues from the lymph-glands and reach the glands from an area of infection in the pharynx, a salivary gland, or the mouth. Pus does not form at all or only minute encapsulated foci form, probably because the bacteria are of greatly attenuated virulence or because

the local vital resistance is at a high level to these bacteria. Inflammation occurs, there is copious exudation, and enormous amounts of fibrous tissue form. If pus forms, it may discharge spontaneously in six or seven weeks. The causative bacteria are often attenuated pyogenic microbes. In one of Réclus's cases diphtheria bacilli were found, and this case got better after being given antitoxin. Cases have been reported which were caused by pneumococci.

Wooden phlegmon is occasionally found in syphilitics, but it is not a syphilitic condition. Neither Bright's disease nor diabetes has anything to do with its origin. It may be mistaken for actinomycosis or tuberculosis. It is frequently mistaken for sarcoma or carcinoma, in fact, Lange believes it to be cancer. It arises in those who are in ill health rather than in the vigorous or robust. Wooden phlegmon is always dangerous and is sometimes fatal. One of Réclus's cases died of edema of the glottis. We have spoken of woody phlegmon as though it only could involve the neck; as a matter of fact it can involve other parts. Réclus maintains that it can occur in the right iliac fossa, and that perinephric sclerosis is in reality due to it (Powers, in "Jour. Amer. Med. Assoc.," July 20, 1911). A case has been reported by Todd in which the abdominal wall was involved ("Jour. Missouri State Med. Assoc.," February 8, 1912). Duse also reported such a case ("Gazz. d. Osped.," 1910, xxxi). W. W. Grant, of Denver, has reported ligneous phlegmon of the abdominal wall ("Jour. Amer. Med. Assoc.," April 5, 1913). A similar condition may arise in the perineum, after urinary extravasation or fistula formation. Charles A. Powers, of Denver, has reviewed this subject and presented the report of an admirably studied case (Ibid., July 20, 1911).

*Treatment.*—Extirpation, if feasible, is the best plan. It is seldom feasible, and the surgeon instead makes numerous incisions and usually dresses with antiseptic poultices. In these cases free suppuration occasionally occurs after a long delay, and when it does occur a cure may promptly follow evacuation. An autogenous vaccine should be made and injected into the indurated area. The surgeon must be prepared to do tracheotomy should an emergency arise.

**Furuncle,** or **boil,** is an acute and circumscribed inflammation of the deep layer of the true skin and the subcutaneous cellular tissue following bacterial infection of a hair-follicle or a sebaceous gland and resulting in local necrosis of the dermis. A boil is caused by infection of a hair-follicle through a slight wound (by scratching, shaving, etc.) with the Staphylococcus pyogenes aureus. Boils are very common in individuals with Bright's disease, diabetes, gout, lithemia, tuberculosis, and disorders of menstruation and digestion; and crops of boils are apt to appear during convalescence from typhoid fever. Boils are commonest in the spring, and sometimes an epidemic of furunculosis appears in a hospital, a jail, or an asylum.

The **symptoms** of a boil are as follows: a red elevation appears, which stings and itches; this elevation enlarges and becomes dusky in color; a pustule forms that ruptures and gives exit to a very little discharge which forms a crust. Inflammatory infiltration of adjacent connective tissue advances rapidly, and the boil in about three days consists of a large, red, tender, and painful base capped by a pustule and a little crusted discharge. In rare instances, at this stage, absorption occurs, but in most cases the swelling increases, the discoloration becomes darker, the skin becomes edematous, the pain becomes severe and pulsatile, and the center of the boil becomes raised. About the seventh day rupture occurs, pus flows out, and a "*core*" of necrosed tissue is found in the center of a ragged opening. This core consists of the sebaceous gland and hair-follicle, which have undergone coagulation necrosis (Warren). In a day or two more the core will be discharged, and healing by granulation will begin. A *blind boil* lasts only three or four days and has no core. The

constitution often shows reaction during the progress of a boil. Boils may be either single or multiple. The development of one boil after another, or the formation of several boils at once, is known as "*furunculosis*." Boils are commonest upon the neck and the back.

The **treatment** consists in excision or in crucial incision, removal of necrotic tissue, irrigation with peroxid of hydrogen, touching with pure carbolic acid, and the application of hot antiseptic fomentations.

**Aleppo Boil** (*Endemic Boil of the Tropics; Delhi Boil; Oriental Sore, etc.*).—It is not a pyogenic process. Papules appear upon the exposed parts of the body. These papules, which ulcerate, do not cicatrize for at least a year, and leave ineradicable scars. This condition is due to a protozoön. Man is infected by means of flies, lice, or other insects. The Aleppo boil was once apparently confined to India, Arabia, Persia, Egypt, Algeria, etc. Of late it is said to have appeared in Panama, Philippine Islands, and Hawaii.

**Carbuncle** (*benign anthrax*) is a circumscribed infectious inflammation of the deeper layer of the true skin and of the subcutaneous tissue, with fibrinous exudation, multiple foci of necrosis arising, and the tissue adjacent to each necrotic plug becoming gangrenous. The infection takes place through a hair-follicle. It is really a boil with extensive infiltration of adjacent tissues. A boil may become a carbuncle, and pus from a carbuncle inoculated into a healthy person may cause either a boil or a carbuncle. The causative organism seems to be the Staphylococcus pyogenes aureus. Carbuncle is most common in the upper part of the back and on the back of the neck. In this region the skin is very thick; each hair-follicle holds only a downy hair, is shallow, and projects but a short distance into the cutis vera. Columns of fatty tissue run from the subcutaneous tissue in an oblique direction to join the point and sides of the hair-follicle. These columns are known as *columnæ adiposæ*, and each one contains a sweat-gland (Fig. 72). When pus runs down one of these columns, it seeks an outlet; it cannot spread easily to the sides, so it slowly works its way to deeper tissue and from one to another interspace and finds its way to the surface through other fatty columns (Warren's "Surgical Pathology") (Fig. 73). When pus finds its way to the surface, an opening forms, hence the numerous foci of pointing; finally a large opening forms (Fig. 74). Carbuncles are most common in the spring of the year. In persons with diabetes and Bright's disease carbuncles not unusually occur.

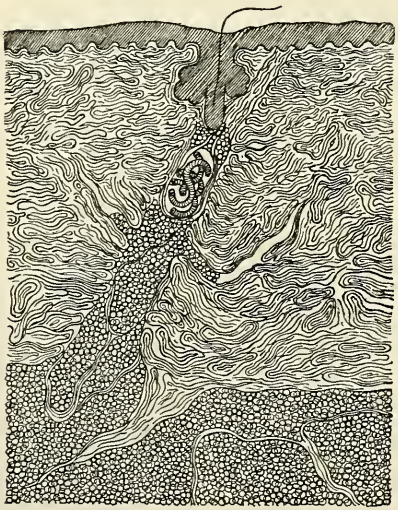


Fig. 72.—Columna adiposa (Warren).

The local **symptoms** in the beginning resemble those of a boil, but the constitution sympathizes from the very start (perhaps a chill and always a septic fever) and the pain is usually severe. The inflammatory area begins as a papule with an indurated base, it enlarges enormously, is boggy to the touch, is dusky in color, is edematous, and the skin is not freely movable over the deeper parts. In a few days many pustules appear, each pustule marking the site of a focus of necrosis. Large vesicles filled with bloody

serum very frequently form. In some cases about the tenth day the pustules rupture, the necrotic plugs are discharged, and the case slowly progresses toward cure; but in many cases the carbuncle spreads at the periphery while pustules are rupturing near the center of inflammation, and pus forms in the deeper tissues, reaching the surface through many small openings, each of which is partly blocked by a plug of dead tissue. A carbuncle in this stage

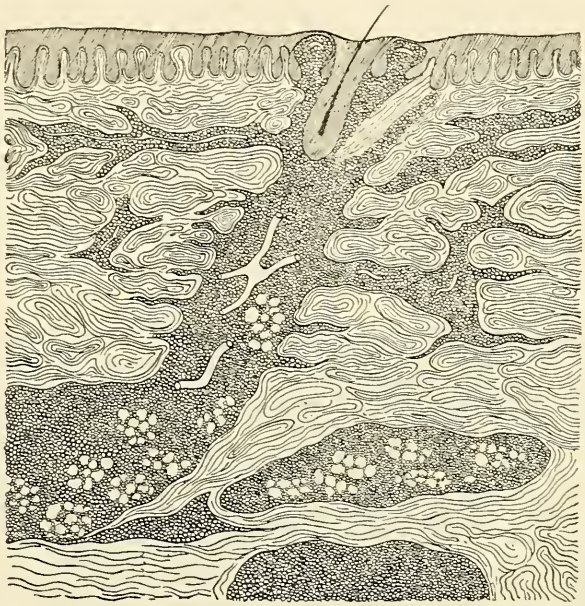


Fig. 73.—Infiltration of columna adiposa and subcutaneous tissue with pus in carbuncle (Warren).

resembles a honeycomb (Fig. 74), discharges bloody pus, and large masses of skin and subcutaneous tissue are destroyed. The entire carbuncular mass may become gangrenous, and a sudden and almost complete cessation of pain points to this complication. An ordinary carbuncle remains acute for about three weeks, but healing requires a month or more. The most dangerous situations in which to have a carbuncle are the face and neck (tends to produce septic

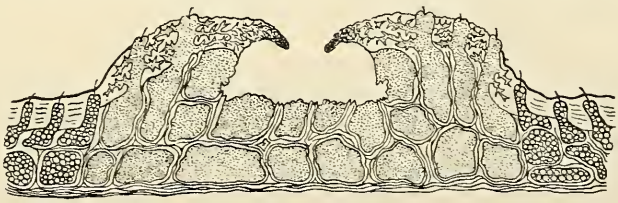


Fig. 74.—Diagram of a carbuncle (Warren).

phlebitis, septic clots in the facial, jugular, or ophthalmic veins, or in the cerebral sinuses, or infective emboli). The mortality of facial carbuncle is at least 50 per cent. The most usual positions for carbuncle are the neck, the back, and the buttocks. The diagnosis of carbuncle is made by noting the multiple foci of necrosis and the profound constitutional involvement. A carbuncle may produce death by causing septicemia, pyemia, or profuse hemorrhage.

**Treatment.**—Some have suggested the treatment of a carbuncle in an early stage by injecting 5 to 30 drops of carbolic acid (80 per cent.) into and around the inflammatory mass. Such a method does not promise success and necessitates dangerous delay. The best treatment if the case is seen sufficiently early is thorough extirpation while the patient is anesthetized. The entire area of the infection is thus removed, and the large wound heals by granulation and is subsequently skin-grafted. When the condition is too far advanced to admit of complete extirpation, the following useful plan should be employed:

Give ether, make free crucial incisions, remove dead and necrosing tissue and also the points of the skin-flaps with the scissors and forceps, curet pockets, arrest hemorrhage by pressure and hot water, cauterize with *pure* carbolic acid, dust with iodoform, pack with iodoform gauze, and dress with hot antiseptic fomentations. Cover the gauze with a piece of some impermeable material and lay a hot-water bag upon the dressing. Every day, or several times a day, remove the dressings, wash with peroxid of hydrogen, irrigate with corrosive sublimate solution, dust with iodoform, and reapply the iodoform gauze and antiseptic fomentation. Keep up this treatment until sloughs are separated,

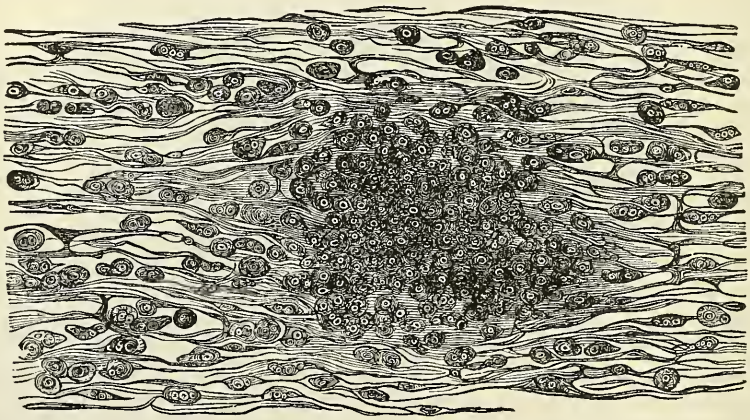


Fig. 75.—Infiltration of connective tissue of cutis (× 500) with beginning suppuration in the center (Senn).

then dress with dry antiseptic gauze. Secure sleep by morphin, give quinin, milk-punch, and nourishing diet, and maintain the action of the bowels and kidneys.

**Acute Abscesses.**—An acute abscess is a circumscribed cavity of new formation containing pus. We emphasize the fact that it is a *circumscribed cavity* —circumscribed by a mass of leukocytes and proliferating fibroblasts. A *purulent infiltration* is not circumscribed, hence it does not constitute an abscess. An essential part of the definition is the assertion that the pus is in a cavity of *new* formation, in an abnormal cavity; hence pus in a natural cavity (pleural, pericardial, synovial, or peritoneal) constitutes a *purulent effusion*, and not an abscess, unless it is encysted in these localities by walls formed of inflammatory tissue.

An acute abscess is due to the deposition and multiplication of pyogenic bacteria in the tissues or in inflammatory exudates. These bacteria attack exudates or tissues, form irritants which cause inflammation or intensify existing inflammation, and by exerting a peptonizing action on intercellular substance and the fibrin of the exudate, liquefy tissue and the products of inflammation, and form pus. As a rule, within twenty-four hours after lodg-

ment of the bacteria the exudation increases in amount, the migrated leukocytes gather in enormous numbers, the fibers of tissues swell, and the connective-tissue spaces distend with cells and fluid. The connective-tissue cells, acted on by pus cocci, multiply by karyokinesis, develop many nuclei, lose their stellate projections, degenerate, and constitute one form of pus-corpuscle, leukocytes forming the other. All the small vessels are choked with leukocytes, this blocking serving to cut off nourishment and tending to produce anemic necrosis. Liquefaction occurs at many foci of the inflammation, drops of pus being formed, the amount of each being progressively added to and many foci coalescing (Fig. 75). The pus-cavity is circumscribed, not by a secreting pyogenic membrane, but by a mass of fibroblasts, whose cells and intercellular material have not as yet broken down; such a mass of fibroblasts is often called embryonic tissue, and it is circumscribed by a zone of inflammation in which there are hordes of migrated leukocytes (Fig. 76). As an abscess increases in size, the embryonic tissue from within outward liquefies into pus, and the zone of inflammation beyond continually enlarges and forms more embryonic tissue. After a time the inflammation reaches the surface, the embryonic tissue glues the superficial to the deeper parts, the superficial parts inflame and become embryonic tissue, and the intercellular substance is liquefied. When pus has all but reached the surface, a thin layer of tissue only being undestroyed, an elevation or tit of thin tissue is formed, due to the fluid pressure. This process is known as *pointing*. The elevation or point thins from tension and liquefaction, and finally gives way and *spontaneous evacuation* occurs. When an abscess forms in an internal organ or in some structure which is not loose, like connective tissue—for instance, in a lymphatic gland—a mass of pyogenic bacteria, floating in the blood or lymph, lodges, and these bacteria by means of irritant products cause coagulation necrosis of the adjacent tissue and inflammatory exudation around it. The area of coagulation necrosis becomes filled with white blood-cells, and the dry necrosed part is liquefied by the cocci. Suppuration in dense structures causes considerable masses of tissue to die and to be cast off, and these masses float in the pus. Death of a mass with dissolution of its elements is necrosis, or inflammatory gangrene. Pus travels in the line of least resistance. It may reach a free surface, or may break into a cavity or joint, may invade bone or destroy a vessel. When an abscess ceases to spread or is evacuated, the fibroblastic layer forming the walls becomes vascularized and is converted into *granulation tissue*. An abscess heals by the collapse of its walls and fusion of the granulations (union by third intention), or by granulation (union by second intention). In either case granulation tissue is ultimately converted into fibrous or scar tissue.

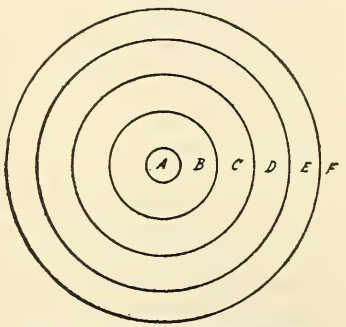



Fig. 76.—Diagram of an abscess: *A*, Pus; *B*, layer of fibroblasts; *C*, tissue infiltrated with leukocytes; *D*, zone of stasis; *E*, zone of active hyperemia; *F*, healthy tissue.

**Forms of Abscesses.**—The following are the various forms of abscesses: *Acute*, which follows an acute inflammation. *Strumous, cold, lymphatic, tuberculous*, or *chronic* abscess is due to the bacilli of tuberculosis, and does not contain true pus unless there is secondary pyogenic infection. It presents no signs of inflammation. Such abscess occasionally forms in a week or two, and hence is not necessarily chronic. *Caseous* or *cheesy* abscess, a cavity containing thick cheesy masses, is due perhaps to the fatty degeneration of inflammatory exudate and pus-corpuscles, but most commonly results from the caseation of a tuber-

culous focus. *Circumscribed* abscess is one limited by a layer of fibroblasts. *Diffused* abscess is an unlimited collection of pus, in reality not an abscess, but either a purulent effusion or a purulent infiltration. *Congestive, gravitative, wandering,* or *hypostatic* abscess is a collection of pus or tuberculous matter which travels from its formation point and appears at some distant spot (as a psoas abscess). *Critical* or *consecutive* abscess is one which arises during an acute disease. *Diathetic* abscess finds its predisposing cause in a diathesis. *Embolic* abscess is due to an infected embolus. *Tympanitic* or *emphysematous* abscess is one which contains air or the gases of putrefaction. *Encysted* abscess, in which pus is circumscribed in a serous cavity. *Fecal* or *stercoraceous* abscess is one containing feces in consequence of a communication with the bowel. *Follicular* abscess is one arising in a follicle; *hematic* abscess, one arising around a blood-clot, as a suppurating hematoma; *marginal* abscess, which appears upon the margin of the anus. *Pyemic* or *metastatic* abscess is the embolic abscess of pyemia. *Milk* abscess is an abscess in the breast in a nursing woman. *Ossifluent* abscess arises from diseased bone. *Psoas* abscess is a tuberculous abscess arising from vertebral caries, the matter following the psoas muscle, and usually pointing in the groin (see page 241). A *sympathetic* abscess, arising some distance from the exciting cause, such as a suppurating bubo from chancroid, is not in reality sympathetic, because infective material has been carried from the primary focus. *Thecal* abscess is a purulent effusion in a tendon-sheath. *Tropical* abscess is an abscess of the liver, so named because it occurs chiefly in those dwelling in tropical countries: it usually follows dysentery. *Urinary* abscess, caused by extravasated urine. A *verminous* abscess is one which contains intestinal worms and communicates with the bowel. A *syphilitic* abscess occurs in the bones during tertiary syphilis, and is gummatous and not primarily pyogenic. *Brodie's* abscess is a chronic abscess of the head of a long bone, most common in the head of the tibia (see page 497). A *superficial* abscess occurs above the deep fascia; a *deep* abscess occurs below the deep fascia. A *residual* or *Paget's* abscess is a recurrence of active changes; it may be after years around the residue of a former tuberculous abscess (see page 237).

**Symptoms of Acute Abscesses.**—In an acute abscess, as before stated, a part becomes inflamed and a quantity of fibroblasts are formed; fibroblastic tissue is liquefied (as above noted) and pus is produced. An acute abscess can occur in a person of any constitution.

**Local Symptoms.**—Locally, there is intensification of inflammatory signs and enormous increase of the swelling. At first the area is hard, but afterward becomes soft, and it finally fluctuates. The discoloration becomes dusky. The pain becomes throbbing and the sense of tension increases. The pain is greater the denser the implicated tissue and the greater the number of nerves it contains. At every pulse-beat the tension in the abscess increases temporarily, and hence the pain momentarily increases. Pain is increased by a dependent position of the part. There is great tenderness. The pain may be felt at the seat of suppuration or may be referred to some distant point. Tenderness is located at the focus of disease. The cutaneous surface, if the abscess is adjacent, is seen to be polished and edematous, and after a time pointing is observed and fluctuation can be detected. If pus is deeply situated the skin may not be reddened, and perhaps the area of induration cannot be palpated. In such a case there is often rigidity of the muscles overlying the abscess (as in abdominal suppurations), the skin may be edematous (as in deep abscess of the neck), and besides local pain there may be pain due to pressure upon a nerve-trunk, the pain perhaps being referred to a distant point.

**Constitutional Symptoms.**—If there is a small collection of pus in an unimportant structure, there may be no obvious constitutional disturbance.

If the abscess contains much pus or affects an important part, disturbances generally appear, from slight rigors or moderate fever to chills, high temperature, and drenching sweats. The constitutional condition typical of an abscess is due to the absorption of retained toxins, and is known as "suppurative fever." When an abscess is open but ill-drained, or when it is unopened and deep-seated, long-continued suppuration causes a fever which is markedly periodic: the temperature rises in the evening, attaining its highest point usually between 4 and 8 P. M., and sinks to normal or nearly normal in the early morning (from 4 to 8 A. M.). When the temperature begins to fall, profuse sweating takes place. This fever is known as *hectic*. As previously stated, the temperature may be normal or subnormal in abscess of the brain. Prolonged suppuration causes albuminoid changes in various organs, notably in the liver, spleen, and kidneys. Albuminoid changes are especially common when there has been mixed infection of a tuberculous area and long-continued suppuration. It also occurs as a result of syphilis.

J. C. DaCosta, Jr. ("Clinical Hematology") tells us that "in both trivial and extensive pus foci the number of leukocytes may be normal or even subnormal; in the former instance because systemic reaction is not provoked, and in the latter because it is overpowered. Leukocytosis may also be absent in case toxic absorption is impossible, owing to the complete walling off of the abscess. In all other instances save these a definite and usually well-marked leukocytosis occurs, amounting on the average to a count of about twice the mean normal standard, but frequently greatly exceeding this figure in the individual case."

The signs and symptoms of an abscess are somewhat modified by location, and it is wise to discuss acute abscesses in different situations.

**Acute Abscesses in Various Regions.**—*Abscess of the brain* may follow cerebral concussion or fracture of the skull, may arise during a general infection, but in about 50 per cent. of cases results from chronic suppurative disease of the middle ear. In *abscess* of a silent region of the *brain* symptoms may long be entirely absent. The usual symptoms are a temporary initial rise of temperature, which soon gives place to a normal and in one-half of the cases to a subnormal temperature, headache, vomiting, delirium, drowsiness, and choked disk. Localizing symptoms, spasmodic or paralytic, may be present. There is usually but not always leukocytosis. In but few uncomplicated cases are there elevated temperature and sweats. Toward the end of the case there may be elevated temperature and delirium. In extradural abscess there is fever from beginning to end (see page 805).

*Appendiceal* or *appendicular abscess* results from inflammation, usually but not always with perforation of the vermiform appendix, plastic peritonitis leading to agglutination of the mesentery and omentum, adhesion of the bowels and mesentery, and the formation of a barrier of leukocytes and a mass of fibroblasts. This process circumscribes the pus. If the pus in suppurative appendicitis has been formed by colon bacilli or staphylococci, it will probably be circumscribed and limited. If the pus has been formed by streptococci, it will probably not be limited, and the peritoneum will be attacked by diffuse septic peritonitis. The signs of appendicular abscess are pain, tenderness, muscular rigidity, and the existence of a mass in the right iliac fossa. The mass may be palpated through the abdominal wall or perhaps the rectum and is dull on percussion. There may be vomiting, and sometimes constipation and sometimes diarrhea. Very seldom is there skin edema and fluctuation. The patient lies upon his back, usually with one or both thighs flexed. In appendicular abscess there is fever, usually higher at night than in the morning, profuse sweating occurring during the fall. In some cases the temperature is persistently high; in some the elevation is trivial; in some chills occur. A sud-

den fall of temperature with shock is produced by rupture of the abscess wall. If this accident happens, general peritonitis quickly arises. In appendicular abscess there is marked leukocytosis, unless the walls are very thick or unless the process has diffused and general peritonitis has taken place, in which conditions it may be absent. Appendiceal abscess may be assumed to exist when the symptoms of appendicitis persist after the fifth or sixth day, or when, after the symptoms have subsided, they reappear a day or two later (see page 1006).

*Abscess of the liver* may not be announced by symptoms until rupture. It may follow dysentery, may be a result of the lodgment of infected clots from the hemorrhoid veins, may follow upon the infective phlebitis of appendicitis, may result from septic cholangitis or suppuration of a hydatid cyst. Abscess from dysentery is apt to be solitary. Portal infection induces multiple abscesses. We speak now of solitary abscess. The bacterial origin of this is in doubt. Ameba are usually present. We usually find fever of an intermittent type, profuse sweats, pain in the back, the right shoulder, or the right hypochondriac region, enlargement of the area of liver-dulness, and hepatic tenderness. Sometimes there are fluctuation and skin edema over the liver, and the general cutaneous surface may be a little jaundiced. The symptoms vary as the pus invades adjacent organs. When there are pain on respiration and evidences of diaphragmatic pleuritis, the pus is probably breaking into the pleural sac. There may or may not be leukocytosis (see page 1035).

*Deep Abscess of the Neck.*—The majority of these abscesses are due to suppuration of lymph-glands, bacteria having reached the glands from an adjacent area of infection, cutaneous, mucous, or osseous. Suppuration beneath the deep fascia induces dusky discoloration of the surface, great pain, extensive edematous swelling, and often interference with respiration. The constitutional evidences of suppuration are noted. Acute suppuration under the deep fascia of the submaxillary region causes extensive inflammatory edema, interference with respiration and deglutition, violent constitutional symptoms, and often sloughing of tissues (see Ludwig's Angina). A deep abscess over the carotid artery is lifted by each arterial beat and may be mistaken for aneurysm, but the pulsation is not expansile. The pus of a deep cervical abscess may track its way into the mediastinum or axilla, or the abscess may break into a large blood-vessel, the pharynx, the wind-pipe, or the gullet.

*Axillary Abscess.*—Superficial abscesses are usually multiple, are in reality furuncles, and result from infection of the sweat-glands and hair-follicles.

Deep abscesses are in most instances due to suppuration of the axillary lymph-glands. The most common cause is an infected wound or a focus of suppuration about the hand, forearm, arm, or chest, but it may result from caries of a rib or may follow a deep cervical abscess. An axillary abscess may be lifted at each beat of the artery and to this extent it resembles an aneurysm, but the pulsation is not expansile.

*Acute retropharyngeal abscess* is due to pyogenic infection of the retropharyngeal tissues. The abscess usually forms upon one of the lateral halves of the pharynx. It may be due to traumatism, to acute infectious diseases, to infective processes of the mucous membrane of the mouth, ear, and nasopharynx, or to pyogenic infection of a tuberculous abscess. In the great majority of cases the disease is due to suppuration of the deep cervical glands. There is pain, difficulty in swallowing, dyspnea, nasal voice, bulging into the pharynx, which is detected by inspection and palpation, enlargement of the deep cervical glands, fever, sweats, and great weakness. Tuberculous Retropharyngeal Abscess is considered on page 241.

*Subphrenic* or *subdiaphragmatic abscess* is apt to begin beneath the diaphragm, though in some few instances the pus forms above this muscle, and sub-

sequently gains access to the region beneath. Such an abscess may contain not only pus, but gas, and in some cases also fluid from the stomach or intestine. The gas of a subphrenic abscess may have entered from a perforation of a hollow viscus or may have been made by gas-forming bacteria. Subphrenic abscess may arise after perforation of the bowel or stomach, or it may result from Pott's disease, perinephric abscess, traumatism, abscess of liver, kidney, spleen or pancreas, empyema, or pneumonia (Greig Smith). Inflammation of the gall-bladder or appendicitis may cause it. The symptoms are pain, fever, sweats, dyspnea, cough, and the physical signs of a collection of fluid beneath the diaphragm and often of gas in the cavity of the abscess. There is usually leukocytosis (see page 143).

*Abscess of the lung* gives the physical signs of a cavity; the expectoration is offensive and contains fragments of lung-tissue. An abscess may often be located by the use of the *x*-rays. Pyemic abscesses may exist and yet escape discovery. (See Surgery of Respiratory Organs.)

*Abscess of the mediastinum* may arise secondarily to deep abscess of the neck or vertebral suppuration; suppuration of the mediastinal glands, lung, or pleura; caries of a rib or of the sternum, ulceration of the esophagus or pericarditis. It causes throbbing retrosternal pain, pain in the back, chills, fever, sweats, irregular pulse, and often dyspnea. A lump may appear which pulsates and fluctuates, but the pulsation is not expansile.

*Perinephric abscess* usually causes tenderness and pain in the lumbar region or about the hip-joint, which pain runs down the thigh and is accompanied by retraction of the testicle. Induration, fluctuation, or edema of the skin may be observed in the lumbar region. The constitutional symptoms of suppuration usually exist (see page 143). There is a high leukocytosis.

*Abscess* or *empyema of the antrum of Highmore* is a collection of pus within the maxillary antrum. It results from inflammation of the jaw, the teeth, or the mucous membrane of the nose. It causes pain, edematous swelling of the overlying soft parts, and crepitation on pressure upon the superior maxillary bone. Pus may escape from the nostril of the diseased side when the head is bent in the direction of the healthy side. A rhinoscopic examination discloses the fluid passing into the nares. The antrum on the side of the abscess cannot be transilluminated by an electric light in the mouth (Garel's sign). The constitutional symptoms of suppuration usually arise.

*Alveolar abscess* is suppurative dental periostitis due to diseased teeth. The simplest form is a *gum-boil*, a collection of pus between the gum and the bone external to the inflamed root of a tooth. In more severe cases the suppuration begins within the tooth socket and the pus escapes around the neck of the tooth; a distinct and local abscess may be situated at the end of the root, absorption of bone having occurred, or a considerable cavity may form in the bone, the external maxillary plate being perforated. In the very severe cases the cheek is involved. An alveolar abscess may break through the gum into the mouth or it may break externally through the cheek. Alveolar abscess causes intense pulsatile pain, marked swelling of the gum and cheek, and sometimes very great edematous and dusky swelling of the face. A sinus may follow its evacuation. Dead bone may form.

*Abscess of the larynx* invariably causes laryngeal edema, which obstructs respiration and puts life in jeopardy. Such an abscess is most apt to appear upon the oral surface of the epiglottis, but may arise within the larynx. It induces violent cough, pain, interference with the voice, swallowing, and breathing, and the swelling can often be felt with a finger and can always be seen by the aid of a laryngoscope.

An *ischiorectal abscess* is situated in the areolar tissue of the ischiorectal fossa. The pyogenic organisms usually gain entrance to the lymphatics by

way of an abrasion, fissure, or ulceration of the rectum or anus. A perforation made by a foreign body may inaugurate the condition. In rare cases bacteria reach the fossa in the blood-stream. The pain is severe and throbbing; there are great tenderness, redness and edema of skin, induration, and usually the constitutional symptoms of pus formation. Fluctuation is a very late sign because of the density of the fascia.

*Prostatic abscess* may result from catheter infection, from infection of the bladder or urethra, or from traumatism, but the commonest cause is gonorrhea. There may be one abscess, several abscesses, or many abscesses. Pus may break into the rectum, the bladder, or the urethra, or may break externally. A prostatic abscess is manifested by chills, fever, sweats, frequency of micturition, tenderness of the perineum and rectum, and agonizing pain, developing during an attack of acute prostitis. A finger in the rectum can palpate the swollen gland.

*Abscess of the breast* follows absorption of pyogenic bacteria from a fissure or abrasion of the nipple. Some surgeons maintain that the bacteria enter along the milk-ducts, while others assert that they gain entrance by the lymphatics. It is most common in nursing women. Its symptoms are swelling, tenderness, pulsatile pain, dusky discoloration, skin edema, fluctuation, and usually constitutional disorder. (See Mastitis.)

*Orbital abscess* is a diffuse suppuration, due to cellulitis or a collection of pus due to caries or necrosis of the orbital wall, suppuration of the accessory nasal sinus, facial erysipelas, or dental caries. In severe orbital cellulitis the movements of the eye are limited, the lids are very red and edematous, the conjunctiva is red and swollen (chemosis), and, if the case is not promptly relieved, optic neuritis may arise and sloughing of the cornea occur.

*Von Bezold's Abscess.*—In this condition the pus of a suppurating mastoid process breaks through the mastoid near the tip and enters into the sheath of the digastric muscle or the sheath of the sternocleidomastoid. There exist extensive inflammatory swelling of the neck, a history of mastoid trouble, usually a lessened amount of pus from the ear, pain in the neck, and constitutional symptoms. The condition suggests thrombosis of the lateral sinus, but the symptoms are not so violent and are not pyemic as they are in that disease.

*Abscess of the Groin or Pyogenic Bubo.*—Such an abscess may have mounted up from the pelvis, tracked forward from the sacro-iliac joint, or descended in the psoas sheath from the vertebræ, but in a very great majority of cases it is due to suppuration of the lymphatic glands. A bubo may be tuberculous, venereal, or pyogenic. A pyogenic bubo results from an area of infection in the trajectory drained by the lymph-vessels of the inguinal or femoral glands. The glands involved may be superficial or deep. The symptoms are those ordinarily linked with suppuration. Occasionally, the pulsations of the great vessels may lift the mass.

*Abscess of the Popliteal Space.*—This results from traumatism, mixed infection of a tuberculous or syphilitic area, suppuration of the contained lymph-glands, of one of the adjacent bursæ, or of the neighboring bone. In rare cases it arises as a result of suppuration of the sac of an aneurysm. The symptoms are severe pain, swelling, flexion of the knee, and edema of the leg. The pulsations of the popliteal artery may be transmitted to the abscess. These pulsations are not expansile, as in aneurysm. Pus may pass under the deep fascia, up or down the extremity, or may break into the knee-joint.

*Suppurative thecitis* or *felon* is a form of diffuse suppuration. (See Felon.)

*Palmar abscess* is a purulent effusion (see page 721).

*Furuncle* and *carbuncle* are discussed on pages 137 and 138.

*Empyema* is a purulent effusion into the pleural sac (see page 891). It is technically an abscess if it becomes encapsuled.

**Diagnosis.**—The diagnosis of an abscess rests upon—(1) its history; (2) fluctuation; (3) pointing; (4) surface edema; (5) the use of the tubular exploring needle; (6) leukocytosis.

*Fluctuation* is the sensation imparted to a finger held against a sac containing fluid when a wave is started in the fluid by striking the mass with a finger of the other hand. Fluctuation cannot be obtained if the amount of fluid is small. It should never be sought for across a limb, but rather along it, because a false sense of fluctuation can always be obtained across the muscles of the limb. *Pointing* and *surface edema* have been discussed.

A suspected abscess in a part containing large blood-vessels under no circumstance should be opened by a bistoury without knowing that the diagnosis is certainly correct. This knowledge is obtained in some cases by inserting a small aspirating needle and observing the nature of the fluid which exudes. This operation must be performed with aseptic care; otherwise, if there is no abscess, infection may be inaugurated; if there is an abscess, mixed infection may occur. The older operators used a grooved exploring needle, but many able surgeons object to its use, on the ground that when plunged into an infected area pus bathes the track of puncture and may cause infection of

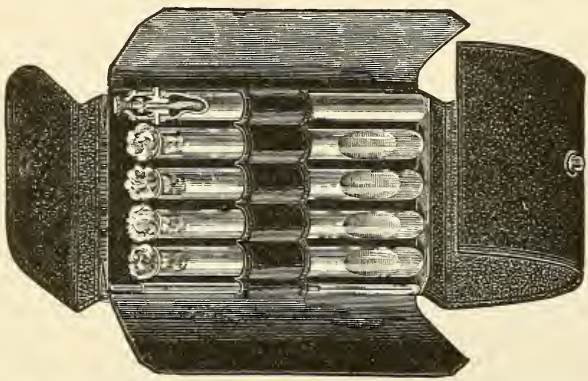


Fig. 77.—Vischer's case for carrying culture-tubes for inoculation.

other tissues and diffusion of the pyogenic process. The tubular exploring needle is the proper instrument.

An abscess which moves with the pulse because it rests upon an artery may be confounded with an aneurysm. The pulse movements of such an abscess are in one direction only; the abscess is lifted with each pulse-beat, but does not enlarge, and if a finger is laid upon either side of it the fingers will be lifted, but not separated. The pulse movements of an aneurysm are in all directions; they are expansile, the sac grows larger, and the fingers will not only be lifted, but will also be separated. The small tubular exploring needle may be used in doubtful cases; if aseptic, it will do no harm even to an aneurysm. A rapidly growing, small-cell sarcoma feels not unlike an abscess, but the exploring needle discovers blood and not pus. A cystic tumor is separated from an abscess by the absence of inflammation, or, if it inflames, by the nature of the contained fluid. Ordinary caution will prevent one confounding an abscess with strangulated hernia. A tuberculous abscess is separated from an acute abscess by the absence of inflammatory signs in the former. The contents of the acute abscess differ from those of the tuberculous abscess. When an abscess exists in an important region (brain, appendix, liver, etc.) cultures of the pus should be taken after incision. Such studies often give valuable information as to the probable course of the condition,

and an accumulation of many accurate observations will add greatly to scientific information. Figure 77 shows a convenient case for carrying culture-tubes.

**Prognosis.**—The prognosis varies according to the number of abscesses, their location and size, the strength of the patient, and the virulence of the causative bacteria.

**Treatment.**—In the treatment of an abscess there is one absolute rule which knows no exception, namely, that whenever and wherever pus is found the abscess should be evacuated at once, and, after evacuating it, thorough drainage must be provided for. It should be opened early, if possible, even before fluctuation and positively before pointing, to prevent tissue destruction, subfascial burrowing, and general contamination. Drainage is continued until the discharge becomes scanty, thin, and seropurulent.

*Alveolar abscess* requires prompt incision through the gum, extraction of the diseased tooth in most cases, and the rinsing of the mouth at frequent intervals with hot fluid. Heat should not be applied to the cheek or jaw externally, as it would favor external rupture. If spontaneous rupture externally is inevitable, then an incision must be made at the point where the abscess is nearest the surface. The cut will leave less scar than will spontaneous evacuation. It is sometimes necessary to gouge through the external table of the bone, pus being lodged within the two osseous plates.

*Abscess of the liver*, if the liver is adherent to the parietal peritoneum, is opened at one operation; if the liver is not adherent, some surgeons operate in two stages. In the two-stage operation an incision is made along the edge of the ribs down to the liver, which organ is then stitched to the edges of the wound. In a day or two after the first operation the two layers of peritoneum are firmly adherent, and the abscess can be opened without danger of the passage of pus into the peritoneal cavity. The abscess, located by an aspirating needle, is opened by the Paquelin cautery, is washed out with salt solution, and a tube is inserted. If care is taken, the operation can be safely completed in one séance even if the liver is not adherent to the parietal peritoneum. If this course is determined on, after the liver is exposed by incision, the exposed surface of the organ is surrounded with iodoform gauze, the abscess is located by an aspirating needle, is opened by the cautery, is irrigated and drained as directed above. Some physicians try to locate an abscess by plunging an aspirating needle into the liver before making an abdominal incision. This procedure seems to me uncertain and dangerous.

*Abscess of the dome of the liver* may be reached by resecting a rib, incising the pleura, and opening through the diaphragm (transthoracic hepatotomy).

*Abscess of the mediastinum*, like all other abscesses, requires incision and drainage. This is effected, if the abscess can be reached from in front, by cutting between the rib cartilages or by trephining the sternum. Abscess of the posterior mediastinum can be reached only by resecting portions of several ribs near their vertebral ends.

In *abscess of the lung* an incision is made and the pleura is exposed. The incision is usually through an intercostal space; but if the spaces are narrow, it will be necessary to resect a rib. If the two layers of pleura are found adherent, the operation is proceeded with. If they are not adherent, they are stitched together with catgut sutures, and the surgeon waits forty-eight hours before continuing. This precaution is taken in order to prevent collapse of the lung from acute traumatic pneumothorax, and to save the pleura from receiving pus during operation. The operation is completed by locating the pus by means of an aspirating needle, evacuating it by the cautery at a dull-red heat, and inserting a drainage-tube into the abscess-cavity.

A *subphrenic abscess* requires operation at once. Immediately before operating, if in doubt, it may be justifiable to endeavor to locate pus with an aspirating needle. Incise the abscess and open any secondary abscesses. Many abscesses point below the diaphragm, and are easily reached by an incision in the loin or in the epigastric region. Lannelonge resects the eleventh and twelfth ribs and raises the pleura out of the way. Some surgeons prefer to practice rib resection and incise the adherent pleural layers and the diaphragm. After drainage has been continued for a time it may be necessary to do a secondary operation in order to cure the lesion causative of the abscess, for instance, it may be necessary to close a chronic gastric perforation.

In *abscess of the antrum of Highmore* bore a gimlet-hole through the superior maxillary bone, above the canine tooth, or perforate the bone by means of a trocar. Irrigate daily with boiled water or normal salt solution. Keep the opening from contracting by inserting a small tent of iodoform gauze. In persistent cases it may be necessary to draw a tooth, break through the socket of the first or second bicuspid into the antrum, and insert a silver or hard-rubber tube, and also to perforate the antrum from the inferior meatus and keep the opening patent. In very persistent cases osteoplastic resection of a portion of the upper jaw will be demanded.

In *appendicular abscess* incise, support the abscess walls with gauze, remove the appendix in most cases, but not in all, and insert a drainage-tube and strands of gauze (see page 1018).

An *ischiorectal abscess* must be opened early. The surgeon never waits for fluctuation. Fluctuation is a very late symptom. To wait for it entails great destruction of tissue and serves no useful purpose. Place the patient on his side, with the legs drawn up. Insert a finger in the rectum, lift the abscess toward the surface, and incise it from the surface. The incision runs from the anal margin like a spoke from the hub of a wheel. Irrigate with salt solution, inject iodoform emulsion, insert a drainage-tube, dress, and let the patient know he is in danger of developing a fistula.

A *retropharyngeal abscess* must be opened early, because delay may lead to fatal obstruction and because if spontaneous evacuation occurs the patient may be suffocated. Some surgeons open it from within the mouth, but this exposes the patient to the danger of septic bronchopneumonia from inhalation of purulent elements and to serious gastro-intestinal disorder from swallowing quantities of pus. Again, if opened through the mouth, the abscess is liable to become putrid. It is better to open it from the neck by Hilton's method, the incision being carried through the sternocleidomastoid muscle or posterior to it. Drainage is inserted and the abscess treated in the usual way.

In *abscess of the breast* make an incision radiating from the nipple, or, what is better, incise under the breast by means of a cut at the inferior thoracic mammary junction and enter the abscess from beneath.

In *abscess of the brain* the skull should be trephined, the membranes incised, and the abscess sought for, opened, and drained (see page 804).

In suppuration within the *orbit* due to cellulitis, incise from the conjunctiva and drain. In suppuration due to caries or necrosis of the upper orbital wall make a transverse incision through the upper lid, reach the pus by Hilton's method (see page 150), remove carious or loose necrotic bone, and drain.

A *perinephric abscess* requires an incision in the lumbar region and free drainage.

An *abscess of the larynx* requires immediate scarification and inhalation of steam to abate swelling. In a severe case the surgeon should at once perform tracheotomy.

*Bezold's abscess* requires one or more incisions in the neck for drainage.

Then the mastoid is exposed, its tip, including the osseous fistula, is removed, and its interior is cleared out by a complete operation.

A *prostatic abscess* should be opened promptly by a perineal incision.

In an ordinary *superficial abscess*, after cleansing the parts, make the skin tense, locate the superficial vessels and nerves, and plan the incision to avoid them. Incise with a sharp-pointed curved bistoury at the most dependent part of the abscess or through the region of pointing. If the abscess is upon the face or neck, make the incision in the line of the skin creases so as to limit the scar. The incision must not be made suddenly and fiercely, neither should it be made with hesitation and uncertainty. Thomas Bryant says: "It should be done, as ought every other act of surgery, with confidence and decision, boldness and rapidity of action being governed by caution and made subservient to safety" ("Practice of Surgery"). Permit the pus to run out spontaneously; pressure, as a rule, is undesirable because it may damage the abscess wall and cause diffusion of the infection. If tissue shreds block the opening, they must be picked out with forceps. If the atmospheric pressure will not cause the pus to flow out, make light pressure with warm, moist, aseptic gauze pads. After the pus has come away gently wash the cavity with normal salt solution or boiled water, and drain with a tube for two or three days when the discharge becomes serous. It is not desirable to overdistend the abscess-cavity with fluid, because the hydrostatic pressure might break down the wall of young cells and infection be diffused. Do not irrigate with powerful disinfectants. They cannot be used strong enough to really disinfect, but may easily be used strong enough to cause necrosis of an abscess wall. Peroxid of hydrogen is not to be used unless the incision is large, because the gas it generates may tear the abscess wall and diffuse the infection. Peroxid of hydrogen is a dangerous agent to inject into the cavity of a deep abscess of the neck, as the liberated gas may not escape from the opening, but may pass widely into the tissues and cause great distention. The author saw a child who narrowly escaped death after such an injection. In this patient the gas passed beneath the pharyngeal mucous membrane and the swelling almost occluded the air-passages. If an abscess contains putrid pus the incision should be free, and after evacuation it should be irrigated with hot salt solution or peroxid of hydrogen and injected with iodoform emulsion. Pursue rigid antisepsis in dealing with purulent areas. It is true we already have infection with pyogenic bacteria, but infection can also take place with organisms of putrefaction, causing pus to become putrid, or with other bacteria, for instance, those of tetanus. If a tube is not used and the cavity is packed with iodoform gauze, remember that gauze will not drain pus and requires to be changed once a day or oftener. An abscess should be dressed with hot, moist, antiseptic dressings (antiseptic fomentation) and the part must be put at rest. When the discharge becomes thin and scanty, dry aseptic or antiseptic dressings are used.

In a *deep abscess* or an abscess situated near important vessels do not boldly plunge in a knife. Hilton says to "plunge in a knife is not courageous, as it is without danger to the surgeon, but may be fatal to the patient." Remember also that a large amount of pus displaces normal anatomical relations. *Hilton's method* of opening a deep abscess (as in the axilla or neck) is to cut to the deep fascia, nick the fascia with a knife, and then push into the abscess a grooved director until pus shows in the groove; along the groove push a pair of closed dressing forceps; after they reach the depths take out the director, open the forceps, and withdraw them while open, and so dilate the opening; then insert a tube and gently irrigate with warm salt solution.

Always endeavor to open an abscess at its most dependent part, remembering that the situation of this part may depend upon whether the patient is to

be erect or recumbent. If we do not make the opening at the lowest point, all the pus will not run out and the walls will not completely collapse. A deep abscess must be drained thoroughly until the discharge becomes seropurulent. When the tube is removed it is wise to insert a tent of iodoform gauze just through the outlet of the abscess. This tent prevents the skin from closing over the channel. It is removed and a new one inserted every day until it is clear that there is no longer danger of fluid becoming blocked and retained. When an abscess contains diverticula or pouches they should be slit up or a counteropening ought to be made. A counteropening is made by entering the dressing forceps at the first incision, pushing them through the abscess to the point where we wish to make our counteropening, opening the blades, and cutting between them from without inward. The blades are then closed and projected through the incision; they are opened in order to dilate the new door, and are closed again upon a drainage-tube, which is pulled through from opening to opening as the instrument is withdrawn. When pus burrows, insert a grooved director in each channel and slit the sinus with a knife. An abscess may make an opening through dense fascia, the opening being small like the neck of an hour-glass (*shirt-stud abscess*). Always examine to see if such a condition exists, and if it is found, incise the fascia.

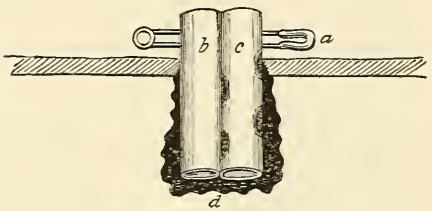


Fig. 78.—Drainage-tubes for abscess requiring irrigation.

In a deep abscess containing putrid pus frequent irrigation is desirable. In such a case two tubes may be employed (Fig. 78). The tubes are prevented from slipping in by the use of a safety-pin (*a*). The irrigating fluid is passed into the cavity (*d*) through the tube *b*, which is without fenestra, and most of it runs out through the tube *c*, which possesses fenestra.

Rest is of the first importance in the healing of an abscess, and we try to obtain it by bandages, splints, and pressure, which will immobilize adjacent muscles and approximate the abscess walls. If an abscess is slow to heal, use as a daily injection a solution of corrosive sublimate of the strength of 1:1000, or 3 drops of nitric acid to 1 oz. of water, or 3 gr. of zinc sulphate to 1 oz. of water, or a 5 per cent. solution of carbolic acid, or a 2 per cent. aqueous solution of pyoktanin, or 20 drops of tincture of iodin to 1 oz. of water, or a 2 per cent. solution of acetate of aluminum. The constitutional treatment of an abscess depends upon the severity of the morbid process and the importance of the structures involved. In a serious case the patient should be put to bed, opiates should be given with a free hand, the bowels be kept active by calomel and salines, skin activity be maintained, the taking of nutritious food insisted on, and stimulants liberally employed.

*Purulent Effusions.*—(See Suppurative Thecitis, Palmar Abscess, Suppurative Synovitis, Purulent Peritonitis, Empyema, etc.)

## VII. ULCERATION AND FISTULA

An **ulcer** is a loss of substance due to molecular death of a superficial structure. The molecular death is brought about by bacteria. Ordinary ulcers are caused by pus organisms. The action of the pus organisms is the same as in an abscess. A broken abscess becomes an ulcer, and an ulcer is in structure a half-section of an abscess. The floor of an ulcer consists of

granulation tissue and corresponds to the abscess wall. An abscess arises from molecular death within the tissues; an ulcer, from molecular death of a free surface. An ulcer may increase in size by molecular death of adjacent structures or by sloughing, that is to say, by death of visible masses of tissue. A wound healing by granulation is often wrongly called an ulcer. An ulcer must not be confounded with an excoriation. In an ulcer the corium is always, and the subcutaneous tissue is generally, destroyed, and a scar is left after healing. In an excoriation the mucous layer of epithelium is exposed, or this is destroyed and the corium is exposed. In an excoriation the corium is never destroyed, and no scar remains after healing. An ulcer heals by granulation (see page 117). Embryonic tissue by vascularization becomes granulation tissue, granulation tissue is converted into fibrous tissue, the fibrous tissue contracts, and by pulling the edges of the ulcer toward each other lessens the size of the cavity. When the granulations reach the level of the skin the epithelium at the edges of the ulcer proliferates and the sore is soon covered over with new epithelium.

**Necrosis** of a superficial part may arise from—(1) Inflammation. The pressure of the exudate can cut off the circulation, or bacteria may directly destroy tissue. Suppuration occurs. (2) The action of pus bacteria, causing primary cell-necrosis. (3) Bacteria of putrefaction and organisms of suppuration acting upon a wound. (4) Traumatism or irritants, producing at once stasis, which is added to by secondary inflammation, the exudate undergoing purulent liquefaction. (5) Prolonged pressure. (6) Deficient blood-supply. (7) Faulty venous return. (8) Degeneration of a neoplastic infiltration (gummatous, malignant, or tuberculous). (9) Trophic disturbance. (10) Nutritional disturbances (as scurvy). Most ulcers are due directly to pus organisms, and areas of necrosis of superficial parts that arise from something else (as gummatous degeneration) suppurate.

**Classification.**—Ulcers are classified into groups according to the condition of the ulcer and the associated constitutional state. In the first group we find the varicose, hemorrhagic, acute, chronic, irritable, neuralgic, etc. In the second group are placed the tuberculous, syphilitic, senile, scorbutic, etc. All ulcers, whatever their origin, are either *acute* or *chronic*, and such conditions as great pain, hemorrhage, edema, exuberant granulations, phagedena, sloughing, eczema, gout, syphilis, scurvy, etc., are to be looked upon as complications. The leg is so common a site for ulcers as to warrant a special description of ulcers of this part. In describing an ulcer state the patient's previous history; the supposed cause; the situation; the outline; the duration, and the mode of onset of the ulcer. State if the ulcer is single or if multiple sores exist, and if there is or is not pain; whether or not any healing has ever occurred, and the patient's constitutional condition. Set forth the complications; the state of anatomically related glands; the condition of the edge, the floor, and the parts about the ulcer, and the nature and quantity of the discharge.

**Acute** or **inflamed ulcer of the leg** may follow an acute inflammation and may be acute from the start, or may be first chronic and then become acute. It is especially common in drunkards, and among those of dilapidated constitutions. It is characterized by rapid progress and intense inflammation. There is rarely more than one ulcer. In outline these ulcers are usually oval, but may be irregular. The floor of an acute ulcer contains no granulations, but is composed of the raw and inflamed tissues, or is covered by a mass of gray aplastic lymph, or it may have upon it large greenish sloughs. The edges are thin and undermined. The discharge is very profuse and ichorous, excoriating the surrounding parts. The adjacent cutaneous surface is inflamed and edematous, and there is much burning pain. In some cases the

glands in the groin enlarge. Constitutionally, there is gastro-intestinal derangement, but rarely fever. When the ulcer spreads with great rapidity and becomes deeper as well as larger in surface area, it is called "phagedenic." The formation of sloughs indicates that tissue death is going on so rapidly that the dead portions have not time to break down and be cast off. Limited stasis produces molecular death; more extensive stasis, a slough. If a chronic ulcer becomes acute, existing granulations are destroyed.

**Treatment.**—In treating an acute ulcer of the leg, give a dose of blue mass or calomel, followed in eight or ten hours by a saline (2 dr. each of Rochelle and Epsom salts), and order light diet. Deny stimulants except in a case of diphtheritic ulcer. Administer opium if pain is severe. Spray the ulcer with hydrogen peroxid, use the scissors and forceps to get rid of sloughs, and after sloughs are removed wash the ulcer with corrosive sublimate solution (1:1000) or paint it with pure carbolic acid. Paint the skin adjacent to the ulcer with equal parts of tincture of iodin and alcohol. Dress with hot antiseptic fomentations. Apply a bandage from the toes to well above the ulcer. Insist on the patient remaining in bed with the leg slightly elevated. Change the dressings before they become cool and always as soon as they are saturated with discharge. Every day or two paint the parts about the ulcer with equal parts of iodin and alcohol.

Many cases do very well after antiseptization and dusting the ulcer with iodoform, lead-water and laudanum being applied to the inflamed parts around the ulcer; but in a bad case hot antiseptic fomentations, compression, and elevation are more useful until sloughs separate. If the discharge is offensive, apply acetanilid, aristol, or iodoform, or use 3 gr. of chloral to 1 oz. of water before applying hot fomentations or ordinary antiseptic dressings. A 25 per cent. ointment of ichthyol is very useful when applied to parts around the ulcer. If sloughs continue to form, touch the sloughing area with a 1:8 solution of acid nitrate of mercury or with a solution of pure carbolic acid, and reapply antiseptic fomentations. If an ulcer continues to spread, cleanse with peroxid of hydrogen, dry with absorbent cotton, touch with nitrate of mercury solution (1:8), and apply antiseptic fomentations. Repeat the application of nitrate of mercury every day until the ulcer ceases to extend and granulations begin to form. When granulations begin to form freely moist hot dressings are no longer desirable, and dry aseptic or antiseptic dressings can be used.

If an ulcer is covered with a great mass of aplastic lymph, touch daily with a solution of silver nitrate (40 gr. to 1 oz.) or with acid nitrate of mercury (1:15), and dress with iodoform and antiseptic fomentations. Give internally tonics, stimulants, and nutritious liquid food. In any case, when granulations form, dress antiseptically with dry dressings, or employ a non-irritant ointment, such as cosmolin. If granulations form slowly touch them every day with a solution of silver nitrate (10 gr. to the oz.) and dress antiseptically, or apply a stimulating ointment (resin cerate or 1 dr. of ung. hydrarg. nitratis to 7 dr. of ung. petrolii, or an ointment of copper sulphate, 3 gr. to the oz.), or dress with gauze soaked in a solution of 3 drops of nitric acid to 1 oz. of gum arabic. When the granulations are healthy cicatrization can be greatly hastened by an application of scarlet red ointment (8 per cent.). This is kept in place for twenty-four hours at a time. It is used intermittently.

**Chronic ulcer of the leg** is characterized by low action and slow progress. It may be chronic from the start, or it may result from acute ulcer. Usually it is found as a solitary ulcer 2 inches above the internal malleolus. *Syphilitic ulcers* often occur in a group, are usually crescentic, and are frequent upon the front of knee. A *tuberculous ulcer* may have no granulations, but is usually covered with pale edematous granulations, which signify

the existence of a tendency to venous stasis. The edges of the tuberculous ulcer are undermined and irregular, the parts about it are livid and tender, and the discharge is thin and scanty (see page 246). An *ordinary chronic ulcer* is circular or oval, and is surrounded by congested, discolored, and indurated skin, this induration being due to fibrous tissue, and there is often eczema or a brown pigmentation of the neighboring skin. The floor of the ulcer is uneven, and usually is covered with granulations, each of which is red and the size of a pin-point, but which may be exuberant or edematous. If granulations are absent, the ulcer has the appearance of a piece of liver, or is smooth and glazed. The edges are thick, turned out, and not sensitive to the touch. Occasionally, but rarely, they are thin and undermined. Some ulcers are indurated and adherent; this adhesion to the deeper structures prevents healing by antagonizing contraction. An ulcer may fail to heal because of severe infection; because of want of rest; because of absence of granulations resulting from deficient blood-supply; because of edematous granulations; because of exuberant granulations; because of adhesion to deep structures, or because of some constitutional disease.

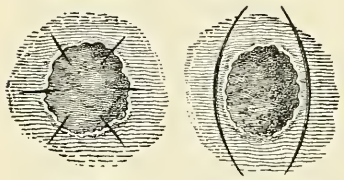


Fig. 79. Fig. 80.

Figs. 79 and 80.—Incisions for adherent ulcer.

**Treatment.**—In treating a chronic ulcer, give a saline cathartic every day or so. Treat any existing diathesis. Insist on rest and, if possible, elevation. Asepticize the ulcer. Draw blood by shallow scarifications of the bottom and edges of the ulcer and the skin about it. If the ulcer is adherent to deeper structures, make incisions like those shown in Figs. 79 and 80, each cut going through the deep fascia. These incisions, besides permitting contraction, allow granulations to sprout in the cuts and absorb exudate. Nussbaum advocated encircling the ulcer with an incision about $\frac{1}{2}$ or $\frac{2}{3}$ inch away from the edge of the ulcer, the incision passing through the skin. After incision keep the part elevated and dressed antiseptically for two days. In two days after scarification or incision scrape the ulcer with a curet until sound tissue is reached. Use hot antiseptic fomentations for two days more, then paint the parts adjacent to the ulcer with tincture of iodin and alcohol (1:3), dress the parts about the ulcer with ichthyol ointment, and dress the ulcer antiseptically or with sterile gauze. In a day or so the use of ichthyol should be discontinued and the ulcer be dressed with sterile gauze, normal salt solution, boric acid, solution of acetate of aluminum, chlorin-water, a solution of permanganate of potassium, sulphur, glutol, protonuclein, or bovinin. Glutol (formalin-gelatin) is very useful in some cases and so is protonuclein. When healing begins, treat as outlined for healing acute ulcer (see page 153). Many cases can be cured by baking in the hot-air apparatus from half an hour to an hour daily. A moderate heat is indicated, and in the intervals of treatment an elastic bandage should be used and, if possible, the patient should be kept in bed.

*Unna's dressing* is satisfactory in many cases. It is applied as a fluid, painted on when hot. It solidifies on cooling and resembles rubber. The paint is made as follows: Dissolve 4 parts of the best gelatin in 10 parts of water by means of a hot-water bath. While the fluid is hot add 10 parts of glycerin, and then 4 parts of powdered white oxid of zinc and stir energetically until the mixture is cold. Melt the paint before using by placing the receptacle in a hot-water bath. The extremity must be clean and thoroughly dry. Apply the paint from just above the roots of the toes to just below the knee. Cover the layer of paint with a gauze bandage; put over this another layer of paint,

then another bandage, and so on until three, four, or five bandages have been applied. To prevent wrinkling apply the gauze in short pieces. The outer layer of the dressing is a coat of the paint. This dressing is worn from four to eight weeks unless it loosens sooner. When it loosens, it is changed. If the ulcer discharges freely and stains the dressing, cut a trap-door in the dressing and through this cleanse the ulcer and apply dressings and a bandage as often as necessary (Michel, in "Chicago Clinic," No. 8, 1900).

An excellent treatment if the patient must walk about is *camphor*, first recommended by Schulze ("Münchener medicinische Wochenschrift," March 19, 1901). It is most conveniently used, as Walbaum shows, in the form of spirits of camphor (Ibid., June 25, 1901). He applies the dressing in the following manner: Clean the ulcer with green soap and dress it daily with dressings wet with a 2 per cent. solution of the acetate of aluminum. In about three days the discharge will become scanty and free from odor. It is at this period that camphor should be used. A small piece of gauze wet with spirits of camphor is applied directly and only to the ulcer. Over this is applied a large piece of dry sterile gauze, a rubber-dam, a large piece of absorbent cotton, and a bandage from the toes up. Every other day the dressings are removed, the ulcer is washed with a 2 per cent. solution of carbolic acid, and the dressings are reapplied. Usually the ulcer is healed in three weeks.

**Complications.**—Remove by scissors and forceps any badly damaged tissue. Take out dead bone; slit sinuses; trim overhanging edges. Treat **eczema** locally by washing with ethereal soap and applying powdered oxid of zinc or borated talcum, the leg then being wrapped in cotton. Unna's paint is very useful in chronic eczema. If the part is crusted, the crusts should be removed by applying some oily materials and washing with ethereal soap and water. Ordinary soap should not be used. In an acute case soap and water always do harm and the part is to be cleaned by "gently wiping with cold cream or petrolatum" (Stelwagon, on "Diseases of the Skin"). If crusting is very marked it may be necessary to remove it by means of an ordinary poultice or, better, a starch poultice made with a 2 per cent. solution of boric acid. When scales or crusts are slight or absent or when they have been removed, the remedial agent should be applied. The remedies for eczema are legion. Among them are a solution of lead acetate; lead-water and laudanum; a powder composed of 30 gr. of powdered boric acid and ½ oz. each of talc and zinc oxid; ung. picis liquidæ, 1 dr., with sufficient ung. zinci oxidi to make 1 oz.; ½ oz. of liquor carbonis detergens to 1 pint of water. In every case of eczema place the patient upon a plain and nutritious diet; order him to avoid wines and liquors; give an occasional saline laxative; keep the skin and kidneys active, and if the patient is gouty or rheumatic, give appropriate remedies. The value of arsenic in eczema has been much overrated.

**Varicose veins** may demand either ligation at several points, excision, Trendelenburg's operation (see page 462), or the continued use of a flannel roller or a Martin rubber bandage. Never operate on varicose veins if phlebitis exists unless a clot has formed, in which case apply a ligature above the clot. Never operate on the veins for varicose ulcer unless the ulcer is in an aseptic state. *Inflammation* of the ulcer is met by rest, elevation, painting the neighboring parts with dilute tincture of iodin, and applying about the ulcer ichthyol ointment. For *calloused edges*, blister, employ radiating incisions, or cut the edges away. Ordinary *thick edges* should be strapped. In strapping use zinc oxid adhesive plaster and do not completely encircle the limb (Fig. 81). When the parts are *adherent* the ulcer is immovable, being firmly anchored to structures beneath it. In such a condition completely or partly surround the sore with a cut through the deep fascia (see Figs. 79, 80). This cut sets the ulcer free

from its anchorage and permits it to contract. *Edematous granulations* require dry dressings and pressure by a flannel bandage, a rubber bandage, or adhesive plaster. If the bottom of the ulcer is *foul*, dry it and touch with a solution of acid nitrate of mercury (1:8) or with crystals of pure carbolic acid. Repeat this every third day and dress with hot antiseptic fomentations until granulations appear. *Superfluous granulations* (proud flesh) should be cut away with scissors, scraped away, or burned down with a strong solution of silver nitrate, with the solid stick of lunar caustic or, better, with pure carbolic acid, which causes much less pain than does silver. *Absence of granulations* or scantiness of granulations means deficiency of blood-supply. The surgeon endeavors to bring more blood to the part, and to do this induces inflammation. The usual method of procedure is to apply daily to the sore a solution of nitrate of silver (10 or 15 gr. to the ounce). Argyrol of a strength of 25 per cent. is not painful and is as efficient. In obstinate cases blister the ulcer or scrape it, or paint it with tincture of iodin, or apply pure carbolic acid, or touch it with the actual cautery. In many cases granulation is greatly stimulated by a twenty-four-hour application of an 8 per cent. ointment of scarlet red. If it causes irritation its use is suspended for a day or two, and then the ointment is reapplied. If an ulcer of the leg becomes painful at one point (see page 157), touch with pure carbolic acid after curetting, or find the painful spot with a probe and divide the exposed nerve filament with a tenotome.

Fig. 81.—Strapping an ulcer of leg ("Keen's Surgery").

If healing entirely fails, *skin-graft*. Among the methods of skin-grafting are—(1) Reverdin's, (2) Thiersch's, and (3) Wolfe's. (See Plastic Surgery.)

When a man having an ulcer must go out and about, the camphor treatment can be employed (see page 155), Unna's dressing may be applied (see page 154), the patient can use a firmly applied roller or, better still, a Martin bandage. Martin's bandage, which is made of red rubber, limits the amount of arterial blood going to the ulcer and favors venous flow from the sore and its neighborhood. The bandage should be used as follows: Before getting out of bed spray the sore with hydrogen peroxid by means of an atomizer, remove the froth with absorbent cotton, wash the leg with soap and water, dry it with a towel, dust the skin with borated talcum powder, and apply the bandage from the toes up. All of these things should be done before putting the foot to the floor. At night, after getting on the bed, remove the bandage, wash it with soap and water, dry it with a towel, hang it unrolled over the back of a chair to air, and again cleanse the leg and ulcer. If these rules are not strictly observed, the Martin bandage will produce pain, suppuration, and eczema of the leg.

**Tuberculous Ulcers.**—(See pages 246, 247.)

**Syphilitic Ulcers.**—(See page 328.)

A **healthy ulcer** is covered with small, bright-red granulations which do not bleed on touching, are painless, and grow rapidly. The edges of the sore are soft and show the opalescent blue line of proliferating epithelium. The sore is movable, the discharge is purulent and yellow, and the parts about are not inflamed.

**Various Ulcers.**—The **fungous** or **exuberant ulcer of the leg** is produced by interference with the return of venous blood from the part, and it is specially common after burns and other injuries when cicatricial contraction causes venous obstruction. The granulations are large, deep red in color, bleed when touched, form rapidly, and mount above the level of the skin. The discharge from a fungous ulcer is profuse, thin, and bloody. In the treatment of such an ulcer venous return must be favored by bandaging and by elevation of the part. If the edges are very thick, divide them in a number of places. The superfluous granulations should be burnt down with lunar caustic or pure carbolic acid or should be cut off. Strapping with adhesive plaster or the use of a rubber bandage does good. The sore can be dressed with europhen, aristol, or dry aseptic gauze.

A **varicose ulcer of the leg** is an ulcer complicated by varicose veins. It is usually single, is oval, round, or irregular in outline, and is most often seen above the inner malleolus. Its edges are thick, everted, and swollen. The swelling is largely due to edema, and is found to pit on pressure. The edges are not undermined, but slope gently to the floor of the ulcer. The floor is usually covered with rather large granulations which bleed freely on touching. In a varicose ulcer the destruction of tissue often begins at the margin of a congested area and advances toward the center. Such an ulcer is usually surrounded by eczema. To aid the healing of a varicose ulcer it is first of all necessary to favor the return of venous blood from the part by position and bandaging. Martin's bandage is very useful and the daily use of the hot-air apparatus is of value. It may be necessary to operate on the veins.

**Erethistic, irritable,** or **painful ulcers** are very sensitive, a condition due to the exposure of nerve-filaments and destruction of nerve-sheaths. Irritable ulcers are especially found near the ankle, over the tibia, in the anus (fissure), or in the matrix of the nail (ingrowing nail). Fissure of the anus is considered on page 1177. An *ingrowing nail* is sometimes encountered on the finger, but far more commonly affects the toe. The great toe is especially apt to suffer. We call it ingrowing nail, but the condition is really overgrowing skin. As a result of wearing ill-fitting boots or stockings, especially shoes which are too short or are pointed, the toes are forced together and the skin at the edge of the nail is squeezed. After a time an ulcer forms.

When a nail begins to ingrow the condition can usually be arrested by wearing well-fitting shoes and stockings, allowing the nail to grow somewhat long and cutting it square across instead of cutting away the troublesome corner. Daily a little absorbent cotton should be packed under the ingrowing corner. In more severe cases, under local anesthesia cut away the overlapping skin and a portion of the flesh on the side of the toe, split the nail longitudinally, remove the ingrown portion of nail and a corresponding part of the matrix.

An erethistic ulcer of the cutaneous surface is treated as follows: Curet and touch with pure carbolic acid or with the solid stick of silver nitrate. Chloral, 20 gr. to the ounce, allays the pain; so do cocain and eucain for a time. In some cases the painful area can be located by a probe and the nerve-filament divided by a tenotome.

The **indolent ulcer of the leg** shows no tendency to heal. In such an ulcer there is usually venous congestion from varicose veins or from cardiac weakness. A great mass of scar-tissue forms at the base and edges, which fastens the ulcer to bone or fascia, so that the edges cannot contract. Healthy granula-

tions cease to form. The edges of such an ulcer are thick, smooth, immovable, and free from tenderness. Granulations are entirely absent or there are seen here and there a few unhealthy granulations. The discharge is thin, sero-

Fig. 82.—Marjolin's ulcer (epithelioma) in a man twenty years of age, arising in the cicatrix of a burn.

purulent, and offensive. The parts about the ulcer are congested and pigmented. The pigmentation is due to the fact that in the area of chronic con-

Fig. 83.—Same case as Fig. 82, after excision and skin-grafting by the Thiersch method.

gestion numbers of red blood-cells have been disintegrated. Such an ulcer is treated by making incisions to loosen the base and edges, so that contraction can take place. Venous congestion is corrected by means of position, the

use of compression, and in some cases the administration of cardiac stimulants. In all cases the surgeon employs stimulating applications to the ulcer in order to increase the supply of arterial blood. Scarlet red ointment (8 per cent.) strongly stimulates granulation.

The **callous ulcer of the leg** is the most chronic form of indolent ulcer and is sunken deeply below the level of the skin. Its border is hard and knobby. Its floor shows no granulations, and is either smooth and glistening or foul and liver colored. The discharge is thin and scanty, and the ulcer varies little in appearance from week to week or even from month to month. The treatment consists in scraping and cauterizing the ulcer; making radiating incisions through the margins and floor or elliptical incisions about the ulcer; applying antiseptic dressings and firm bandages. In some cases the ulcer should be strapped. The daily baking in the hot-air oven is often of great benefit. In severe cases it is necessary to extirpate the ulcer and apply skin-grafts.

**Hemorrhagic ulcers** bleed easily and profusely. Pressure must be applied; it is sometimes necessary to cut or burn away the granulations.

**Phagedenic Ulcers.**—The phagedenic ulcer results from the profound microbic infection of tissues debilitated by local or constitutional disease, and is commonly venereal. This ulcer has no granulations and is covered with sloughs; its edges are thin and undermined, and it spreads rapidly in all directions. Such an ulcer should be touched with strong caustics or Paquelin's cautery, and dressed with iodoform gauze and antiseptic fomentations. Tonics and stimulants should always be administered.

The **edematous ulcer** may result from inpediment to the venous return or, as De Nancrede points out, may be produced by the persistent use of poultices or wet dressings upon any ulcer.[1] It is most often met with in tuberculous processes and is occasionally seen in the leg when varicose veins exist. The granulations are large and pale, and are apt to bend over like unsupported vines. The discharge is profuse and seropurulent. The edges are softened and desquamating. An edematous ulcer requires dry dressings, stimulation, and compression.

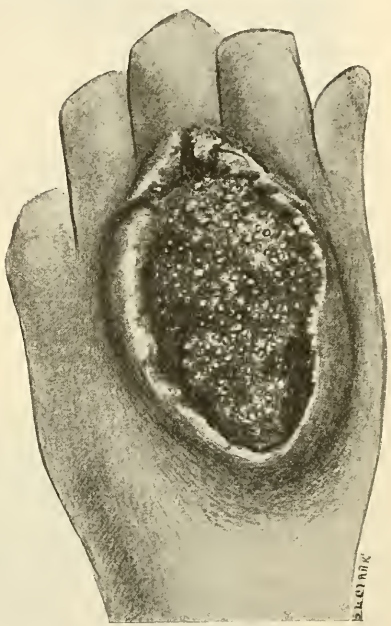


Fig. 84.—Marjolin's ulcer (epithelioma) from the scar of a burn.

A **rodent** or **Jacob's ulcer, noli me tangere,** or **cancroid ulcer,** is a superficial epithelioma developing usually from sebaceous glands, sweat-glands, or hair-follicles. It requires scraping and cauterization, the application of the *x*-rays, or, what is better, excision (see page 394).

**Marjolin's ulcer** (Figs. 82–86) is an epithelioma arising from a chronic ulcer or an old cicatrix. The malignant change begins at some point of the edge of the ulcer, and its first evidence is induration. The induration spreads slowly and comes to involve a considerable part of or even the entire ulcer. Marjolin's ulcer is the seat of scalding, darting pain; the discharge is profuse, ichorous, and foul, and the floor of the ulcer is uneven, warty, or cauliflower-

[1] "Principles of Surgery."

like. The anatomically related lymph-glands eventually become involved. This involvement is seldom early because induration has blocked lymph-channels. In order to confirm the diagnosis a bit of tissue should be removed, and the removed piece must include a portion of the edge of the ulcer and of some apparently sound tissue beyond it. If a microscopic examination

Fig. 85.—Epithelioma arising in the scar of a burn (Marjolin's ulcer).

shows epithelial infiltration of the apparently sound tissue, a diagnosis of malignant disease must be made. In an early stage of such an ulcer free extirpation and removal of the anatomically related glands may cure the patient. In a more advanced case, if an extremity is involved, amputate and clear out the related lymphatic area. In a very advanced case use the *x*-rays.

Fig. 86.—Marjolin's ulcer arising from a chronic ulcer of the leg.

Fig. 82 shows a Marjolin ulcer in a man twenty years of age. It arose in the cicatrix of a burn. I removed it and applied Thiersch grafts to the raw surface. Figs. 84, 85, and 86 show Marjolin's ulcers.

**Decubitus,** or **bed-sore,** is due to pressure upon an area of feeble circulation (see page 180). It is in most instances a condition of gangrene.

**Neuroparalytic** or **trophic ulcer** is due to impairment of the trophic nerve-fibers or of the trophic centers in the cord.

The **perforating ulcer,** as it was named by Vesigne, is believed to result from peripheral neuritis. It is certain, however, that in some of these cases there is arteriosclerosis, and it has been held that the vascular sclerosis is the real cause and that the nerve changes are secondary to the vascular changes. My own belief is that perforating ulcer is a condition dependent upon both arteriosclerosis and peripheral neuritis, traumatism usually being the exciting cause of the ulcer. It is met with most frequently in diabetics, but may be encountered in the victims of chronic alcoholism, injuries and diseases of the spinal cord, injuries and diseases of nerves, Bright's disease, and syphilis. I have seen this ulcer in an individual with a fractured spine, in several tabetics, and in not a few diabetics. The perforating ulcer commonly affects the plantar surface of the metatarsophalangeal joint or the pulp of the great toe or little toe about a callosity or corn. It may arise on the heel or the sole or the side of the foot. It is usually unilateral, but sometimes both feet are affected. In very rare cases more than one ulcer is present on the foot. Very rarely it affects the palm of the hand. The parts about the corn inflame, and pus forms and reaches into the bone. A sinus evacuates the pus by the side of the corn or callosity or the center of the callosity exhibits a blister containing seropus. A portion of the callous mass is cast off and a shallow ulcer is often exposed. This ulcer is small, has a punched-out appearance, and is surrounded by calloused margins. The ulcer penetrates deeply and after a time the bone is laid bare or the joint opened. The margins of the ulcer or sinus exhibit sprouting granulations and these are encircled by an area of markedly thickened epidermis. The discharge from a perforating ulcer is thin and scanty, and the ulcer, which slowly advances, is very chronic. It is not painful and is slightly, if at all, tender. The foot is cold and often edematous and the parts about the ulcer may be anesthetic. The ulcer may heal when the patient is kept in bed and open again when he gets out. The disease is far more common among males than among females and is most often met with in the fifth or sixth decades of life. As this ulcer may be present in anesthetic leprosy, in diabetes, peripheral neuritis, syphilis, in a paralyzed limb, and tabes dorsalis, and as the part on which it occurs is apt to be sweaty, cold, and more or less anesthetic, and as the sore may be hereditary, it is usually set down as trophic in origin. In treatment of a perforating ulcer I follow the plan suggested by Treves. This consists in putting the patient to bed and applying poultices to the sore. Every time a poultice is removed the raised epithelium around the ulcer is cut away and then the poultice is reapplied. In about two weeks an ulcer remains surrounded by healthy tissue. Treves treats this sore with glycerin made to a creamy consistency with salicylic acid, to each ounce of which mixture 10 min. of carbolic acid have been added. He directs the patient to wear during the rest of his life some form of bunion-plaster to keep off pressure. If in a perforating ulcer the bone is diseased, it must be removed. If the patient is diabetic he must be placed on antidiabetic diet and drugs. Nerve-stretching has been recommended as the proper treatment for perforating ulcer, but I have never tried it. No matter what treatment is employed, the sore is apt to reappear in the old situation or an adjacent region when the part is subjected to pressure. In order to prevent pressure upon the region of ulceration some advise the use of an artificial leg, the knee being kept bent. It may be necessary to amputate the toe or the foot.

The **scorbutic ulcer** is covered with a dark-brown crust, beneath which are pale and bleeding granulations. The parts adjacent are of a violet color.

Epitheliomatous, sarcomatous, tuberculous, and syphilitic ulcers and ulcers of the stomach and duodenum are considered under these respective diseases.

**Curling's Ulcer.**—This is an ulcer of the first portion of the duodenum which in very rare cases follows an extensive burn or scald of the cutaneous surface. Curling described this condition in 1841, but Sir Berkeley Moynihan points out in his book upon "Duodenal Ulcer" that Long, of Liverpool, described it in 1840. It is small, clean cut, and deep. It may be due to toxic material in the bile, the toxic material being due to the burn, but against this is the occurrence of the ulcer well above the opening of the bile-duct. Sir Berkeley Moynihan regards it as a toxic ulcer and points out that the ulcer practically never occurs unless there were septic changes in the burnt area. Septic emboli may be the cause. So far no case of Curling's ulcer seems to have been treated surgically. As Sir Berkeley Moynihan says, "there is no reason why it should not prove successful if the condition of the patient were not too exhausted by the extent or severity of the original injury." If perforation occurs the treatment is as for any other perforating duodenal ulcer.

A **fistula** is an abnormal communication between the surface and an internal part of the body, or between two natural cavities or canals. The first form is seen in a rectal fistula, a urethral fistula, or a biliary fistula; and the second form is seen in a vesicovaginal fistula. Fistulæ may result from congenital defect, as when there is failure in the closure of the branchial clefts, and can arise from sloughing, traumatism, and suppuration. Fistulæ are named from their situation and communications. For instance, a pleural fistula, an intestinal or fecal fistula, a rectal fistula, an anal fistula, a gastric fistula, a bronchial fistula, a vesical fistula, a biliary fistula, etc. Many fistulæ are tuberculous and lead to some deeply placed tuberculous focus. A fistula in communication with a viscus (for instance, the gall-bladder) may be maintained by an obstruction of the duct of that viscus; the removal of such an obstruction cures the fistula.

A **sinus** is a tortuous track opening usually upon a free surface and leading down into the cavity of an imperfectly healed abscess. A sinus may be an unhealed portion of a wound. Many sinuses are due to pus burrowing subcutaneously. A sinus fails to heal because of the presence of some irritant fluid, as saliva, urine, or bile; because of the existence of a foreign body, as dead bone, a bit of wood, a bullet, a septic ligature, etc.; or because of rigidity of the sinus walls, which rigidity will not permit collapse. Sinuses may be maintained by want of rest (muscular movements) and general ill health. The walls of a tuberculous sinus are lined with a material identical with the Volkmann's membrane of a cold abscess.

**Treatment.**—In treating a fistula or a sinus, remove any causative obstruction and any foreign body, lay the channel open, curet, brush with pure carbolic acid, and pack with iodoform gauze. Sometimes cure of a tuberculous sinus may be secured by repeated injections of iodoform emulsion or by injecting a paste of subnitrate of bismuth and vaselin (see page 894). The mixture remains in the sinus and serves as a framework for granulations. When a sinus closes after injections of bismuth paste, bismuth concretions sometimes form and lead to reopening after weeks or months. In obstinate cases of fistula or sinus entirely extirpate the fibrous walls, sew the deeper parts of the wound with buried catgut sutures, and approximate the skin surfaces with interrupted sutures of silkworm-gut. To stimulate a sinus to granulation it is sometimes necessary to touch it throughout with the actual cautery, nitric acid, pure carbolic acid, nitrate of silver fused on a metallic probe, or in a solution of a strength of 40 gr. to the ounce, or argyrol of a strength of 50 per cent. Fresh air is a necessity to the patient, and nutritious food and tonics must be ordered. There is some testimony, although scarcely as yet evidence, that the use of bacterial vaccines may at times be of value in the treatment of certain sinuses (see page 47).

## VIII. MORTIFICATION, GANGRENE, OR SPHACELUS

Mortification, or gangrene, is death in mass of a portion of the living body—the dead portions being large enough to be visible—in contrast to ulceration, or molecular death, in which the dead particles have been liquefied, cannot be seen, and are cast away. When all the tissues of a part are dead, the process is spoken of as *sphacelus*. Gangrene is, in reality, a form of necrosis, but clinically the term "necrosis" is restricted to molar death of bone or to death of parts below the surface *en masse*. In gangrene a portion of tissue dies because of anemia, and the dead portions may either desiccate or putrefy. Gangrene may be due to tissue injury, either chemical or mechanical, to heat or cold, to failure of the general health, to circulatory obstruction, to nerve disorder, the nerves involved being the vasomotor or possibly the trophic, or to microbic infection. A microbic poison can directly destroy tissues. It can indirectly destroy them by causing such inflammation that the products obstruct the circulation, but gangrene can occur when no bacteria are present. The essential cause of gangrene is that the tissues are cut off from a due supply of nourishment, and cell-nutrition is no longer possible. In other words, the essential cause of gangrene is the cutting off of arterial blood. De Nancrede says: "Indeed, except when the traumatism physically disintegrates tissues, as a stone is reduced to powder, heat or strong acids physically destroy structure, or cold suspends cellular nutrition so long that when this nutrition becomes a physical possibility vital metabolism cannot be resumed, gangrene always results from total deprivation of pabulum ("Principles of Surgery").

**Classification.**—Gangrene is divided into the following three great groups:

(1) **Dry gangrene,** which is due to circulatory interference, the arterial supply bing decreased or cut off. The tissues dry and mummify.

(2) **Moist gangrene,** which is due to interference not only with arterial ingress, but also with venous return or capillary circulation, the dead parts remaining moist.

(3) **Microbic gangrene,** arising from virulent bacteria. In this form the bacterial process *causes* the gangrene, and is not merely associated with it.

The above classification, if unqualified, suggests erroneous ideas. It indicates that there is an essential difference between dry gangrene and moist gangrene, which is not the case. If, when gangrene begins, the tissues are free from fluid, the patient develops dry gangrene; if they are full of fluid, he develops moist gangrene. If the arterial supply is gradually cut off, the tissues are sure to be free from fluid, and the gangrene will certainly be of the dry form. If arterial blood is suddenly cut off, the gangrene may be dry or moist, according as to whether the tissues are or are not drained of fluid. When gangrene results from inflammation, strangulation, and infection it is certain to be of the moist variety, because the tissues are sure to be filled with fluid.

De Nancrede says, in his very valuable work on the "Principles of Surgery": "Yet, let accidental inflammation have preceded the final blocking of an artery, or let ligation of the main artery cause gangrene because the collateral circulation cannot become developed, and if an aneurysmal sac is so situated as to interfere with a free return of venous blood and lymph, this anemic gangrene will in both instances prove moist and not dry."

There are many gangrenous processes which belong under one or other of the above heads, namely: *congenital* gangrene, a rare form existing at birth; *constitutional* gangrene, arising from a constitutional cause, as diabetes; *cutaneous* gangrene, which is limited to skin and subcutaneous tissue, as in phlegmonous erysipelas; *gaseous* or *emphysematous* gangrene, in which the

subcutaneous tissues are filled with putrefactive gases and crackle on pressure; *hospital* gangrene, which is defined by Foster as specific serpiginous necrosis, the tissues being pulpefied: some consider it a traumatic diphtheria; *cold* gangrene, a form in which the parts are entirely dead (sphacelus); *hot* gangrene, which is associated with inflammation, as shown by heat; *dermatitis gangrænosa infantum*, or the multiple cachectic gangrene of Simon; *idiopathic* gangrene, which has no ascertainable cause; *mixed*, which is partly dry and partly moist; *primary*, in which the death of the part is direct, as from a burn; *secondary*, which follows an acute inflammation; *multiple*, as gangrenous herpes zoster; *diabetic* or *glycemic gangrene*, which arises during the existence of diabetes; *gangrenous ecthyma*, a gangrenous condition of ecthyma ulcers; *pressure*, which is due to long compression; *purpuric* or *scorbutic*, which is due to scurvy; *Raynaud's* or *idiopathic symmetrical*, which is due to vascular blocking, perhaps from nerve disorder; *senile*, the dry gangrene of the aged; *venous* or *static*, which is due to obstruction of circulation as in a strangulated hernia; *trophic*, which is due to nutritive failure by reason of disorder of the trophic nerves or centers; *thrombotic*, which is due to thrombus; *embolic*, which is due to embolus; and *decubitus*, *decubital* gangrene, or bed-sores due to pressure.

**Dry gangrene** arises from deficiency of arterial blood. For this reason De Nancrede calls it anemic gangrene.

This form of gangrene is far more apt to result from the gradual than from the sudden cutting off of the supply of arterial blood, and is more common if the blood-vessels are atheromatous than if they are healthy; but even in a person with healthy arteries gangrene will ensue upon blocking of the main artery, if the collaterals fail to supply the part with blood. This form of gangrene can occur after laceration, ligation, or the lodgment of an embolus in the main artery of a limb; but in such accidents considerable fluid usually remains in the tissues and the gangrene is apt to be moist rather than dry.

**Gangrene Due to Embolism or Thrombosis.**—An embolus may cause gangrene in rare instances, hence the cause of embolism is responsible for the gangrene. There may be vascular disease. There may be or have been an infectious process (typhoid fever, pneumonia, influenza, diphtheria, etc.). If an embolus causes gangrene, it is probable that the blocking was not at once complete. Why an embolus in a young person causes gangrene is perplexing, because in such a person we can tie a large artery with comparatively little fear of gangrene. It must be that a clot forms proximally to the embolism, fills a considerable extent of the vessel, and cuts off the collaterals. When an embolus lodges in an artery and causes dry gangrene, the case runs the following course: sudden severe pain at the seat of impaction, and also tenderness; pulsation above, but not below, this point, after obstruction has become complete; the limb below the obstruction is blanched, cold, and anesthetic; within forty-eight hours, as a rule, the area of gangrene is widespread and clearly evident; the limb becomes reddish, greenish, blue, and then black; the skin becomes shriveled and its outer layer stony or like horn because of evaporation. The entire part may become dry; but usually there are spots where some fluid remains, and these spots are soft and moist, and the dead tissue, where it joins the living, is sure to be moist. The moist areas become foul and putrid, but the dry spots do not. In dry gangrene, at the point of contact of the dead and living tissues, inflammation arises in the latter structures, a bright-red line forms, exudation occurs, and ulceration takes place. This line of ulceration in the sound tissues is called the *line of demarcation*. It is Nature's effort at amputation, and in time may get rid of a large portion of a limb, and then heal as any other ulcer. A line of demarcation rarely causes hemorrhage, because it enters a vessel only after inflammation has caused occlusion by throm-

bosis. In dry gangrene from arterial obstruction there is gastro-intestinal derangement and also some fever. The gangrene does not extend up to the point of obstruction, but only to a region in which the anastomotic circulation is sufficiently active to permit of the formation of a line of demarcation. Below this point inflammatory stasis arises, but before this can go on to ulceration the parts die. In cases in which the arterial obstruction is sudden and complete the limb swells decidedly. This is due to the sudden loss of *vis a tergo* in the arterial system, venous reflux occurring and fluids transuding. In such a case the tissues contain fluid and putrefy, and the process, though due to the cutting off of the arterial circulation, is moist gangrene. Embolic gangrene attacks the leg more often than the arm. A thrombus in an artery rarely causes gangrene except in the aged, as the collateral circulation has time to adjust itself: but gangrene may follow thrombus formation, and when it does it comes on more slowly than does gangrene from embolus, and is certain to be of the dry form.

*Treatment of Thrombotic and Embolic Gangrene.*—When injury or blocking of a healthy artery causes us to fear the onset of gangrene, the patient should be placed in bed, the extremity should be elevated a little, kept wrapped in cotton-wool, and surrounded with bottles filled with warm water. It is useless when an artery is extensively diseased to incise it and remove a clot, because another clot will form at once. If a clot forms in a limited area of disease, it may be possible to excise a small section of the artery and do end-to-end anastomosis, and insert by implantation a section taken from a distant vein. In cases of thrombosis, however, the best operation is probably arteriovenous anastomosis (see page 463). In embolism it is a logical procedure to make a longitudinal incision in the artery, remove the embolus, and suture the wound in the vessel. So far arteriotomy for embolism has not been as successful as Carrel's experiments lead us to infer it should be; the probable reason is that most reported operations were upon elderly people, in whom, because of diseased arteries, extensive clots existed. Lejars believes that in the young results are sure to be better. Mosny and Dumont report a successful arteriotomy for embolism of the femoral artery in a case of mitral stenosis (quoted in "Lancet," March 9, 1912). If in spite of our efforts gangrene begins and progresses, wait for a line of demarcation, and while waiting dress the dying and dead parts antiseptically, wrap the extremity in cotton and keep it warm, and see to it that the patient gets plenty of sleep and nourishment. It is also advisable to give tonics and stimulants. When a line of demarcation forms, amputate well above it.

Fig. 87.—Gangrene from thrombo-angiitis obliterans.

**Presenile Spontaneous Gangrene.**—I have adopted Buerger's suggested name for this condition. The vascular disease responsible for the gangrene is in dispute. Winiwarter and others believe it is obliterative endarteritis of all the arteries of a leg. Buerger regards the condition as thrombo-angiitis obliterans, appearing in the larger arteries and also in the veins of the leg ("Amer. Jour. Med. Sciences," Oct., 1908).

It occurs among young adults (from twenty to forty) and is especially noticed among the Polish Jews in our large cities. It usually, but not always, begins in the left leg. It may start almost simultaneously in both legs. If it begins in one leg, the other tends to become affected sooner or later. It comes on with attacks of severe pain in the toes, foot, or leg, the extremity feels cold and looks bloodless, and no pulse can be detected in the dorsalis pedis and posterior tibial arteries. Such attacks are at first brought on by cold, later they appear to arise spontaneously.

When the foot is warmed some color returns and feeble pulse may be appreciated. Many of these patients get so that any attempt at walking causes violent pains in the calf muscles, pain so violent as to make the victim limp in torture or at once stop walking (*intermittent claudication*).

After a case has lasted a number of months *erythromelagia* may develop. In this condition when the foot is hung down the toes and dorsum of foot rather rapidly grow bright red.

After a case has lasted for many months it will be impossible at any time to detect a pulse in the dorsalis pedis or posterior tibial. The patient is wearing out with violent pain and cannot walk. Whenever the foot hangs down it becomes red or cyanotic. A bleb or ulcer may form upon the foot or great toe; it is sure to be intensely painful, and finally dry gangrene occurs.

I have seen 5 such cases of gangrene and in 3 of them I performed amputation. The examination of the vessels in the amputated legs accorded with Buerger's view that the condition is one of thrombo-angiitis obliterans, the veins often suffering as well as the arteries. Perivascular inflammation exists and in cases which have lasted for years there is arterial sclerosis, but cases seldom last for years without gangrene. The clot often fills the vessels of the toes, the dorsalis pedis, the plantar, and may extend well up both tibials toward the popliteal. Before gangrene arises various diagnoses are apt to be made (Raynaud's disease, intermittent claudication, erythromelagia).

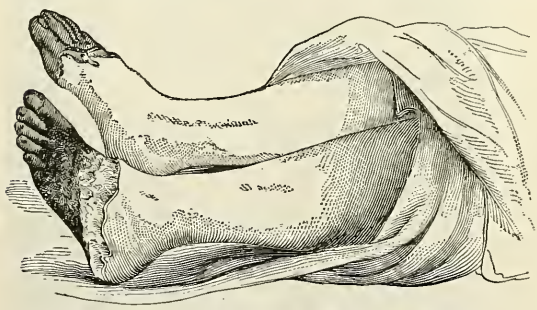


Fig. 88.—Senile gangrene of the feet (Gross).

The characteristic combination is violent pain, with absent pulse in the dorsalis pedis and posterior tibial arteries.

**Treatment.** — I have never seen the slightest lasting benefit result from medication. Sooner or later these cases come to amputation. In some cases violent pain compels amputation even before gangrene begins. Gangrene imperatively calls for it. Not unusually, weeks or months after amputation of one leg, the other leg has to be sacrificed.

The amputation should always be above the knee in order to be well above the thrombi in the vessels. Surgeons have used Moskowicz's test to determine where to amputate. (See Senile Gangrene, page 169.) Since Carrel's studies on reversal of circulation surgeons have attempted to prevent this and other forms of gangrene by arteriovenous anastomosis.

Early in a case, when we may assume that the veins are free from clot, the operation is clearly justifiable and may perhaps prove successful. Bloodgood has had success in a case which seems to have been of this type (Bernheim, in "Amer. Jour. Med. Sciences," Feb., 1912).

**Senile gangrene, chronic gangrene, Pott's gangrene** (Fig. 88), is a form

of gangrene due to feeble action of the heart plus obliterating endarteritis or atheroma of *peripheral* vessels. The vessels do not carry a normal amount of blood, and may at any time be occluded by thrombosis. In a drunkard or in a victim of syphilis or tubercle the changes supposed to characterize old age may appear while a man is young in years. It was long ago said, with truth, "a man is as old as his arteries." Senile gangrene most often occurs in a toe or the foot.

*Symptoms.*—A man whose vessels are in the state above indicated is generally in feeble health and has a fatty heart and an arcus senilis (a red or white line of fatty degeneration around the cornea). His toes and feet are cold and feel numb, and they "go to sleep" very easily, and he suffers from cramp of the legs and feet. He is dyspeptic and short of breath, and his urine is frequently albuminous. The arteries are felt as rigid tubes, like pipe-stems. He is in danger of edema of the lungs and of dry gangrene of the toes. A slight injury of a toe—for instance, cutting a corn too close—will produce extensive inflammatory stasis followed by thrombosis, which completely cuts off the blood-supply and causes gangrene of the part. Gangrene is usually announced by the appearance of a purple and anesthetic spot followed by a vesicle which ruptures and liberates a small amount of bloody serum and exposes a dry floor. In the parts about the gangrenous area there is often burning pain. The circulation in the tissues immediately adjacent to the dead spot is retarded or stagnated, the parts being purple and the color not disappearing or disappearing *slowly* under pressure. If the color fades under pressure it returns *slowly* when pressure is removed. The parts a little further removed are hyperemic, the color disappearing rapidly on pressure, and returning rapidly when pressure is removed. The dead parts do not putrefy at all or do so but slightly, hence the odor is never very offensive and is usually trivial. They are anesthetic, hard, leathery and wrinkled, and resemble a varnished anatomical specimen or the extremity of a mummy (hence the term *mummification*). Before the line of demarcation forms there is burning pain; after it forms pain is rarely present. If an embolus or thrombus in a diseased vessel of some size causes gangrene, the pain is severe at the point of impaction. In senile gangrene the distal portion of the dead area is always dry, the part nearer the body being generally somewhat moist. The process may be very limited or it may spread up along the dorsum of the foot and the leg, even to the knee. As it spreads the area of hyperemia advances at the margin, the area of stasis follows, and the zone of gangrene becomes more extensive. When tissues are reached, the blood-supply of which is sufficiently good to permit of inflammation going beyond the stage of stasis and to allow of stasis without extensive thrombosis, Nature tries to limit the gangrene by the formation of a *line of demarcation*. A line of demarcation may begin, but prove abortive, the tissue mortifying above it. This proves that tissue near the line is in a state of low vitality. The line of demarcation may

Fig. 89.—Dr. Keller's case of spontaneous amputation of a foot and part of a leg in a condition of senile gangrene.

prove durable and in some few cases spontaneous amputation takes place (Fig. 89). When a limited area is gangrenous, constitutional symptoms are trivial or absent; but when a large area is involved, the fever of septic absorption exists. Death may ensue from exhaustion caused by sleeplessness and pain, from septic absorption, from embolism of internal organs, or from some complication (renal, pulmonary, or cardiac). In many cases of senile gangrene clots are formed in the superficial femoral artery or its branches, an observation it is important to bear in mind when amputating.

*Prevention of Senile Gangrene in the Predisposed.*—Such a patient must avoid injuring the toes and feet. Cutting corns carelessly is highly dangerous, and any wound, however slight, requires rest and antiseptic dressing. The victim of general atheroma must wear woolen stockings, put a rubber bag containing warm water to his feet on cold nights, and attend to his general health. He must avoid overeating and is to be particularly moderate in the use of meats, should have a daily bowel movement, and should drink water in plenty between meals. He should avoid as far as may be work and worry, and enough sleep is imperative. A little whisky after each meal is indicated, and occasional courses of nitroglycerin are desirable. Courses of iodid of potassium, given in small doses, may retard the progress of the sclerosis.

*Treatment of Senile Gangrene.*—When gangrene occurs, if it is limited to one toe or a portion of several toes, if it is a first attack, if there is no fever or exhausting diarrhea, if there is no tendency to pulmonary congestion, if the appetite is fair and sleep refreshing, it is best to avoid radical interference and to await the formation of a line of demarcation. While awaiting the line of demarcation dress the part antiseptically, raise the foot several inches from the bed, and surround the part with bottles of moderately warm water. Very warm water may do harm. Give the patient nourishing diet, stimulants, and tonics; see to it that he sleeps, and during the spread of the gangrene watch for fever, diarrhea, pulmonary congestion, and kidney failure. When a line of demarcation forms, dress with warm antiseptic fomentations and iodoform, and every day pick away dead bits with the scissors and forceps. A tendon or ligament should be cut through and a protruding phalanx should be divided with a Gigli saw. If, after separation, an ulcer forms, skin-grafts may be applied. In many cases healing will occur; but even when the parts heal the patient will always be in deadly peril of another attack. If the gangrene shows a tendency to spread, if it involves more than a portion of several toes, if it is not a first attack, if there is sleeplessness, fever, exhausting diarrhea, anorexia, or a strong tendency to pulmonary congestion, do not delay, but at once amputate high up. If the gangrene shows no tendency to limit itself, or if the patient develops sepsis or exhaustion, at once amputate high up. The best point at which to amputate is above the knee, so that the deep femoral artery, which rarely becomes atheromatous, can nourish the flap and gangrene will not occur. It has been pointed out that the superficial femoral artery and its branches often contain a clot. Never amputate below the tubercle of the tibia. Some operators disarticulate at the knee-joint. Heidenhain affirms that so long as the gangrene is limited to one or two toes we should merely treat it antiseptically, elevate the limb, and wait for the dead part to be cast off spontaneously; if, however, it extends to the dorsum or sole of the foot, we should amputate at once above the knee. He further states that gangrene of the flaps almost always occurs in amputations below the knee, and high amputation is indicated in advancing gangrene with or without fever.[1] Personally, I still follow Heidenhain's rule. Many surgeons dissent from it and believe that in certain cases we can amputate lower down. Moskowicz suggested a method of determining the viable area. He applies an elastic bandage from

[1] "Deutsche medicinische Wochenschrift," 1891, p. 1087.

the toes to high up the thigh, puts in place the tourniquet band, and removes the bandage. In five or ten minutes he removes the band and notes the color of the limb as it is invaded by reactionary hyperemia. This wave of color travels toward the periphery. High up the extremity the reactionary blush appears quickly. The nearer the area of vascular obstruction, the slower the manifestation of color. In doubtful areas the blush comes slowly and imperfectly, and patches of white show here and there in the color. In a region of total ischemia no reactionary blush occurs.

Operation must be performed in the region where there was a complete red reaction, and never through an area where anemic patches were noted. This very ingenious test of Moskowicz has not seemed to me conclusive, and I have fancied in cases of threatened gangrene that it helped to bring about the very condition we feared. It is only just to say that in other hands the test seems to have proved successful. When amputation has been performed and the Esmarch band has been removed and no arterial bleeding takes place from the superficial femoral artery, a clot is lodged in that vessel. If such a condition exists, insert into the artery a fine rubber catheter or a filiform bougie and break up the clot. When blood flows we are sure that the clot has been washed out.[1]

Some surgeons have practised arteriovenous anastomosis between the femoral vein and common femoral artery in hope of establishing sufficient circulation to prevent impending or to cure existing gangrene. The results are as yet inconclusive, but some successes have been reported (see page 183).

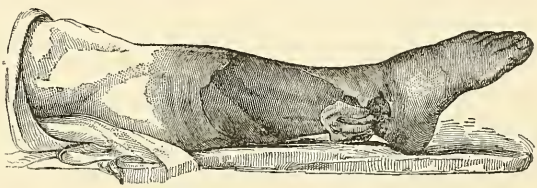


Fig. 90.—Acute gangrene (Gross).

**In moist or acute gangrene** (Fig. 90) the dead part remains moist and putrefies. As De Nancrede points out, there are two forms of moist gangrene: "that limited to the areas actually killed by a traumatism, with some surrounding tissue which dies," and "that which tends to spread widely, this latter being usually caused by specific micro-organisms, an intense, widespread, pyogenic inflammation resulting, involving the subcutaneous and intermuscular cellular planes, by strangulation of the vessels by which all blood-supply to the remaining soft parts is destroyed."[2] In a case of moist gangrene the parts remain moist, either because the main artery has become suddenly blocked, and the tissue fluids are not urged by sufficient *vis a tergo* to cause them to flow out of the limb, or because the main vein is blocked. It may arise in a limb after ligation, obstruction, or destruction of its main artery, main vein, or both; after long constriction, as by a tight bandage; after crushes and lacerated wounds, and after thrombosis of the vein. Moist gangrene may follow severe pyogenic infection or may be due to local constriction (strangulated hernia), crushing, chemical irritants, heat, and cold.

**Moist gangrene of a limb** may be seen typically in certain cases in which the main vein or artery or both vein and artery are constricted, damaged, or destroyed. The leg swells greatly and is pulseless below the obstruction; the skin, at first pale, cold, and anesthetic, becomes livid, mottled, purple, or greenish. A greenish color signifies putrefaction. Blebs are formed which contain a reddish or brown fluid. "These blebs, being caused by the accumulation of serum beneath epithelium which has lost its vital connection with the

[1] Severeano. See Mancozet's report before the Second Pan-American Medical Congress.
[2] De Nancrede's "Principles of Surgery."

derm, can be slipped around upon the surrounding true skin, the epithelium readily separating for long distances around, as in a cadaver" (De Nancrede). The extremity swells enormously, there may be pain at the seat of obstruction, but there is no pain in the gangrenous area, and sapremic symptoms quickly develop. The bullæ break and disclose the brown derm and sometimes the deeper structures, which are swollen and edematous. The fetor is horrible. Slight or moderate fever usually exists. In mild cases a line of demarcation soon forms. In severe cases in which virulent saprophytes are present the process spreads with great rapidity, neighboring glands enlarge, the temperature is much elevated, no line of demarcation forms, there is profound exhaustion, and gases of decomposition accumulate in and distend the tissues and cause crackling when the parts are pressed upon. Such severe cases are, in reality, examples of foudroyant or emphysematous gangrene.

**Moist gangrene from inflammation** is due to pressure of the exudate, cutting of the blood-supply, or to loss of blood-circulation because of microbic involvement of vessels and clotting of blood. It occurs typically in phlegmonous erysipelas. When an inflammation is about to terminate in gangrene all the signs of inflammation, local and constitutional, increase; swelling becomes very great and may be due partly to fluid and partly to gas. If gas is present pressure will cause crackling. The color becomes livid or purple. The anatomically related glands are enlarged and the symptoms of sapremia or suppurative fever exist. When gangrene is actually present the signs of inflammation have passed away, bullæ and emphysema are noted, with great swelling and all the other symptoms of molar death. The sudden cessation of pain is very suggestive of gangrene. The constitutional symptoms are those of suppurative fever and sapremia, or possibly of septic infection.

When a wound becomes gangrenous the surface looks like yellow or gray tow, the discharge becomes profuse and very fetid, and the parts about swell enormously and gradually become gangrenous.

*Treatment of Moist Gangrene.*—In extensive moist gangrene of a limb, if the condition is of the form described as mild, in which there are not severe symptoms of sepsis and in which the gangrene is not rapidly progressive, wait for a line of demarcation, and amputate clear of and above it. While waiting for the line to form, dress the dead parts antiseptically, wrap the extremity in cotton, apply warmth, and slightly elevate the limb. Give opium, tonics, nourishing food, and stimulants. In the severe form of moist gangrene (really foudroyant gangrene) amputate at once high above the gangrenous process. In inflammatory gangrene, such as is sometimes associated with phlegmonous erysipelas, relieve tension by incisions, cut away the dead parts, brush the raw surface with pure carbolic acid, dust with iodoform, and dress with hot antiseptic fomentations. Stimulate freely, administer nourishment at frequent intervals, and treat the patient in general as we would a case of sapremia or suppurative fever. A gangrenous wound is treated as pointed out in the section on Sloughing.

**Acute microbic gangrene, fulminating gangrene, emphysematous gangrene, gaseous phlegmon, gangrenous emphysema, gangrène foudroyante,** or **traumatic spreading gangrene,** results from a virulent infection of a wound. It was first described in 1853 by Masionneuve under the name of gangrène foudroyante. In 1864 Pirogoff called it *acute gangrenous edema.* The condition may be due to a mixed infection with virulent streptococci and organisms of putrefaction; or to infection with the bacilli of malignant edema, and putrefactive organisms. Some cases are due to the *bacillus of malignant edema* alone; some are due to the *Bacillus aërogenes capsulatus* of Welch and Flexner. These gas bacilli are found in soil, in animal and human feces, in street dirt, and the dust of floors. The injury is usually severe—often a crush which destroys

the main artery and renders an anastomotic circulation impossible, sometimes a compound fracture or a gunshot-wound. In such severe accidents the limb is much swollen and the pulse below the seat of injury is imperceptible, and the surgeon is often at this time uncertain whether to amputate at once or to wait. Emphysematous gangrene is commonest after compound fractures, and begins within forty-eight hours of the accident. The extremity becomes enormously swollen from edema and gas. The gangrene does not begin at the periphery, as does ordinary moist gangrene, but at the wound edges, which turn red, green, and finally black; the extremity soon undergoes a like change and becomes mortified. The skin peels off, emphysematous crackling, due to gas formed and retained in the tissues, can be detected over large areas, and the extremity becomes anesthetic and pulpy. The gases formed in the tissues are sulphid of hydrogen, sulphid of ammonium, volatile fatty acids, and ammonia. Great fetor is soon noted. The gangrene spreads up and down from the wound, and red lines, due to lymphangitis, run from above the wound. The adjacent lymph-glands swell. I have seen the gangrene involve an entire limb in thirty-six hours. No line of demarcation forms. The system is soon overwhelmed with ptomains, and the patient suffers from putrid intoxication, with delirium, and often passes into profound collapse with coma and subnormal temperature. Traumatic spreading gangrene must not be confused with erysipelas. In erysipelas the color is red, pressure instantly drives it out, and on the release of pressure it at once returns. In early gangrene the color is purple, pressure fails to drive it out at all or only does so very slowly, and if the surface is blanched by pressure, on the release of pressure the color crawls slowly back. Sometimes emphysematous gangrene, in the form of gangrenous cellulitis, follows a trivial injury such as a puncture, the entrance of a splinter, an abrasion, or a slight cut. The region about the injury becomes red, then livid, and finally green or black. Enormous swelling takes place, partly due to edema, partly to gas, and the swelling and discoloration spread rapidly. Red lines, subsequently becoming greenish, run toward enlarged lymphatic glands above the gangrenous part. The tissues are rapidly separated and destroyed and the bone is often quickly exposed and infected. The symptoms point to overwhelming sepsis. There is high fever and delirium, and coma and death are apt to ensue. The patient may die in from twenty-four to forty-eight hours. Welch estimates the mortality from gaseous phlegmon at almost 60 per cent.

*Treatment.*—In acute spreading gangrene of an extremity following a severe injury no delay is admissible. To wait for a line of demarcation is to expect the impossible, and a delay dooms the patient inevitably to death. Amputation must be performed at once high up, the flaps should be brushed with pure carbolic acid, and then every effort is to be made to sustain the patient's strength by the administration of food and stimulants. Antistreptococcic serum may possibly be useful. In cases of acute spreading gangrene following trivial injuries it may be possible to arrest the process by free incisions, thorough drainage, hot antiseptic fomentations, the continuous hot bath or continuous antiseptic irrigations, stimulants, etc., but in some cases amputation is necessary. Some surgeons, notably Doerfler ("Münchener medicinische Wochenschrift," April 23 and 30, 1901), oppose amputation in cases of spreading gangrene following trivial or moderately severe injury. Doerfler maintains that cases which recover after amputation would have recovered if amputation had not been performed. From this positive statement I am obliged to dissent. (See article on this form of gangrene by Blake and Labey, "Jour. Amer. Med. Assoc.," May 21, 1910.)

**Hospital gangrene** or **sloughing phagedena** is a disease that has practically disappeared from civilized communities. It formerly occurred in

crowded, ill-ventilated hospitals. Some consider it traumatic diphtheria. Koch thinks it is due to streptococci. Jonathan Hutchinson says: "Hospital gangrene is set up by admitting to the wards a case of syphilitic phagedena." It may show itself as a diphtheritic condition of a wound, as a process in which sloughs which look like masses of tow form, or as a phagedenic ulceration. The surrounding parts are inflamed and painful, and buboes form in adjacent lymphatic glands. The system passes into a low septic state.

*Treatment.*—In treating hospital gangrene ether should be given, the large sloughs removed with scissors and forceps, the parts dried with gauze and cauterized with bromin. The surgeon should take a tumblerful of water and into it pour the bromin, which will fall to the bottom of the glass. The drug can be drawn up with a syringe and injected into the depths of the wound. The wound should be plentifully sprinkled with iodoform and dressed with hot antiseptic fomentations. When the sloughs separate the sore can be treated as an ordinary ulcer. The constitutional treatment is that employed for sepsis.

**Special Forms of Gangrene.**—**Raynaud's disease** may be responsible for **symmetrical gangrene.** Raynaud's disease (Fig. 91) was first described

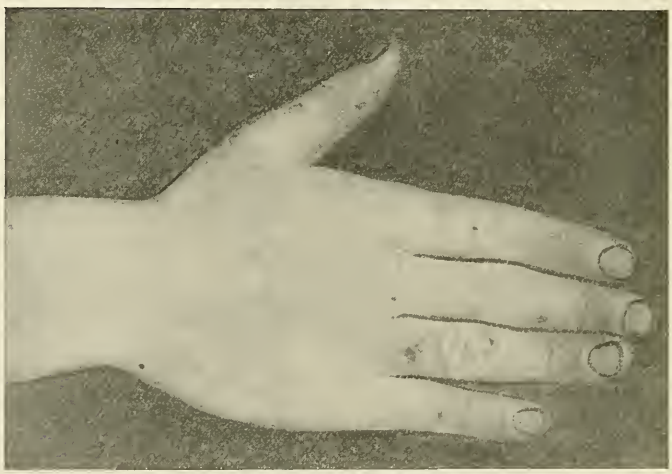


Fig. 91.—Raynaud's disease (Horwitz).

as a distinct malady by Maurice Raynaud in 1862. It is usually regarded as a vasomotor neurosis, is seen particularly in children and young female adults, but is sometimes met with in men. Chlorotic and hysterical women seem more apt than others to suffer from it. The condition is much commoner in winter than in summer, and cold seems to be an exciting cause. The well-known chilblain is an area of local asphyxia. The essential cause of Raynaud's disease is uncertain. In some acute cases associated with fever, albuminuria, and splenic enlargement it seems to be a part of an acute infectious disease. It can occur in a variety of toxic conditions and in a number of infectious diseases (typhoid fever, for instance). It may develop in the course of gout and also of diabetes. In many cases neuritis exists; in some there is either obliterative endarteritis or angiothrombosis of the larger peripheral vessels. Some cases seem to be purely hysterical. The fact that attacks of Raynaud's disease are sometimes accompanied by hemoglobinuria has suggested malaria as a possible cause. Raynaud's disease is characterized by attacks of cold, dead bloodlessness in the fingers or toes as a result of exposure to cold or of emotional excitement (*local syncope*). In the more severe cases

there are capillary congestion and mottled, livid swelling (*local asphyxia*). A case may begin with attacks of local syncope, and after a longer or shorter time get instead attacks of local asphyxia. Attacks may be occasional and far apart, may be frequent, or the condition of "dead" bloodlessness or asphyxia may be almost continuous. In some cases they only occur in winter; in others the slightest chill develops them. The patient complains of pain, tingling, numbness, coldness, and stiffness in the affected parts. In some few cases the skin of the face or trunk is attacked. Local syncope is thought to be due to vascular spasm, and local asphyxia to some contraction of the arterioles, with dilatation of the capillaries and venules. It is after local asphyxia that gangrene may appear. Attacks of Raynaud's disease occur again and again, and may never eventuate in gangrene.

Raynaud's disease is seldom fatal and is often recovered from.

Ever since Raynaud's day it has been generally maintained that the attacks are always symmetrical and that the blood-vessels are not diseased. We now know that many cases are successive but not symmetrical in the beginning, and that some never become symmetrical. This is especially true in the lower extremities. It has been shown of recent years that cases of thrombotic blocking of the larger arteries of the legs exhibit symptoms of Raynaud's disease before gangrene occurs, and that cases of Raynaud's disease of the feet and of erythromelalgia are apt to suffer from vascular thrombosis and gangrene from this cause.

Fig. 92.—Raynaud's gangrene. Patient has lost most of the terminal phalanges of the fingers and also the left leg. Right leg was amputated soon after this picture was taken. (Patient of Dr. T. E. Wannamaker, Jr., of Cheraw, S. C.)

**Raynaud's gangrene** (Fig. 92) is a dry gangrene and is most commonly met with upon the ends of the fingers or the toes, but it may attack the lobes of the ears, the tip of the nose, or the skin of the arms or the legs. Sometimes the disease is seen upon the trunk. The gangrene may be symmetrical from the beginning, may be successive, or may remain asymmetrical. Certain it is that many cases which we formerly regarded as clear cases of Raynaud's disease of the lower extremities develop gangrene, due either to obliterative endarteritis or angiothrombosis (see page 165). In such cases there is usually violent pain for weeks or months before gangrene begins. When gangrene is about to occur in Raynaud's disease the local asphyxia at that point deepens, anesthesia becomes complete, and the part blackens and feels cold to the touch. The epidermis may be raised into blebs at the margin of the gangrene, which blebs rupture and expose dry surfaces. A line of demarcation forms, and the necrosed area is removed as a slough. Widespread gangrene from pure Raynaud's disease is rare; there is not often an extensive area involved—rather a small superficial spot. Small areas are usually recovered from if not upon the lower extremity.

*Treatment of Raynaud's Disease and of Raynaud's Gangrene.*—If an individual suffers from attacks of Raynaud's disease, every effort should be made to improve the general health and to avoid chilling the surface of the body. During the attack employ gentle massage, place the extremity in warm water, and, if pain is severe, give morphin hypodermatically. Amyl nitrite is without value in this condition. When attacks of Raynaud's disease are so severe as to threaten gangrene, put the patient to bed; if the feet are attacked, elevate the legs slightly, wrap the affected extremities in cotton-wool, and apply warmth. If the hands are affected, wrap them in cotton-wool, elevate them slightly, and apply warmth. Massage is useful. Arteriovenous anastomosis has been employed for threatened gangrene, and several apparent successes have been reported (see page 166). When gangrene occurs, dress the part antiseptically until a line of demarcation forms, and then remove the dead parts by scissors, forceps, and antiseptic fomentations. If amputation becomes necessary, wait for a line of demarcation. In gangrene of a toe, toes, or foot due to angiothrombosis or obliterative endarteritis, amputation is always necessary. The terrible pain calls for it, and even if the gangrenous area is very small it is certain to spread or new areas are sure to arise. This form of gangrene has been treated by several surgeons by anastomosing the femoral vein to the femoral artery. Several times this operation seems to have proved successful. In an advanced case with thrombosed veins it cannot succeed (see page 166).

**Diabetic gangrene** often resembles strongly senile gangrene, but in many cases the dead portions remain somewhat moist and putrefy. Some surgeons attribute it directly to the sugar in the blood. Some think diabetes causes gangrene indirectly by rendering the tissues less resistant to infection and less capable than normally of repair. We know that sugar in blood removes fluid of the tissues and causes wound fluids to contain sugar. Hence wound fluids become excellent culture-media and body cells lose resisting power (see Morris, page 116). There is a great deficiency of oxygen in the tissues, which probably predisposes to infection (Lockwood, in "Lancet," February 10, 1912). The frequency in diabetes of boils, carbuncles, and spreading suppuration indicates lessened resistance to infection, and any infected area may become gangrenous. If phthisis exists or arises in a diabetic it develops with fearful speed toward a fatal issue. J. C. DaCosta, Jr., and Beardsley demonstrated that diabetic blood-serum shows a lowered opsonic index for tubercle bacilli, staphylococci, and streptococci. It is an interesting fact in this connection that the blood-serum of a pancreatectomized dog has a very low bactericidal power. Some hold that diabetic gangrene is of neurotic origin, being the result of nerve degeneration. Heidenhain believes that it is due to arterial sclerosis. That most of the victims of diabetic gangrene suffer from arteriosclerosis is certain. It seems probable that the gangrene is due to infection of tissue predisposed to infection by the presence of sugar and lessened amount of oxygen, and weakened by changes in the nerves and blood-vessels. Diabetic gangrene is most usually met with upon the feet and legs of elderly people, but it may arise at any age and may attack the genital organs, thigh, lung, buttock, eye, back, finger, or neck. It may affect only a single area, may attack several areas, or may be symmetrical. It may arise in any stage of diabetes, from the earliest to the latest. It may begin as a perforating ulcer. It is much more common in men than in women. There are clearly two forms of this condition. In one there is a slowly progressive, fairly dry gangrene, probably due chiefly to arterial sclerosis. In such a case a small, dry, dead patch sometimes lasts months before spreading begins. As in senile gangrene, a trivial injury is apt to be the exciting cause. In such a case the urine contains sugar and perhaps albumin, but seldom either acetone or diacetic acid. In the other form an injury, perhaps a trivial one, is

followed by a rapidly spreading cellulitis, which seldom forms pus and which eventuates in moist gangrene. In such a case the urine is apt to contain acetone and diacetic acid and there is grave danger of coma. When the gangrene follows a traumatism there are no prodromic symptoms. When it arises spontaneously in the skin, it is often preceded by pain of a neuralgic nature and attacks of "livid or violaceous discoloration of the skin, with lowered surface temperature and sometimes loss of sensation" (Elliot). In diabetic gangrene of an extremity the pain is often most violent, due probably to neuritis. In fact, neuritis may precede the gangrene. Diabetic gangrene is often superficial, but may become deep if it follows an injury or ulceration. Many of these patients are cyanosed (Lockwood, in "Lancet," February 10, 1912). A sufferer from diabetic gangrene is liable to cardiac failure, collapse, and coma.

**Diabetic coma** is an acid intoxication due to accumulation in the blood of acids which do not belong there, the chief of which is beta-oxybutyric acid. Acid gathers in the blood because of lack of proper oxidation. Diacetic acid is formed from beta-oxybutyric acid, and the breaking up of diacetic acid in the urine forms acetone. Hence, *acetonuria* means *acidosis*. Acetone is found in the urine when carbohydrates are not taken in sufficient quantity or are not assimilated by the tissues. Coma in acidosis commonly comes on rapidly, with vomiting, abdominal pain, weakness, restlessness, and drowsiness, which soon passes into coma. Sometimes it is ushered in by collapse, at other times by confusion of thought and speech. In coma the respiration is usually slow and deep, but may be sighing. The pulse is small, of low tension, and usually rather frequent. The temperature becomes subnormal, although during the onset it may be elevated. Cyanosis arises, the breath smells of acetone, the patient lies quietly in bed, and the pupils are dilated. In from twenty-four to forty-eight hours, as a rule, the patient dies.

Never fail to carefully examine the urine in every surgical case and especially in every case of gangrene, because diabetes may exist when least suspected. If albumin is present in the urine of a diabetic it means increased peril; if casts are found the prognosis is still worse. In such a case uremic coma may occur and be mistaken for diabetic coma.

*Treatment of Diabetic Gangrene of Foot.*—Most surgeons had grown very shy of amputating for diabetic gangrene until Küster, of Berlin, warmly advocated amputating above the knee without waiting for a line of demarcation. He showed that if amputation is performed below, the flaps will become gangrenous, and that after high amputation sugar may disappear from the urine. Of 11 amputations by Küster, 6 recovered and 5 died; and of these 5, 3 had albumin in the urine as well as sugar.

Heidenhain warmly advocated early high amputation, with the making of short flaps, and, in the United States, Powers, of Denver, defended the views of Küster ("Amer. Jour. of Med. Sciences," Nov. 11, 1892). I always amputate through the lower third of the thigh. Most writers now advocate high operation without waiting for line of demarcation if the gangrene is moist and due to bacteria attacking tissue weakened by diabetes. The same practitioners, if dry gangrene due to arterial changes exists, advocate awaiting the formation of a line of demarcation before amputating. I agree with Klemperer ("Therapie der Gegenwart," Jan., 1907) that we should reach a conclusion as to the proper course to pursue more from the character of the diabetes than from the nature of the gangrene. If neither diacetic acid nor acetone is in the urine, the glycosuria can be much improved and the general health vastly benefited by restricting the carbohydrates and administering codein or opium. In many cases sugar rapidly disappears. In such a case during the improvement of the glycosuria we await the formation of a line of demarcation, and while waiting

the gangrenous area and the parts immediately above are dressed with warm antiseptic fomentations. When a line of demarcation forms, spontaneous healing will probably occur. If it does not occur, amputate high up.

If acetone and diacetic acid persist in the urine, and if that excretion contains not only sugar, but also quantities of albumin, we cannot dare to wait for a line of demarcation because of the high probability that during the wait the patient will perish of septicemia (Klemperer, "Therapie der Gegenwart," Jan., 1907). In such a case perform an immediate high operation.

If the urine contains acetone and diacetic acid a line of demarcation will almost never form, and high amputation must be performed at once.

To put such a patient on a pure meat diet increases the risk of diabetic coma. In order to lessen the risk of coma give bicarbonate of sodium before and after operation. Twenty grains of this drug dissolved in Vichy water are given every two or three hours. If coma arises give intravenously 40 gr. of carbonate of sodium in 1 quart of water.

The worst cases are those in which the urine contains sugar, albumin, casts, acetone, and diacetic acid. In any case if over 1 gr. of ammonia is excreted in twenty-four hours, postpone operation until ammonia is reduced by diet.

In operating, ether and chloroform (especially chloroform) are to be avoided, because either is liable to induce acid intoxication. The operation may be performed under spinal anesthesia, although 1 case in which I amputated under spinal anesthesia died of coma. In a recent successful case I infiltrated all the nerves with cocain and amputated. There was no pain and apparently no shock.

My experience in diabetic gangrene of the leg comprises 6 cases. In 4 cases the urine contained sugar, acetone, and diacetic acid, and in 2 of these cases albumin was also present. In 2 cases sugar was in the urine, but no albumin, acetone, or diacetic acid. In each case a high amputation was performed—1 case (one of those with albumin) died of shock; 2 cases died in coma; 1 case died of sepsis; 2 cases recovered.

*Treatment of Diabetic Gangrene in General.*—In gangrene of some region other than an extremity, place the patient on antidiabetic treatment, treat locally with hot antiseptic fomentations, remove tissue which is completely dead with scissors and forceps, and sustain the strength.

**Gangrene from Ergotism.**—Ergotism is a diseased condition resulting from eating bread made from rye which has been attacked by a fungus (*Claviceps purpura*). In former days it was not unusual to have epidemics of ergotism from time to time, but at present the disease is found in individuals or, at most, in a few of a community. Ergotism is very rare in the United States, but it is not uncommon in southern Russia. It has occurred during the administration of ergot as a drug. Billroth reported such a case. It is never seen in unweaned children. The eating of bread made of diseased rye provokes gastro-enteritis, the evidences of which are abdominal pain of a crampy character, vomiting, diarrhea, and exhaustion. The patient complains of formication and itching of the skin of the extremities; severe, cramp-like, and tingling pains in the limbs, and disorders of vision. The pulse becomes small and slow. In some cases very painful spasms attack the muscles of the extremities and, finally, tonic spasm is noted, and the patient probably perishes from exhaustion after developing general convulsions and passing into coma. In other cases certain areas exhibit "gradual blood-stasis" (Osler), anesthesia, and, finally, gangrene. The gangrene is dry and peripheral. It usually affects the fingers or toes, but may involve an entire limb and may be symmetrical. Chronic ergotism is usually recovered from, but acute cases die in from seven to ten days.[1] The ingestion of ergot in quantity sufficient to produce chronic

[1] Pick, in Heath's "Surgical Dictionary."

poisoning causes tonic contraction of the peripheral blood-vessels, degeneration of the inner coats, and thrombosis of some arterioles. It is also maintained that degeneration of the posterior columns of the spinal cord takes place.

*Treatment.*—Ergotism is treated by forbidding the eating of the poisonous bread, allaying gastro-enteric inflammation, favoring elimination, and administering nourishment and stimulants. If gangrene is threatened, endeavor to prevent it by gentle massage and the application of warmth. If superficial gangrene occurs, dress with warm antiseptic fomentations and elevate the part, and every day take scissors and forceps and remove the loose crusts. If deeper and more extensive gangrene arises in an extremity wait for a line of demarcation and amputate above it.

**Gangrene from Frost-bite.**—Frost-bite is most common in the fingers, toes, nose, and ears, but the genital organs, the cheeks, the chin, the feet and legs, and the hands and arms may be attacked. Cold causes a primary contraction of the vessels and pallor and numbness of the part. After reaction the vessels dilate, the part reddens and swells, and a burning sensation or actual pain is experienced. In a trivial frost-bite the swelling and redness usually disappear after a few days, but in some cases the redness is permanent, and in many cases the redness, in the form of local asphyxia, returns under the influence of slight cold. (See Chilblains.)

In a more severe frost-bite the affected part becomes purple and covered with vesicles, and gangrene may or may not follow. When a part has been badly frozen it is whiter than normal, painless, anesthetic, and the peripheral portion dries. The part is deprived of all blood because of contraction of the vessels and because plasma coagulates at a few degrees above freezing. Cold disorganizes the blood, breaking up white corpuscles with the liberation of fibrin ferment. Coagulation of plasma and destruction of red corpuscles with the liberation of hemoglobin subsequently takes place. The thrombosis which is established prevents circulation, and the tissue-cells are damaged beyond repair. The part is bloodless and anesthetic, and a line of demarcation forms; hence we note that severe frost-bite causes dry gangrene. Areas of superficial gangrene are not uncommon. If a part which is not so badly frozen is brought suddenly into a warm atmosphere, hyperemia takes place when the blood runs into the frosted tissues, blebs form, and moist gangrene may result. Baron Larrey ("Surgical Memoirs"), in speaking of the retreat from Russia, tells a dreadful story of the suffering from cold. He says: "Persons were seen to fall dead at the fires of the bivouacs. Those who approached them sufficiently near to warm their frozen feet and hands were attacked by gangrene."

*Treatment of Frost-bite and of Gangrene from Frost-bite.*—A frost-bite in which the skin is livid and not as yet gangrenous should be treated by frictions with snow or rubbing with towels soaked in iced water. Larrey says that if frictions with snow or ice fail the part should "be plunged in cold water, in which it should be bathed until bubbles of air are seen to disengage themselves from the congealed part. This is the process adopted by the Russians for thawing a fish" ("Surgical Memoirs"). Whatever method is used, as the skin becomes warmer and congestion disappears the part should be subjected to dry friction and wrapped in cotton-wool. As previously stated, a sufferer from frost-bite should not suddenly be brought into a warm room. When gangrene follows frost-bite, if only small areas are involved, allow the dead parts to come away spontaneously, applying in the meanwhile hot antiseptic fomentations. If separation be delayed by cartilage, ligament, or bone, cut through the restraining structure. If amputation becomes necessary, await a line of demarcation, as it is not possible otherwise to be certain how high tissue damage extends, and to amputate through devitalized parts would mean renewed gangrene.

**Noma** is a rapidly spreading gangrenous process which is most apt to begin upon the mucous membrane of the gums or cheeks. Noma of this region is known as *cancrum oris*. Occasionally it begins in the ears, the genitals, or the rectum. When it attacks the vulva it is called *noma pudendi*. It may originate in the mouth and subsequently attack other regions. Noma is a very rare disease, is chiefly met with in children between the ages of three and ten, but it may attack older persons. O. Zusch[1] reports a case in a man sixty-six years of age. King reports a case in a woman of fifty-nine.[2] It occurs in girls oftener than in boys. The disease is most frequently encountered in children recovering from an acute disease. It is seen after scarlatina, typhoid, pneumonia, erysipelas, nephritis, dysentery, and especially after measles; in fact, Osler says that over one-half the cases follow measles. Children of tuberculous tendencies seem more liable than others. Young children who live amidst filth and squalor, in damp and ill-lighted apartments are most prone to

Fig. 93.—Noma. Seven days after first appearance of measles child showed gangrenous condition of mouth. Now, three days later, it involves both cheeks and under surface of upper and lower lips. Left cheek perforated. Two days before death a septic diarrhea developed which was uncontrollable (Crandon, Place, and Brown).

suffer, but that such conditions are not essential to the genesis of the disease is shown by the report of an epidemic of noma in the Albany Orphan Asylum. In this excellently situated, well-lighted, and well-ventilated building the children are carefully fed and cared for, and yet 16 cases of noma occurred after an epidemic of measles. (See "An Epidemic of Noma," by Geo. Blumer and Andrew MacFarlane, in "Amer. Jour. of Med. Sciences," Nov., 1901.) The disease is thought by many to be due to pus organisms. Lingard describes a bacillus which he considers causative. Blumer and MacFarlane conclude that the disease begins as a simple infection and a mixed infection takes place later. The mixed infection is not always due to the same organism, but is usually due to a long organism of a leptothrix type (Ibid.). Some

[1] "Münchener medicinische Wochenschrift," May 14, 1901.
[2] "Jour. Amer. Med. Assoc.," 1911, vol. lvi.

think that cases of noma are due to a variety of spirochetes, and it is an interesting observation that in noma the Wassermann serum reaction can be obtained.

In 1908 Crandon, Place, and Brown studied an outbreak of noma. Measles had been through the ward, 46 children had had gangrenous stomatitis, and 6 of them developed gangrene of the lip and cheek. A seventh case of gangrene appeared from another source ("Boston Med. and Surg. Jour.," April 15, 1909). They decided that the lesion was necrosis due to fusiform bacilli which invade living tissue, but rapidly die in necrotic tissue. Of these 7 cases, 2 recovered. The writers state that the duration of the disease is from four to ten days. They do not regard it as proved that noma is contagious and do not advise isolation.

*Symptoms.*—The disease begins as a sloughing ulcer; thrombosis and gangrene are soon observed. The edges of the ulcer are dark red and indurated. The gangrene usually spreads with very great rapidity, but in some cases it remains apparently stationary for days at a time. There is little or no pain. The odor is horrible. The disease is frightfully destructive, and if the mouth is involved is apt to destroy the cheeks, lips, eyelids, and large portions of the jaws. There is usually fever, but the temperature may be normal or even subnormal. The pulse is rapid, and exhaustion appears early and deepens rapidly. The mortality is large: Bruns says 70 per cent.; Rilliet and Barthez say 95 per cent. ("Amer. Jour. of Med. Sciences," Nov., 1901). Out of Nicoll's 11 cases, 9 died ("Progressive Med.," March, 1912). The cause of death is exhaustion, pyemia, or septic bronchopneumonia.

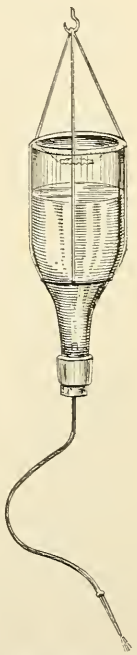


Fig. 94.—Improvised apparatus for the irrigation of a wound.

*Treatment.*—Administer an anesthetic and destroy the gangrenous area with the Paquelin cautery. In noma of the mouth chloroform is used instead of ether because the hot iron is to be applied in a region surrounded with anesthetic vapor, and ether vapor is inflammable. In noma in some other regions ether can be given. After cauterization directions are given to wash the part every few hours with peroxid of hydrogen, irrigate it with hot salt solution or boric acid solution, and dress it with compresses soaked in Labarraque's solution (Blumer and MacFarlane, in "Amer. Jour. of Med. Sciences," Nov., 1901). Nourishing food is given at frequent intervals, alcohol is administered, and strychnin is used to combat weakness. Rumple and Nicoll have each employed salvarsan and they believe with benefit. In one of Nicoll's successful cases two doses of 3 gr. each were given intravenously at an interval of three days. Each injection produced a great local reaction in the cheeks. If the surgeon succeeds in arresting the gangrene it will probably be necessary later to perform a plastic operation in order to replace loss of substance.

**Sloughing** is a septic process by which visible portions of dead tissue are separated. These visible portions are called "sloughs"; if they were large they would be called "gangrenous masses." A large septic slough is a gangrenous mass; a small gangrenous mass is a slough; there is no difference in the process, which corresponds to the formation of a line of demarcation.

**Treatment.**—Sloughing requires thorough and frequent irrigation by an antiseptic fluid, removal of the sloughs, and antiseptic treatment. An irrigator can be improvised from an ordinary bottle (Fig. 94). Warm antiseptic fomentations are applied until granulation is well advanced. In some cases

continuous irrigation with a hot antiseptic fluid is useful; in other cases continued immersion in a hot antiseptic solution is employed.

**Phagedena** is a process of ulceration and gangrene (most common in venereal sores) in which the surrounding tissues are rapidly eaten up, the sore becoming jagged and irregular, with a sloughy floor and thin edges. The discharge is thin and reddish, and the encircling tissues are deeply congested. This ulcer has no tendency to heal. Phagedena may attack wounds, but in this age is almost never seen except in venereal sores. When it does so the wound discharge is arrested, the parts about the wound become dark red and swollen, a black slough forms upon the wound, and the process spreads rapidly in all directions. The process when it attacks a wound is similar to or identical with a mild case of hospital gangrene, differing from the gangrene in the fact that in most cases a line of demarcation forms and the depression is not so great. Phagedena is probably due to mixed infection with pus organisms.

The **treatment of phagedena** consists in repeated touching with tincture of chlorid of iron and the local use of iodoform, the employment of continued irrigation or immersion in hot antiseptic fluids, or the application of the cautery, chemical or actual. After using the cautery the part is dressed with hot antiseptic fomentations. Whatever else is done, tonics, stimulants, and nutritious diet must be given and opium is often required.

**Decubitus, Decubital Gangrene, or Bed-sore.**—A bed-sore is the result of local failure of nutrition in a person whose tissues are in a state of low vitality from age, disease, or injury. The arterial condition of the aged favors the development of bed-sores. Such sores are due to pressure, aided it may be by some slight injury or by the irritation produced by urine, feces, sweat, crumbs or other foreign bodies in the bed, or by wrinkling of the sheets. The pressure destroys vascular tone, stasis results, thrombosis occurs, and gangrene follows. Sores occur over the heels, elbows, scapulæ, trochanters, sacrum, and nucha. In some cases after pressure is removed there are stasis, vesication, suppuration, and the formation of an ugly ulcer, surrounded by a zone of swelling and hyperemia. These ordinary pressure-sores arise like a *splint-sore* due to the pressure of a splint upon the tissues over a bony prominence. The pressure interferes with the blood-supply, the weakened tissues inflame, vesication occurs, sloughs form, and an ugly ulcer is exposed. When a bed-sore is about to form the skin becomes red and edematous. Pressure with the finger drives the color out rather slowly. The color becomes purple or black, a slough forms and separates, and a large, irregular, foul cavity is exposed. The discharge is profuse and offensive. The parts about are swollen and red. If the sore is not upon an anesthetic part, much suffering is produced by it. Bed-sores are most common in paralyzed parts; such parts are anesthetic, and injurious pressure is not painful and does not attract attention, and in such parts there is vasomotor paresis.

The **acute bed-sores of Charcot** are seen during certain diseases and after some injuries of the nervous system. These sores are usual over the sacrum in acute myelitis, and may appear in four or five days after the beginning of that disease or the infliction of an injury upon the spinal cord. The surgeon sees acute bed-sores upon the buttock of the paralyzed side after brain-injuries, and over the sacrum and other bony points after spinal injuries. Some believe these sores are due to vasomotor disorder; but others, notably Charcot, attribute them to disturbance of the trophic nerves or centers.

**Treatment of Bed-sores.**—The "ounce of prevention" is here invaluable. From time to time, if possible, alter the position of the patient, keep him clean, maintain the blood-distribution to the skin by frequent rubbing with alcohol and a towel, keep the sheet clean and smooth, and in some situations use a ring-shaped air-cushion to keep pressure from the part. When conges-

tion appears (*paratrimma*, or beginning sore), at once use an air-cushion or a water-bed and redouble the care to frequently change the position of the patient. Not only protect, but also harden, the skin. Wash the part twice daily and apply spirits of camphor or glycerol of tannin; or rub with salt and whisky (2 dr. to 1 pint); or apply a mixture of ½ oz. of powdered alum, 2 fl.oz. of tincture of camphor, and the whites of four eggs; or paint with corrosive sublimate and alcohol (2 gr. to 1 oz.); or apply tannate of lead or equal parts of oil of copaiba and castor oil; or paint upon the part a protective coat of flexible collodion.

When the skin seems on the verge of breaking, paint it with a solution of nitrate of silver (20 gr. to 1 oz.). When the skin breaks, a good plan of treatment is to touch once a day with a solution of silver nitrate (10 gr. to 1 oz.) and cover with zinc-ichthyol gelatin. We can wash the sores daily with 1:2000 corrosive sublimate solution, dust with iodoform, and cover with soap plaster, with lint spread with zinc ointment, or with dry aseptic gauze. When sloughs form, cut most of them off with scissors after cleansing the parts, slit up sinuses, and use antiseptic fomentations. In sloughing Dupuytren employed pieces of lint wet with lime-juice and dusted the sore with cinchona and charcoal. In obstinate cases use the continuous hot bath. When the sloughs separate, dress antiseptically or with equal parts of resin cerate and balsam of Peru. If healing is slow, touch occasionally with a solution of silver nitrate (10 gr. to 1 oz.). Bed-sores, being expressive of lowered vitality, demand that the patient shall be stimulated, shall be well nourished, and shall obtain sound sleep.

**Ludwig's Angina** (*Angina Ludovici*).—This disease, which was first described by Ludwig, of Stuttgart, in 1836, is an acute septic infection about the submaxillary salivary gland and in the cellular tissue beneath the mucous membrane of the floor of the mouth and of the upper portion of the neck. Ludwig called it "gangrenous induration of the neck" (D. Ludwig, "Med. Correspondez Blatt," p. 21, Feb., 1836, Stuttgart). The disease may arise in an apparently healthy man or during or after an infectious fever. It can arise at any age. The bacteria enter from the mouth by way of abrasions, wounds, ulcerations, or dental caries. It may be caused by delayed development of the third molar, necrosis of the tooth and alveolar process taking place and an abscess forming (G. G. Ross, "Annals of Surgery," June, 1901). In most cases the condition is a pure streptococcic infection or a streptococcic infection associated with infection by some other organism, for instance, staphylococci, pneumococci, or bacilli. In one of Davis's cases it was due to pneumococci alone; in another, to staphylococci alone (Gwillym W. Davis, "Annals of Surgery," August, 1906). In a case reported by Lockwood the bacillus of malignant edema was found. The condition is essentially an acute spreading cellulitis about the submaxillary gland. It usually begins about that gland, but in some cases seems to arise about the sublingual gland. It is usually a violent process from the start, but sometimes it is first an indolent swelling in the submaxillary region and becomes violent and begins to spread rapidly after a few days. It spreads along planes of connective tissue to the sublingual region of the mouth and to the pharynx.

The bacteria reach the submaxillary region in the lymph. A lymph-gland or perhaps several glands enlarge and are rapidly destroyed, the periglandular cellular tissue becomes involved, and after this spread takes place along connective-tissue planes rather than by lymph-paths, and other lymph-glands seldom enlarge.

The localization in the submaxillary region, the violence of the inflammation, the rapidity of the spreading, and the subsequent involvement of the pharynx and floor of the mouth are the characteristic features of Ludwig's angina (T. Turner Thomas, "Annals of Surgery," February and March,

1908). Thomas, in the article just referred to, proves by anatomical studies that the connective tissue in the submaxillary fossa is directly continuous with that in the floor of the mouth. The disease begins as a painful indurated swelling beneath the body of the jaw, the swelling rapidly increases in the neck, and may even pass to the level of the sternum. A board-like feel of this swelling is distinctive. The skin may be pale or dusky red. There may or may not be marked tenderness.

After a few hours or a day or two the floor of the mouth becomes involved; as a result of this the tongue is raised and pushed back, the mouth is kept from closing, swallowing becomes difficult, speech is impaired, the saliva dribbles constantly, and dyspnea becomes an alarming feature of the case.

In some cases the temperature is much elevated, but in most it is moderately or only slightly elevated.

If a spontaneous opening should occur it will be within the mouth. Free suppuration may occur or only a little watery pus may form and the pus may be brown and putrid. In many cases the cellular tissue becomes gangrenous, the gangrene resembling that of noma. The mortality is high. In Thomas's collection of 106 cases it is seen that 43 died (Ibid.), though with the prompt intervention which is now advised the mortality should be much below this figure at the present time. I have had 4 cases and 2 of them died.

Death may take place suddenly. Death is seldom due to septic intoxication or pyemia, but may be. It is usually due to edema of the glottis or to bronchopneumonia.

*Treatment.*—Operate promptly and incise freely. If there is an infective focus within the mouth, remove it. Make an incision through the swelling in the submaxillary region, carry the incision forward to the median line and divide the mylohyoid muscle. "The finger should be passed upward in the wound until only mucous membrane intervenes between it and the mouth. Gangrenous tissue is cut away, the wound is painted with pure carbolic acid, and dusted with iodoform" (T. Turner Thomas, Ibid.). Drainage-tubes are inserted and the part is dressed by antiseptic fomentations. If edema of the glottis exists, tracheotomy should be performed promptly. Stimulants are given with a free hand in Ludwig's angina.

**Carbolic Acid Gangrene.**—Dressings moistened with a solution of carbolic acid of a strength of from 3 to 5 per cent. if wrapped for a number of hours around a finger or toe, a hand or a foot, may cause dry gangrene. There is but little danger when such dressings are applied to the tissues of the trunk, because these thicker tissues are better nourished and cannot be completely surrounded by the wet dressings. When a dressing wet with a watery solution of carbolic acid is wrapped about the part the water evaporates, and as it does so the carbolic acid becomes more and more concentrated. A well mixed solution seldom causes gangrene. A recent aqueous solution often contains free globules of acid and is particularly apt to cause gangrene (Murphy). It is claimed that a solution of carbolic acid in glycerin never causes gangrene (Pautrier, in "Presse Médicale," March 2, 1907). Gangrene of a toe has occurred from the application of carbolized vaselin (Buckmaster's case, "Jour. Amer. Med. Assoc.," January 13, 1912). Two cases have been reported in which gangrene of a finger was caused by carbolized ointment (Brown, "Jour. Amer. Med. Assoc.," November 11, 1911; Schussler, "Jour. Amer. Med. Assoc.," August 19, 1911). In one case reported by DeWitt gangrene of the thumb resulted from injecting a ganglion of the thumb with equal parts of camphor and pure carbolic acid ("Southern Med. Jour.," June, 1909). The application of strong acid rarely causes gangrene, but Lévan found 14 reported cases in which it did (J. Lévan, in "Centralbl. f. Chir.," August 14, 1897), and Wallace has reported several more ("Brit. Med. Jour.," May 11,

1907). A solution as mild as 1 per cent. has caused gangrene. The continuous application of a solution of a strength of 3 per cent. or over is very dangerous and ought never to be practised. The author has seen 7 cases. Harrington saw 18 cases of gangrene in five years in the Massachusetts General Hospital, and collected 132 cases from literature ("Boston Med. and Surg. Jour.," May 2, 1901). Carbolic acid gangrene is due to great exudation into the cellular tissue, blocking the circulation (Housell), and the production of arterial thrombi, a condition to which the patient is predisposed by the injury and often by tight bandaging. The dressing is frequently applied by a druggist; it produces anesthesia of the part, and the dressing may not be removed for days, gangrene perhaps progressing beneath. In the author's 7 cases pain was absent and there was no smokiness of the urine or any other evidence of absorption of the drug. Dressing of lysol and alcohol may produce gangrene, but the necrosis is more superficial than that due to carbolic acid.

**Treatment.**—If the gangrene is very superficial, recovery may be obtained by using hot fomentations and picking the dead parts gradually away. In most cases the finger or toe is completely destroyed, a line of demarcation forms, and amputation is required.

**Postfebrile Gangrene.**—Dry or moist gangrene may follow any fever, but is most frequent after typhoid (may follow typhus, influenza, measles, diphtheria, scarlet fever, etc.). Keen tells us that the gangrene resulting from arterial obstruction is apt to be dry, and that from venous obstruction is usually moist. The same observer has collected 203 cases.[1] It is most usual in the lower extremities, but may appear in the upper extremities, cheeks, ears, nose, genitals, lungs, etc. Some writers have assigned as the cause weakness of cardiac action, but most observers believe an obstructing clot is the usual cause. This clot may come from the heart, but is in most cases secondary to endarteritis due to the action of the toxins of the bacilli of the specific fever. Keen shows that in some cases gangrene is due to obstruction of peripheral vessels and not of a main trunk. In rare cases gangrene arises after thrombophlebitis. Gangrene may begin as early as the fourteenth day of typhoid, but usually appears late in the disease and may arise far into convalescence. In the course of a continued fever frequent examinations should be made to see that gangrene is not arising. Particular examination from time to time should be made of the lower extremities and, in young girls, of the genitals. If gangrene arises in an extremity, apply antiseptic dressings, wait for a line of demarcation, and then amputate. If gangrene occurs in other regions, remove the dead tissue and employ hot antiseptic fomentations.

**Rules When to Amputate for Gangrene.**—In *dry* gangrene, due to obstruction of a non-diseased artery, wait for a line of demarcation. In *senile* gangrene, if it affect only one or two toes, let the dead parts be cast off spontaneously. If a greater area is involved or the process spreads, amputate above the knee without waiting for the line. In *ordinary moist* gangrene, if there are not severe symptoms of sepsis, and if the gangrene is not rapidly progressive, wait for a line of demarcation. In the severer cases amputate at once high up. In *traumatic spreading* gangrene amputate at once high up. In many cases of *diabetic* gangrene amputate at once high up (see page 175). In *ergot* gangrene, in *carbolic acid* gangrene, in *postfebrile* gangrene, in *Raynaud's* gangrene, and in *frost* gangrene wait for a line of demarcation.

**Arteriovenous Anastomosis for the Prevention or Treatment of Gangrene of an Extremity.**—This operation took origin from Carrel's famous demonstration of the reversal of the circulation in a dog's leg. The axillary artery has been anastomosed to the axillary vein; the femoral artery has

[1] Keen on the "Surgical Complications and Sequels of Typhoid Fever."

been anastomosed to the femoral vein. We know that by an anastomosis we can send arterial blood into a vein, but usually a clot soon forms at the junction. Those who advocate the operation assert that the force of the arterial blood-current overcomes the valves of the vein, and that arterial blood goes toward, even if it does not reach, the periphery. Further, that even if a clot does form, it forms slowly, circulation is arrested gradually, and collaterals distend during clotting.

The opponents of the operation assert that the valves cannot be overcome, that the blood flows off into venous branches and returns to the heart. Though it is doubtful how far toward the periphery the blood goes, we believe that the repeated impact of the arterial blood must finally overcome the valves; and in spite of great uncertainty as to the return of blood to the heart, we know that successful reversal seems to have been accomplished.

Bernheim ("Annals of Surgery," February, 1912) has collected 46 cases from literature and has added 6 operations, 2 of which were performed on the same individual (Halsted's case, Finney's case, Bloodgood's 4 cases). In the 52 operations gathered in Bernheim's table the ages of the subjects varied from twenty to eighty years. There were 13 deaths. He considers 15 of the cases successful, that is, "cases in which reversal, as far as one can judge, actually saved the limb from real or threatened gangrene." H. Morriston Davies recently reported a successful operation for gangrene. The artery and vein were completely divided and anastomosed in Hunter's canal ("Annals of Surgery," 1912).

The best plan is lateral anastomosis with proximal ligation of the vein, to prevent the deviated blood from returning at once to the heart. Bernheim and Stone devised this method (Ibid.) and Murphy advocates it. Only one-third of the artery is used for the anastomosis and some blood continues to flow in the artery toward the periphery. The artery carries blood to where the block is; the vein is supposed to carry blood much further. Bernheim points out that if a thrombus forms it will do so on the side of the artery and the limb will be no worse off.

In suitable cases the operation is justifiable and age is no bar. It is useless if veins are occluded as well as arteries, hence it is useless in advanced cases of gangrene from thrombo-angiitis obliterans. If any considerable area of gangrene exists veins are involved and the operation is useless.

## IX. THROMBOSIS AND EMBOLISM

**Thrombosis** is the antemortem coagulation of blood in the heart or in a vessel, the coagulum remaining at its point of origin and plugging up the vessel partially or completely. The process, and also the condition significant of the process, is known as thrombosis; the clot is called the *thrombus*. This process is an essential part in the arrest of hemorrhage; it occurs in phlebitis and arteritis, and affords a frequent basis for embolism. The thrombus is composed of red corpuscles, white corpuscles, fibrin, and platelets in varying proportions. Thrombi may form in the veins, in the arteries, in the capillaries, or in the heart. Clotting is due to destruction of white blood-cells, fibrin ferment being set free, causing the union of calcium and fibrinogen and thus forming fibrin. Thrombosis is more common in the veins than in the arteries, the slow blood-current and the existence of valves favoring the deposit, though not causing it. A thrombus forms gradually, being deposited layer by layer; hence it is stratified or laminated. Figure 95 shows a thrombus in a vein. All thrombi are either *infectious* or *simple*, the latter being also called *aseptic* or *bland*. Thrombi are also spoken of as fibrinous, red, hemostatic, leukocytic, etc.

**Causes of Thrombosis.**—In the formation of thrombi four conditions are to be considered, viz., chemical alterations in the blood, a bacterial attack on the intima, tissue changes in the inner coat of the vessel, and slowing of the circulation. One, several, or all of these conditions may exist in a case of thrombosis. In arteries the chief causes are disease of the coats and embolism. In veins the chief causes are injury and infectious phlebitis. Capillary thrombi may be due to propagation from veins or arteries or may form in the capillaries. The latter condition is seldom seen. The essential cause of all intravascular thrombi is damage to the endothelial coat and in most instances the damage is effected by bacteria, hence most cases of thrombosis seen by the surgeon are infectious. Any condition which causes the blood to contain an excess of fibrin-forming elements favors thrombosis, in the sense that a slight injury of the vascular endothelium will be followed by clot formation. Among conditions favoring thrombosis we must note particularly slowing of circulation, however caused. A special predisposing condition is the retarded circulation in tuberculosis, influenza, and fevers, the blood-clotting behind the vein-valves after the endothelium has been damaged by toxins. Among other inciting states are inflammations; wounds; fractures; the pressure of a bandage or of a splint; varicose veins; ligation of a vessel; injury of a vessel; foreign bodies in a vessel; atheroma in arteries; sutures in a vessel; pregnancy; certain diseases, such as gout, typhoid fever, and septic processes; phlebitis or arteritis arising in the vessel or from extension of surrounding inflammation, and the entrance of specific organisms.

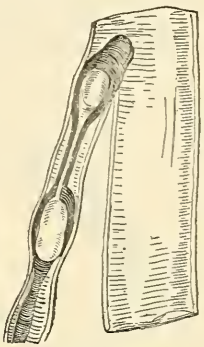


Fig. 95.—Thrombus in the saphenous vein. (Green).

It has been asserted that so long as the endothelium of a vessel is uninjured a clot does not form. Slowing of the blood-current in aseptic conditions, it is now taught, will not cause thrombosis. One of the functions of the endothelial coat is to keep the blood fluid by preventing corpuscular disintegration. A thrombus can form only when fibrin ferment is set free, and fibrin ferment can be set free only when white corpuscles disintegrate. When moving blood coagulates, the third corpuscles or platelets first settle out and form a nucleus and then the leukocytes gather about it. This is known as the *white* or *"ante-mortem" thrombus*—the clot of moving blood. Thrombi from moving blood are rarely pure white; they contain some red corpuscles, forming *mixed thrombi*. White thrombi and mixed thrombi are stratified and are at first soft, but harden as they age. The *red thrombus* plugs vessels which are cut across or ligated; it also occurs in septic processes and is the thrombus formed after death. A *primary thrombus* remains in the original region of thrombosis. A *secondary thrombus* forms about an embolism. A *propagating* or *spreading thrombus* extends a considerable distance from the seat of initial disturbance. A thrombus soon undergoes a change. An aseptic clot usually "organizes"—that is, the clot is absorbed and is replaced by fibrous tissue. The walls of the injured vessel become filled with leukocytes, leukocytes invade the clot, the vascular endothelium proliferates, and the young cells follow the colonies of leukocytes into the thrombus. The thrombus is gradually removed by leukocytes and replaced by fibroblasts, the new tissue is vascularized and becomes granulation tissue, the granulation tissue is converted into fibrous tissue, and the fibrous tissue contracts. In some instances a thrombus is implanted on the wall of the vessel and the tube is not permanently occluded. Such a condition may be obtained by the application of a lateral ligature about a small tear in a large vein. In most instances, after the formation of an intravascular thrombus, the vessel is

converted into a narrow cord of fibrous tissue. A thrombus may degenerate and break down (fatty degeneration), giving rise to emboli, or a thrombus may undergo calcification. A calcified thrombus in a vein is known as *phlebolith.* An infected thrombus may undergo liquefaction, infective emboli being set free (Fig. 96).

A clot may propagate in both directions, that is, toward the periphery and toward the heart. It was taught for many years that when an artery is ligated a thrombus quickly forms and reaches to the first collateral branch above. This view was formulated in pre-antiseptic days. It is now known that when aseptic ligation is performed the thrombus is small and rarely reaches the first collateral branch; and is often actually absent, vascular obliteration being obtained by proliferation of connective-tissue cells and of cells from the endothelial coat. If an infection takes place the clot may reach the first collateral branch. The old rule of surgery was as follows: If an artery is cut near a large branch, tie the branch as well as the artery, in order to permit of the formation of a lengthy clot. This rule is no longer followed unless infection exists or is anticipated.

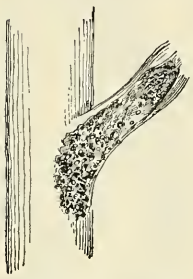


Fig. 96.—Infected thrombus of a vein (schematic).

A clot in a vein often extends a long distance. The author has seen in a postmortem examination a venous thrombus reaching from the ankle to the vena cava. A common example of thrombus in a vein is the clot formed in the uterine sinuses in a condition of puerperal sepsis, a clot which tends to extend into the iliac and femoral veins. In infectious thrombosis of the lateral sinus there is thrombophlebitis, and the clot tends to extend up to the torcular and into other sinuses and down into the jugular. *Phlegmasia alba dolens* or *milk-leg* is a condition in which the leg or the leg and thigh are swollen and painful because of venous thrombosis or sometimes lymphatic thrombosis (see page 187).

*Lymphatic thrombosis* occasionally occurs in the thoracic duct, axillary lymphatics, or inguinal lymphatics. It is most common in the uterine lymphatics during puerperal fever. Lymphatic thrombosis may be due to infection, to cancer, to tuberculosis, or to change in the lymph itself.

**General Symptoms.**—The symptoms are dependent on the seat of the obstruction and the presence or absence of infection. An organ or a part of an organ may exhibit functional aberration. The local signs in a vessel accessible to touch or sight are the presence of a clot; if it be in an artery, anemia and the absence of pulse below the clot; if it be a vein, swelling and edema below it. There is usually pain at the seat of trouble, and anesthesia below it. Moist gangrene may follow venous thrombosis, and dry gangrene, arterial thrombosis. Thrombosis of the mesenteric vein is followed by gangrene of the bowel. Infective thrombophlebitis is a spreading inflammation of a vein. A septic thrombus forms and the condition is an early step in pyemia. We see this condition sometimes in the lateral sinus of the brain as a result of suppuration in the middle ear; in any of the cerebral sinuses after infected compound fracture of the skull; and in the uterine veins during puerperal sepsis. Portal pyemia results from thrombophlebitis of branches of origin of the portal system (see page 1036). Thrombo-arteritis is a spreading inflammation of an artery in which a septic thrombus forms or in which a septic embolus lodges. It occasionally attacks an aneurysmal sac. In infectious thrombophlebitis and in arterial pyemia the symptoms are, of course, those of pyemia. A great danger of thrombosis is embolism, especially pulmonary embolism.

*Infective Thrombosis of the Lateral Sinus.*—(See page 805.)

*Thrombosis of the Jugular Vein.*—This condition is usually infectious and

secondary to infectious thrombosis of the lateral sinus or sometimes of the petrosal sinus. It is occasionally due to cancer, tuberculosis, acute rheumatism, or pyemia, taking origin from a distant focus. If it is infectious, the chills, the high and fluctuating temperature, and the great exhaustion proclaim the existence of pyemia. Locally the vein feels hard, the adjacent tissues are edematous, the branches of the jugular are visibly distended, there may be linear discoloration over the course of the jugular, and the head is held stiffly with an inclination to the diseased side.

*Thrombosis of the Mesenteric Vessels* (see page 988).—The arteries are affected much more commonly than the veins, and the superior mesenteric artery far more often than the inferior. Vascular disease is the cause of arterial thrombosis, and arterial thrombosis occurs chiefly in those beyond middle life. Venous thrombosis may be primary and has been observed after splenectomy, the clot having propagated to the mesenteric veins. It may occur as a result of any gastro-intestinal or general infection (pyemia, appendicitis, typhoid fever). Secondary venous thrombosis is due to portal obstruction or accompanies arterial mesenteric thrombosis.

Mesenteric thrombosis usually produces sooner or later gangrene of the gut, but does not always do so.

The period at which gangrene develops after blocking is uncertain; it may arise in thirty-six hours, it may not arise for two weeks or more. The gut becomes distended, bloody serum exudes into the peritoneal cavity, and in most cases into the lumen of the bowel. The mucous membrane undergoes necrosis and perforation occurs. The area involved varies greatly in different cases. In some cases it is very limited, and is rather apt to be in the large intestine. In other cases it is very extensive, and is apt to be in the small intestine. In a case of the author's in the Jefferson College Hospital practically the entire ileum was gangrenous and numerous perforations existed.

In mesenteric thrombosis pain arises rather suddenly and rapidly becomes severe. It is a persistent pain with paroxysmal exacerbations and is usually generalized, though in many cases it has an area of peculiar intensity. The pain is accompanied by rapid pulse, growing exhaustion, distention, subnormal temperature, tenderness, a mass appreciable by palpation in the region of the mesentery, free fluid in the peritoneal cavity, nausea, and vomiting. The condition suggests intestinal obstruction. The vomited matter consists first of the contents of the stomach, then of bile, finally becomes stercoraceous, and sometimes contains blood.

In nearly one-half of all cases blood in considerable quantity passes from the rectum.

Ballance points out that cardiac disease or arterial degeneration suggests the artery as the seat of thrombosis.

The only chance for recovery without operation is the establishment of the collateral circulation, and as the superior mesenteric vessels are terminal vessels this seldom occurs (in only about 5 per cent. of cases). (See Intestinal Obstruction from Mesenteric Thrombosis.)

*Iliac Thrombosis After Abdominal Operations.*—This complication is occasionally encountered and is most often met with in the left side, even when the operation was in the middle line or the right side. It is a rare complication, occurring, according to Professor Clark, 35 times in a series of 3000 operations.

Many explanations have been given of it. A great many surgeons regard it as infectious, but many cases certainly are not. Clark believes it is due to injury of the deep epigastric vein, forcible and prolonged separation of the wound edges by retractors being a common cause. The free anastomosis between the epigastric veins of the two sides accounts for the appearance of thrombosis

on one side after operation on the other. It is probable that in many slight cases the condition is not recognized, and it will not be recognized unless the clot reaches the femoral vein, and it requires one or two weeks to reach this vein if it does reach it at all. When a clot forms in the femoral vein a milk-leg develops. The entire extremity swells below the seat of thrombus, the temperature is usually normal, but may be distinctly elevated.

*Thrombosis in General Infections.*—In *typhoid fever* a thrombus may form in the heart, the veins, or the arteries. Thrombosis may occur in pneumonia, in influenza and in other fevers, and in tuberculosis. The vessels of a limb, a lung, the brain, or the mesenteric zone may suffer. The condition follows bacterial infection, the veins are most prone to suffer and gangrene may ensue.

*Thrombosis in Appendicitis.*—In about 2 per cent. of cases, according to Sonnenberg, this complication is noted. It may affect the femoral or saphenous vein of either side or of both sides, the portal vein or the vena cava, and may occur during an acute attack, but is more often noted in an interval.

It is not very unusual to find liver abscess follow appendicitis, the infection being carried by the portal vein and the condition being known as *septic pylephlebitis* (see page 1036).

**Treatment.**—If an aseptic thrombus forms in a large vessel of a limb, raise the limb a few inches from the bed, keep it perfectly quiet to avoid detachment of fragments (emboli), apply a bandage lightly from the toes up, and place bags of warm water about the extremity. Maintain rest for four or five weeks. The great danger is the formation of emboli, hence movements and rough handling are to be avoided. Gangrene is another danger, hence it is wise to favor venous return and the development of the collateral circulation by warmth, elevation, and bandaging. In infectious thrombophlebitis, if the vessel is accessible, tie it above and below the clot, open the vessel, remove the clot, irrigate, and pack the wound with iodoform gauze. The general treatment for a septic condition should be stimulating and supporting. Massage is unsafe in any condition of thrombosis, and is particularly dangerous in septic thrombosis. In thrombo-arteritis treat as in the thrombophlebitis. If gangrene of an extremity follows thrombosis, treat as previously directed (see page 164). Gangrene of the intestine in mesenteric thrombosis if not too extensive is treated by resection.

The treatment of infective thrombosis of the lateral sinus is set forth on page 806.

**Embolism** signifies vascular plugging by a foreign body (usually a blood-clot) which has been brought from a distance. The foreign body is called an *embolus*. An embolus usually consists of a separated or ruptured portion of a thrombus, atheromatous material from a diseased artery, or a bit of fibrin from a diseased heart-valve. In some cases an embolus consists of bacteria, of air, of fat, of a fragment of a tumor, or of parasites. In severe burns the blood undergoes changes and jelly-like matter is often precipitated and may cause embolism. Emboli vary in shape, in size, and in consistency. Emboli are divided into *simple*, *bland* or *aseptic* and *infectious*, *toxic* or *septic*. Emboli may arise either in the venous or in the arterial system, but are particularly prone to arise in the veins; they lodge in an artery, in capillaries, or in the veins of the liver. An embolus taking origin in one of the systemic veins passes through the right heart and lodges in a terminal branch of the pulmonary artery. If at this point it disintegrates, smaller emboli pass to the left heart and enter the arterial circulation to be deposited, as are emboli originating in the heart or arteries, in the arteries of an extermity, the kidneys, spleen, or brain. Emboli of the portal circulation lodge in the liver or perhaps pass through that organ and reach the lungs. An embolus is arrested when it reaches a vessel the diameter of which is less than its own. It is usually caught just above a bifur-

cation. When an embolus lodges, it at once partially or entirely obstructs the circulation and increases in size by thrombosis. Figure 97 shows an impacted embolus. A non-septic embolus when lodged usually "organizes," as does a thrombus, and, as described on page 185, is replaced ultimately by fibrous tissue. A soft embolus may disintegrate and permit the re-establishment of the circulation. An embolus may cause an aneurysm. A septic embolus breaks down, forms a metastatic abscess, and sends other emboli onward in the blood-stream.

An embolus is more serious than a thrombus: it causes sudden plugging, which makes dangerous anemia inevitable, and it will produce gangrene if the collateral circulation fails. Embolism of the mesenteric artery causes necrosis of the intestine. In organs with terminal arteries (spleen, kidney, brain, and lung) there is no collateral circulation and embolism causes *infarction*. For instance, if an embolus lodges in the lung it produces an area of ischemia; the removal of all propulsion upon the venous blood causes it to flow back and stagnate, and vascular elements exude and form a wedge-shaped area of red tissue, the embolus being the apex of the wedge. This is known as *hemorrhagic* or *red infarction*, and is often seen in the lung (Fig. 98). The

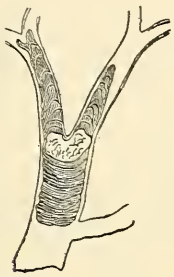


Fig. 97.—Embolus impacted at bifurcation of a branch of the pulmonary artery (Green).

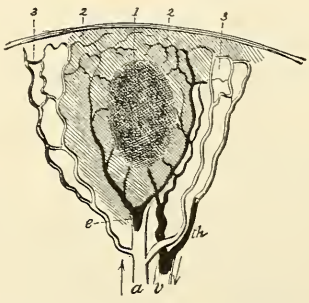



Fig. 98.—Diagram of a hemorrhagic infarct: *a*, Artery obliterated by an embolus (*e*); *v*, vein filled with a secondary thrombus (*th*); 1, center of infarct, which is becoming disintegrated; 2, area of extravasation; 3, area of collaterall hyperemia (O. Weber).

*white infarction*, seen in the brain and kidney, is not due to retrogression of venous blood, but is due to ischemia and resulting coagulation-necrosis. A septic embolus causes septic thrombosis and a septic infarction, and a septic infarction is followed by suppuration and the production of a pyemic abscess. That emboli of the systemic venous circulation usually lodge in the lungs explains the occurrence of pulmonary embolism after certain operations upon and during certain diseases of the regions drained by the systemic veins. Emboli formed in vessels of the systemic circulation lodge most often in the lungs, brain, kidney, or spleen. It is because emboli which pass into the portal vein lodge in the liver that operations upon the rectum may be followed by hepatic embolism and abscess of the liver.

**General Symptoms.**—The symptoms depend upon the organ involved and the presence or absence of infection. They are sudden in onset, and are due to loss of function, which may be permanent or which may be followed by inflammation, softening, or gangrene. In a septic embolus there are symptoms of infection and abscess forms at the seat of lodgment. In the course of pyemia a chill usually means the occurrence of embolism. Embolism of the cerebral arteries may cause aphasia, paralysis, or coma. Embolism of the pulmonary artery may cause almost instant death. Embolism of

a large artery of a limb produces symptoms similar to those of thrombus, except more sudden and decided. Below the obstruction the pulse is absent and the limb is swollen with edema, is cold, and is discolored. There is pain at the seat of obstruction. This condition is frequently followed by gangrene. Embolism of the superior mesenteric artery produces symptoms similar to those caused by acute intestinal obstruction, and results in gangrene of a portion of the intestine.

**Pulmonary Embolism.**—This condition occasionally follows operations and injuries and sometimes develops during certain diseases. I have seen a case after an operation for appendicitis, a case after an operation for varicocele, a case after an operation for hydrocele, a case after operation for perforated duodenal ulcer, a case after gastrostomy, and a case in a man with a large lumbar contusion to which massage was injudiciously applied. It is not very common. Albanus ("Beiträge klin. Chir.," xl) in 1140 abdominal operations found 23 cases. The emboli may be aseptic or septic. The condition is most common as a result of thrombosis of the veins of the lower extremities, appendicitis, and strangulated hernia. Certain postoperative pneumonias are embolic. Very small aseptic emboli may cause no symptoms or slight symptoms. When aseptic hemorrhagic infarction occurs there are symptoms. These symptoms are a chill or chilly sensations, moderate fever which may be transitory, dyspnea, rapid pulse, pain in the chest, sometimes rapidly advancing signs of consolidation, often a pleural friction sound, and bloody expectoration. Sometimes immediate death occurs. The mortality is always large (80 per cent.). I am satisfied that it is a much more common condition than we formerly supposed, and that some cases in which the emboli are very small are not diagnosticated and recover.

A septic embolism causes metastatic abscess and usually suppurating pleuritis, the condition being known as septic embolic pneumonia. Recovery is rare, but occasionally occurs. The symptoms are those of pyemia with the physical signs of consolidation and of pleuritis.

**Embolism of the Mesenteric Arteries.**—The superior mesenteric is the vessel usually affected. It may arise in pyemia, septicemia, arterial or cardiac disease. The symptoms are practically identical with thrombosis of the mesenteric vessels, except they arise suddenly. There is usually endocardial disease (see page 983).

**Treatment.**—Murphy removed an embolus of the femoral and iliac arteries through an incision of the femoral, and then sutured the incision ("Jour. Amer. Med. Assoc.," May 22, 1909). The operation was too late to prevent gangrene, but it emphasizes the truth that an aseptic embolism of a large artery can be treated by incision of the artery, removal of the clot, and suture of the vessel. The treatment long in vogue was as follows: In a limb, keep the part warm in order to stimulate the collateral circulation, elevate the extremity several inches from the bed, apply a bandage lightly from the periphery, and insist on perfect quiet. Massage is unsafe. If gangrene ensues, await a line of demarcation and, when it forms, amputate. In septic embolic arteritis in an accessible region it would be good surgery to act as in septic thrombophlebitis. After an operation upon veins (as the operation for varicocele, for varix of the leg, or for hemorrhoids), after any cutting operation, and after the infliction of a fracture, avoid as much as possible and for some time movements or handling, as fragments of thrombus may be detached.

In mesenteric embolism exploratory laparotomy may disclose a perforation which can be closed or a portion of gangrenous gut which can be resected.

In aseptic pulmonary embolism enforce absolute rest, give strychnin and morphin hypodermatically, and inhalations of oxygen. Trendelenburg has suggested operation for occluding pulmonary embolism (see page 900).

In septic embolic pneumonia pursue the conservative plan of treatment unless a large pulmonary abscess forms or an empyema arises. In either case operate to remove pus.

**Fat-embolism** in the human being was first noted by MacGibbon, of New Orleans, in 1856, and was first thoroughly described by von Recklinghausen in 1884, although Magendie, in 1827, and Virchow, in 1856, developed it experimentally in animals. It is a process which leads to an accumulation in the capillaries of liquid fat after injury to adipose tissue, high tension having forced the fat into the open mouths of veins. Fat may be forced into open veins by muscular action, by efforts at repair, or by concealed bleeding. Fat may get into the blood by means of the lymphatics and it can also enter by way of the synovial membrane. Wilms believes that fat reaches the veins by way of the lymphatics and the thoracic duct. Fat in the blood is quite a common condition, but seldom produces serious trouble, although it is occasionally fatal and is responsible for some otherwise inexplicable sudden deaths after fractures. Fat-embolism may arise during osteomyelitis, after extensive bruises, crushes, lacerations, amputations, fractures, resections, or rupture of the liver.[1] In a fatal case of mine it developed as a result of manipulation of a fracture of the neck of the femur. In another fatal case it followed amputation for cancer of the breast of a very fat woman. This fluid fat accumulates especially in the capillaries of the lungs and brain. It may plug systemic capillaries. If the patient recovers, he does so because the fat has been forced through the vessels; if he dies, the death results from mechanical hindrance to function and nutrition. When the emboli are widely scattered and not large, and when they do not lodge in vital parts of the brain or cord they may produce no symptoms and and do no real harm. Normal blood contains a small amount of finely emulsified fat (from 1 to 3 parts per 1000). In a number of physiological and pathological conditions the circulating blood contains considerable free fat. It may be found in a pregnant woman, a nursing baby, a fat individual, or in anyone during digestion. "It has been noted in the following conditions: chronic alcoholism; diabetes mellitus; certain diseases of the liver, heart, and pancreas; chronic nephritis; splenitis, tuberculosis; malarial fever, typhus fever; Asiatic cholera, and poisoning by phosphorus and by carbon monoxid. Lipemia commonly occurs as the result of lacerated wounds of the blood-vessels situated in fatty tissue, and after fractures of long bones involving injury of the fatty matter" ("Clinical Hematology," by John C. DaCosta, Jr.). In many cases of fracture in adults fat is found in the urine. I have had this demonstrated by repeated observations. When we recall how rarely simple fracture causes death it becomes evident that a moderate amount of fat in the blood is not dangerous or only becomes dangerous if it fails to flow out. In lipemia fatty embolism may occur if the amount of fat becomes excessive or if vascular damage favors plugging. At my suggestion, Dr. Wm. Carrington conducted an investigation to determine the frequency of lipuria after fractures (Essay Awarded the Surgical Prize in Jefferson Medical College in 1908). He determined that fracture

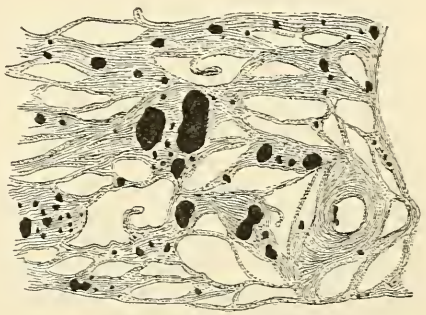


Fig. 99.—Fat-embolism of the lung after fracture of the femur. The fat-globules and masses, stained black with osmic acid, lie in the capillaries of the lung; × 150 (Hektoen).

[1] G. H. Makins, in "Heath's Dictionary."

of long bones invariably causes lipuria, fracture of small bones seldom does; that after fracture of a long bone fat appears in the urine "on different days, in different amounts, and in different forms" (this curious periodicity was first observed by Scriba in 1878); that when fat is present albumin is almost always present and blood is occasionally found; that the urea percentage falls as the fat content rises and rises as the fat content falls; that the condition is rare in young children and that fat-embolism, as a rule, is a benign process; that about the fifteenth day after a fracture fat usually disappears from the urine. Carrington, in 1908, found in literature 276 reported cases of fat-embolism.

**Symptoms** arise only when many emboli block a multitude of the capillaries of an organ, when a large embolism lodges, or when the capillaries of a vital region of the brain are blocked (the medullary centers). Emboli are most apt to form after or during handling of the wound or seat of injury or exhausting movement of the patient. Hence one peril of transportation after an accident, of early massage, of frequent changes of dressings, and of too early getting up after operation (Groendahl, "Deut. Zeitsch. f. Chir.," 1911, cxi). The symptoms are those of edema of the lungs and exhaustion, often with coma or delirium, and sometimes, in the beginning, are wrongly thought to be due to shock. There are restlessness, dyspnea, rapid and weakening pulse and rapid respiration, contracted pupils, and pallor followed by cyanosis. The temperature may be elevated, normal, or subnormal. Many coarse râles are heard in the chest, but percussion gives a clear note. If pulmonary edema becomes marked, the patient spits up a bloody froth. If life is prolonged a day or two, oil is found in the urine. Small amounts of oil may be found in the urine after serious injuries or operations when no symptoms of embolism exist. Nevertheless, the presence of the oil is always a cause of anxiety and is often a warning. It is maintained by Groubé that the amount of fat in the urine is in inverse ratio to the amount in the blood; the greater the amount excreted in the urine, the less the amount retained in the blood. Hence, fat in the urine makes the surgeon anxious, and a *sudden* diminution of the amount in the urine is a sign of grave danger if there develops increasing difficulty in respiration ("Rev. de Chir.," July, 1895). The inverse ratio said to be maintained between fat in the blood and fat in the urine, if it really exists, is similar to a finding of Lépine in diabetes, that is, if a diabetic is given diuretics, the sugar in the urine increases and the sugar in the blood decreases. The symptoms of fat-embolism seldom occur until at least twelve hours after an accident, and rarely before the third day, but may occur as early as three hours. The symptoms occur at a later period than those of shock and at an earlier period than those of ordinary embolism of the lung. The important point emphasized by Carrington is that after the reaction from shock, if there were shock, and for hours or days after the injury in any case, there is a period of freedom from all alarming symptoms, and that the symptoms of fat-embolism come on suddenly and without warning. If some of the oil is forced through the vessels of the lung, it will lodge in other regions and produce other symptoms. Oil may appear in the urine as above stated. Urinary suppression may occur. Delirium may arise, there may be twitching, convulsions or paralysis, or the patient may pass into coma. The eye-ground may show choked disk, hemorrhage, and fat in the vessels (Connel's case and Czerny's case). Cases of fat-embolism with severe symptoms are commonly fatal; milder cases are often recovered from. In mild cases the symptoms last but a few days, in severe cases the condition may prove fatal in from a few hours to seven days after the injury, and in from a few to forty-eight hours after the appearance of symptoms. A patient may have two or three attacks, Connel's patient had three attacks, there being an interval of a week between the first and second and between the second and third attacks.

**Treatment.**—Wilms, acting on his belief that fat enters the lymphatic duct and from there gets into the venous circulation, treated one case of fat-embolism successfully by making a fistula in the thoracic duct. This operation might be justifiable when dangerous brain symptoms are known to be due to fat-emboli. The usual treatment consists in absolute rest of the diseased or injured part and the administration of stimulants, such as strychnin, alcohol, and carbonate of ammonium, the use of external heat; the employment of oxygen by inhalation, and the administration of diuretics and of nitroglycerin hypodermatically. Artificial respiration may tide a patient over a crisis. If an external wound exists, free drainage must be established, and the diseased or damaged part should be thoroughly immobilized if possible. In order to prevent fat-embolism after a severe injury insist on rest. Massage used early after some injuries is dangerous, as it may force fluid fat into the vessels. Very early getting up after operation is not safe. Frequent changes of dressings are undesirable. When severe contusion causes the formation of a large cavity filled with blood, Groubé wisely advises incision to lessen the danger of fat-embolism.[1]

**Air-embolism.**—Air may enter a vein during a surgical operation or it may be injected accidentally while giving a hypodermatic injection, hypodermoclysis, or a saline infusion into a vein. It may follow irrigation of the pleura with hydrogen peroxid. In caisson disease it is taught by some that nitrogen is set free in the blood. It may occur when a cerebral sinus is opened, or in the uterine veins, if the uterus does not remain contracted after delivery. It is very seldom that any symptoms follow. It was long thought that such an accident must be always extremely dangerous. The experiments of my colleague, Professor Hare, indicate that quantities of air may be injected into the veins of a dog without apparent harm. The entry of a small amount of air into the veins of a human being will not be apt to induce dangerous symptoms, but it may be fatal. The more rapidly it is introduced and the greater the amount, the greater is the danger. The manner in which it can induce death is doubtful. Some maintain that it causes blood in the right side of the heart to froth, and thus prevents normal action of the valves, the heart becoming unable to propel blood through the lungs. Others maintain that air reaches the cerebral capillaries and so causes cerebral anemia. Some believe cardiac failure results from the presence of air in the pulmonary capillaries. The first view is the most probable. If a surgeon divides a large vein, air may be sucked in, and there is particular danger in such an accident if a vein at the root of the neck or a cerebral sinus is torn or incised, or if the damaged vessel lies in scar tissue and cannot collapse.

**Symptoms.**—When during an operation air enters a large vein there is a sucking sound, air-bubbles may be noted in the wound, and serious symptoms may or may not follow. Twice I have wounded the subclavian vein and have heard this sound, but no alarming symptoms developed. If serious symptoms are produced, they arise suddenly, and consist of extreme failure of circulation, a curious whirring or churning sound on cardiac systole audible even without a stethoscope, deadly pallor or cyanosis, gasping for air, convulsions, and possibly death.

**Treatment.**—Compress the vein with the finger and clamp it quickly. Suspend the anesthetic, lower the head, employ artificial respiration, give inhalations of oxygen, hypodermatic injections of strychnin, and intravenous infusion of normal salt and adrenalin.

[1] "Rev. de Chir.," July, 1895.

## X. SEPTICEMIA AND PYEMIA

**Septicemia,** or sepsis, is a febrile malady due to the introduction into the blood of pyogenic organisms or the products of saprophytic bacteria. There is no one special causative organism, and any microbe which produces inflammatory and febrile products may cause it. Either streptococci or staphylococci may be present. Pneumococci are a not very unusual cause. Septicemia arises by absorption of septic matter by the lymphatics. Clinically, we distinguish two forms of septicemia: (1) sapremia, septic or putrid intoxication, and (2) septic infection, true or progressive septicemia. In these conditions the area of infection is usually discovered by the surgeon; but when it cannot be located, the disease is called by the Germans cryptogenetic septicemia.

**Sapremia, Septic or Putrid Intoxication.**—This condition is due to the absorption of poisonous ptomains from a putrefying area. The bacteria do not enter the blood, but their toxins do, and as these toxins are active poisons the condition is comparable to poisoning by successive alkaloidal injections, the symptoms and prognosis depending upon the dose. Not unusually there is absorption not only of the toxins of saprophytic bacteria, but also of the toxins of pyogenic micro-organisms. Even if some of the bacteria enter the blood, they do not multiply in this fluid. Slight symptoms and recovery follow a small dose; grave symptoms and death follow a large one. The poison does not multiply in the blood, and a drop of the blood of a person laboring under putrid intoxication will not produce the disease when introduced into the blood of a well person; in other words, the disease is not infective. Considerable putrid material must be absorbed to cause sapremia. What is known as surgical fever is due to the absorption of a small amount of putrid or fermented wound fluid, and is, in reality, a mild form of sapremia. If sapremia arises, it does so soon after the infliction of a wound, and after a large rather than small wound when a considerable amount of wound fluid is pent up under pressure. It may follow labor where putrid fluid is retained in the womb, may follow an injury of or an operation upon a joint, may follow amputation where decomposing blood-clot or wound fluid is pent up within the flaps, or may ensue upon an abdominal operation or injury. In sapremia there always exist a considerable absorbing surface and a large amount of dead matter which has become putrid. Roswell Park[1] points out that sapremia arises from putrefaction of a blood-clot or wound fluids which are retained like foreign bodies in the tissues, and does not arise from putrefaction of the tissues themselves. He speaks of the condition as due to the absorption of poison from a "putrid suppository." Sapremia will not occur after granulations form. The term "putrefaction" is used because this is the usual change, but any fermentative organism may cause the disorder. Sapremia is a malignant form of surgical fever, and its existence means an ill-drained wound, and a fermenting and probably putrid collection of blood-clot or wound fluid.

In sapremia there is congestion of the stomach, intestines, and other abdominal viscera, particularly the kidneys, and also of the brain, and numbers of red blood-cells disintegrate.

*Symptoms.*—The patient often seems to react incompletely from the injury; he feels miserable, complains of headache, nausea, and pain in the back and limbs; or, he may react and in a day or two develop this condition of malaise. In some cases an aseptic fever is directly succeeded by sapremia. In most cases of sapremia, between twenty-four hours and two or three days

[1] "Treatise on Surgery by American Authors."

after labor, after an injury, or after an operation, there is usually a chill or a chilly sensation, though in some cases this is wanting. The temperature rapidly rises to 103° F. or even more. There are severe headache, dry and coated tongue, rapid and weak pulse, nausea and often vomiting, diarrhea, great prostration, restlessness, muscular twitching, and active delirium. The wound is found to be foul, and sometimes there is drying up of wound discharge. There is diminution or suppression of urine, and a strong tendency to congestion of various organs. Jaundice is not unusual. Petechial spots are frequently noticed upon the skin. They occur also upon mucous membranes and serous surfaces, and result from the plugging of small vessels with detritus of broken-down red corpuscles and consequent vascular rupture. Great elevation of temperature often precedes death. In some cases the dose of poison is so large that the patient passes into rapid collapse without preliminary fever. Some cases recover if the initial dose is not overwhelming and if additional doses are not absorbed. Many cases die of exhaustion. Some become linked with fatal pyemia or septicemia. Hemoglobin and red blood-corpuscles are rapidly and notably diminished. Distinct leukocytosis exists, except in those cases in which the organism is overwhelmed with the poison and is unable to react. Cover-glass preparations do not show organisms in the blood, and cultures from the blood are sterile.

*Treatment* consists in at once draining and asepticizing the putrid area and administering very large doses of alcohol and large medicinal doses of strychnin and digitalis. The patient should be purged and diaphoresis favored. The hot bath is valuable to cause sweating. The action of the kidneys must be maintained if possible. Purgatives, diuretics, and diaphoretics are given to aid in removing the toxin, and stimulants are used to sustain the strength of the patient during the elimination of the poison. Vomiting is allayed by champagne, cracked ice, calomel, cocain, or carbolic acid with bismuth. Food should be administered every three hours. The patient is fed on milk, milk and lime-water, liquid beef-peptonoids, beef-juice, and other concentrated foods. Quinin in stimulant doses is of value. Antipyretics are useless. The use of saline fluid by hypodermoclysis or intravenous infusion dilutes the poison and stimulates the heart, skin, and kidneys to activity. Visceral complications must be watched for and should be promptly treated if discovered. Among the possible visceral complications are nephritis, cholecystitis, enteritis, hepatitis, peritonitis, pleuritis, empyema, bronchopneumonia, pericarditis, and endocarditis. Antistreptococcic serum is useless in sapremia.

**Septic Infection, or True Septicemia.**—This condition is a true infective process. In sapremia the blood contains toxins of putrefactive bacteria, but not the bacteria themselves. In septic infection the blood contains both pyogenic toxins and multiplying pyogenic bacteria, the bacteria perhaps being free in the blood or in white cells. In sapremia the causative condition is putrid material lodged like a foreign body in the tissues. In septic infection the tissues themselves are suppurating, and both bacteria and toxins are being absorbed by the lymphatics. Of course, septic infection may be associated with septic intoxication or may follow it. In suppurative fever the tissues suppurate, but only the pyogenic toxins are absorbed, and not the pyogenic bacteria. In septic infection both the pyogenic bacteria and toxins enter the blood, and the bacteria multiply in the blood and produce continually increasing amounts of poison. The symptoms of sapremia depend on the dose. In septic infection only a small number of organisms may get into the blood, but they multiply enormously. The pus microbes cause true septicemia, and reach the blood chiefly through the lymphatics, but to some degree by penetrating the walls of vessels. A drop of blood from a man with septic infection will reproduce the disease when injected into the

blood of an animal; hence the disease is truly infective. The wound in such cases is often small, but may be large, and is commonly punctured or lacerated, and the disease begins later after the infliction of a wound than does sapremia. No wound may be discoverable, the infection having arisen from an unrecognized focus of suppuration—for instance, gonorrhea, middle-ear disease, dental caries, tonsillar suppuration, appendicitis, etc. The initial atrium of infection may or may not be discovered.

The bacteria which exist in the blood and organs in septicemia are usually staphylococci or streptococci, often both. Pneumococci or colon bacilli in some cases are causative. The blood is found to have lost much of its coagulating power; it remains fluid for some time after death, quantities of red corpuscles are destroyed, and minute hemorrhages take place in the brain, mucous membranes, skin, serous membranes, muscles, and various viscera. There may be inflammation of synovial and serous membranes. There is congestion of the gastro-intestinal tube and of the abdominal viscera. The lymph-glands are larger than normal and the spleen is notably enlarged. The wound contains numbers of bacteria.

*Symptoms.*—The type of this condition is met with in puerperal septicemia or in septicemia from an infected wound. When septicemia arises from an infected wound, red lines due to lymphangitis are usually seen about the wound, and there is enlargement of related lymphatic glands. In some cases, however, the wound and the parts about it look normal. A supposed aseptic fever after an injury may continue for an undue time and the surgeon may find that septicemia has developed. Septicemia may arise during the existence or after the abatement of sapremia, or may arise when the aseptic fever has passed away and when there has been no putrid intoxication. It begins in from four to seven days after labor or an injury, usually with a chill, which is followed by fever, at first moderate, but soon becoming high. In some cases there is a chilly sensation, but no distinct chill. There is always great prostration even before the chill. The fever presents morning remissions and evening exacerbations, and may occasionally show an intermission. When the remission begins there is a copious sweat. As the case progresses the temperature may fluctuate, and it often rises very high before death. The pulse is small, weak, very frequent, and compressible. The tongue is dry and brown, with a red tip. Sordes gather on the teeth and gums. Vomiting is frequent, and, as a rule, there is diarrhea. Low delirium alternates with stupor, and coma is usual before death. The great prostration is a noticeable and characteristic feature of the sufferer from septicemia. There are *subsultus tendinum* (twitching of the muscles of the hands and feet) and *carphologia* (picking at the bedclothing). Toward the end the face often becomes *Hippocratic* (hollow temples, pinched nose, sunken eyes, livid skin, lead-colored and cold ears, and relaxed lips). Visceral congestions occur. The spleen is enlarged, ecchymoses and petechiæ are noted, urinary secretion becomes scanty or is suppressed, and the wound becomes dry and brown. Blood examination detects a rapid and great diminution in red corpuscles and hemoglobin. The anemia is in many cases profound. There is marked leukocytosis except when the system is overwhelmed by the poison. Cover-glass preparations made from blood may show bacteria, but often fail to do so. Cultures from the blood are sterile in most cases, but not in all. A negative finding does not disprove the existence of septic infection; a positive finding is of conclusive diagnostic value. Pneumococcic septicemia is extremely violent in manifestation. In some cases death ensues before the lung has consolidated. If it is not so rapid, endocarditis, arthritis, peritonitis, meningitis, or osteomyelitis may develop.

The *prognosis* of true septicemia is very unfavorable, and in some malig-

nant cases death occurs within twenty-four hours, but mild cases often recover. Welch points out that finding the Staphylococcus pyogenes albus in the blood is not particularly ominous, but the presence of other pyogenic cocci is exceedingly threatening. Endocarditis, pericarditis, peritonitis, pleuritis, bronchopneumonia, empyema, nephritis, arthritis, cholecystitis, hepatitis, meningitis, and pyelitis are among the complications which may arise.

*Treatment* in general is the same as for septic intoxication. Antistreptococcic serum is employed by some surgeons, but the value of this method is as yet doubtful. It does not do any harm. It may do good. It is proper to use it, but not to the exclusion of other remedies. The usual dose is 10 c.c. injected into the abdominal wall. The injection may be repeated two, three, or even six times a day, and may be used for a number of days. Because of uncertainty as to the causative organisms polyvalent serum is used by some. Some use bacterial vaccines. Petre has injected fresh warm horse-serum to stimulate leukocytosis. Jayle ("La Presse médicale," 1905, p. 722) and Federman have also used it. All sera and vaccines are as yet of undetermined value in septic infection. Washing the blood by the intravenous infusion of salt solution often produces distinct improvement, which, unfortunately, is usually temporary. Dr. C. C. Barrows commends formalin used intravenously. The strength of the solution is 1 part of formalin to 5000 parts of salt solution. The dose is 500 c.c. I have had no experience with formalin in septicemia, but do not believe that any reagent can be safely introduced which would rapidly and directly kill the bacteria. Even if such an agent could be found, the attempt to use it would be dangerous, as dead bacteria liberate a poison, and the rapid death of immense numbers of bacteria would mean the entrance into the blood of an enormous amount of toxic matter.

**Pyemia** is a condition in which metastatic abscesses arise as a result of the existence of septic thrombophlebitis, the disease being characterized by fever of an intermittent type and by recurring chills. It is not actually due to free pus in the blood, but to the passage into the blood of the clots filled with toxins or, far oftener, of clots infected by streptococci, staphylococci, or both. After a wound is inflicted blood clots in the divided veins. If suppuration occurs, the clots may become filled with the toxins of pyogenic bacteria or be invaded by the bacteria themselves. Thus it becomes evident that pyemia may develop with septicemia. It may also develop when there is suppuration in a wound, but not septicemia, no lymphatic absorption of bacteria or toxins having occurred. A suppurating focus about a vein may cause thrombophlebitis and clot-formation even when no wound exists. This is seen in thrombophlebitis of the lateral sinus secondary to suppuration of the middle ear.

A vessel thrombus runs up in the lumen of a vein, and the apex of the clot softens, a portion of it is broken off by the blood-stream and carried as an embolus into the circulation. Many of these poisonous emboli enter into the blood and lodge in some vessels which are too small to transmit them, and at their points of lodgment form *embolic, secondary,* or *metastatic abscesses.* If the embolus contains only pyogenic toxins the danger is infinitely less than if it contains bacteria. The secondary abscess if caused by a clot containing only toxins may not lead to further dissemination of disease. If the embolus contains bacteria, thrombophlebitis occurs about it, and new infected emboli form and are sent throughout the system. Wounds of the superficial parts and bones produce pyemic infarctions and metastatic abscesses of the lungs. When these infarctions break into fragments particles may return to the heart and lodge, or may be sent out through the arterial system to form other foci in distant organs. Infected areas connected with the portal circulation (intestinal injuries, appendicitis, or suppurating piles) may produce *portal*

*pyemia* and multiple abscesses of the liver (see page 1036). Wounds of bones which open the medullary cavity or diploic structure are particularly apt to be followed by pyemia, and the disease may follow labor, phlegmonous erysipelas, and other conditions. Malignant endocarditis is called "*arterial pyemia*," and is due to endocardial embolic infection. In this disorder infected emboli lodge in the kidneys, the spleen, the alimentary tract, the brain, and the skin (Osler). Idiopathic pyemia is a misnomer. Some primary focus of infection must exist, as was pointed out when discussing septicemia.

**Symptoms.**—The wound often becomes dry and brown, and sometimes also offensive. A severe and prolonged chill or a succession of chills ushers in the disease; high fever follows, and drenching sweats occur. The chills recur every other day, every day, or oftener. A chill arises from the liberation and lodgment of emboli. During the sweat the temperature falls and may become nearly normal, normal, or actually subnormal. The temperature often oscillates violently. The general symptoms of vomiting, wasting, etc., resemble those of septicemia. In some cases the mind remains clear, in many the delirium is purely nocturnal. The skin frequently becomes jaundiced and a profound adynamic state is rapidly established. The blood changes are like those of septicemia. The spleen is enlarged. The lodgment of emboli produces symptoms whose nature depends upon the organ involved. Lodgment in the lungs causes shortness of breath and cough, with slight physical signs. Lodgment in the pleura or pericardium gives pronounced physical evidence. Lodgment in the spleen produces severe pain and great enlargement. The parotid gland not unusually suppurates.

In a suspected case of pyemia always examine an existing wound, and if there is no wound, remember that the infection may arise from gonorrhea, osteomyelitis, suppuration in the middle ear, appendicitis, dental caries, tonsillar suppuration, abscess of the prostate, etc. Chronic pyemia may last for months; acute pyemia may prove fatal in three days. The chief complications are joint-suppuration, bronchopneumonia, pleuritis, empyema, endocarditis, pericarditis, peritonitis, nephritis, cholecystitis, pyelitis, venous thrombosis, and abscesses.

**Treatment** is the same as for septicemia. Open, drain, and asepticize any wound and any accessible secondary abscess. The remarks made as to the use of sera and bacterial vaccines in septicemia apply also to pyemia.

**Erysipeloid** (*reticular lymphangitis, crab cellulitis*) was described by Rosenbach in 1887, although like cases were reported nearly fifteen years before by Morrant Baker under the name of erythema serpens. Gilchrist, in 1904, reported 329 Baltimore cases. I have seen a number of cases in the Jefferson Hospital. The condition is due to infection from handling putrid animal matter, especially fish; bites of crabs, and sticks of fish fins. Rosenbach claimed to find a special organism resembling but larger than a staphylococcus, but other observers fail to find it. The period of incubation is from a few hours to two days. Jopson describes the disease as follows ("Amer. Jour. Med. Sciences," May, 1908): "It appears as a swelling with elevated, sharply defined edges, which soon affects the entire circumference of the finger; and is commonly described as of a dark-red color, with purplish or even livid edges. The finger is tense and only moderately painful, but itching and burning are prominent symptoms. Sections of tissue excised show an inflammation of the entire corium and, to a slight extent, of the subcutaneous tissue, with infiltration of polynuclear leukocytes and small lymph-cells; edema of the epithelial cells of the epiderm, and inflammatory changes, especially marked around the sweat-glands and blood-vessels (Gilchrist). It has a characteristic tendency to spread from its usual point of origin, near the end of the finger, toward the palm, the primarily affected area fading from red to yellow, and thence to

normal. Reaching the palm, it may spread over it; but commonly, it soon affects the neighboring finger; and, when untreated, it may gradually spread to all the fingers and to the back of the hand. There is no fever or other constitutional disturbance, and the lymph-glands are almost never involved. It is more or less self-limited, commonly lasting from ten days to three weeks; and during this time there is a well-marked tendency to relapse. There is no suppuration, pustulation or vesiculation, and no scaling follows."

It is treated by applications of lead-water and laudanum, ichthyol or compresses soaked in a saturated solution of Epsom salt. Jopson applies tincture of iodin, and Gilchrist, 25 per cent. salicylic acid plaster.

## XI. ERYSIPELAS (ST. ANTHONY'S FIRE)

**Erysipelas** is an acute, contagious, spreading capillary lymphangitis due to the streptococci of erysipelas, which grow and multiply in the smaller lymph-channels of the skin and the subcutaneous cellular layers and also in the lymph-channels of serous and mucous membranes. Erysipelas, though contagious, is, as a matter of fact, seldom conveyed as such from one patient to another. Pantou and Adams ("Lancet," Oct. 9, 1909) present this truth convincingly. In St. Thomas's Hospital from 1896 to 1905 erysipelas cases were kept with other cases in the septic wards. In 1906 isolation was begun. The records of 1907 show that isolation had no effect in diminishing the number of cases of erysipelas arising in the septic wards. *Cutaneous erysipelas* is characterized by a rapidly spreading, acutely beginning dermatitis, by a remittent fever due to absorption of toxins, and by a tendency to recurrence. It is always preceded by a wound, a scratch, or an abrasion, which may have been trivial and may never have been noticed. The so-called idiopathic erysipelas is preceded by a breach of surface continuity so small as to escape notice. The initial point of infection may be in the mouth, the nostril, the pharynx, the auditory meatus, between the fingers or toes, at the margin of a nail, or in a cutaneous furrow. The involved area in cutaneous erysipelas seldom suppurates, but sometimes does, very thin, watery pus being formed. If thick pus forms it means mixed infection with staphylococci, but the formation of thin pus does not require a mixed infection, as the streptococcus is identical with the *Streptococcus pyogenes*. In some cases of erysipelas, staphylococcus infection follows and even actually replaces streptococcus infection. The rapid spread of erysipelas is due to the fact that the streptococci prevent coagulation of exudate and are not actively attacked by leukocytes. Erysipelas is most common in the spring and fall, and is most usually met with among those who are crowded into dark, dirty, and ill-ventilated quarters; it attacks by preference the debilitated and broken-down (as alcoholics and sufferers from Bright's disease). The disease may become endemic in special places or localities. The poison of erysipelas will produce puerperal fever in a lying-in woman. The streptococcus was first obtained in pure cultures by Fehleisen. This organism is widely diffused. The question of identity with the Streptococcus pyogenes is discussed on page 50.

*Forms of Erysipelas.—Ambulant, erratic, migratory,* or *wandering* erysipelas is a form which tends to spread widely over the body, leaving one part and going to another. *Bullous* erysipelas is attended by the formation of bullæ. In *diffused* erysipelas the borders of the inflammation gradually merge into healthy skin. *Erythematous* erysipelas involves the skin superficially. *Metastatic* erysipelas appears successively in various parts of the body. *Puerperal* erysipelas begins in the genitals of lying-in women, producing puerperal fever. *Erysipelas simplex* is the ordinary cutaneous form. *Erysipelas neo-*

*natorum* begins in the unhealed navel of a newborn child and spreads from this point. *Typhoid* erysipelas occurs with profound adynamia. *Universal* erysipelas involves the entire body. *Cellulitis* is often erysipelas of the subcutaneous layers. *Phlegmonous* erysipelas involves the skin and the cellular tissues, and causes suppuration, and often gangrene. *Edematous* erysipelas is a variety of phlegmonous erysipelas with enormous subcutaneous edema. *Lymphatic* erysipelas is characterized by rose-red lines due to lymphangitis. *Venous* erysipelas is marked by the dark color of venous congestion. *Mucous* erysipelas involves a mucous membrane. Erysipelas may attack the fauces, producing the very grave condition known as *faucial* erysipelas.

*Clinical Forms.*—The clinical forms are cutaneous erysipelas, cellulocutaneous or phlegmonous erysipelas; cellulitis, and mucous erysipelas.

**Cutaneous erysipelas** most frequently attacks the face. A fever suddenly appears, rises rapidly, reaches a considerable height, is remittent in type and sometimes distinctly fluctuating, and usually terminates in four or five days by crisis. At the time of febrile onset spots of redness appear on the skin. These spots run together, and soon a large extent of surface is found to be red and a little elevated. Any wound, ulcer, or abrasion which exists becomes dry and unhealthy, and its edges redden and swell. The erysipelatous area of redness and swelling extends either in spots with intervening healthy skin or in an uninterrupted line. The margin is usually sharply defined from the healthy skin, and the color fades at the original focus as the disease advances at the periphery of the red area. The border shows the most intense redness, the most marked inflammatory swelling (if there is swelling), and the greatest pain. The point of origin shows the least redness, the least swelling, and the least pain. Thus erysipelas reverses the rule of an ordinary inflammatory process. Milian calls this tendency of erysipelas "the law of centrifugal maximum" ("La Presse médicale," Nov. 5, 1910). The color fades at once on pressure and returns at once when pressure is removed. There is burning pain, which is most intense at the border and which is increased by pressure. In the hyperemic area vesicles or bullæ form, containing first serum and later it may be seropus, but there is rarely genuine suppuration in cutaneous erysipelas. Edema affects the subcutaneous tissues, producing great swelling in regions where there is much loose cellular tissue (as in the eyelids). Anatomically related lymphatic glands may become large and tender. In an ordinarily strong person the color of an erysipelatous area is bright red or, more rarely, dark red. A dusky color precedes suppuration. A blue color precedes gangrene or indicates profound cardiac or pulmonary involvement. Erysipelas spreads now in one direction, now in another, influenced, according to Pfleger, by the furrows of the skin. As facial erysipelas spreads it involves the ear. All subcutaneous inflammations stop short at the ear and cannot invade it because of the close adhesion of the skin to the cartilage. Milian calls this the "ear sign." When the disease ceases to spread, the swelling and redness gradually abate, and after they disappear desquamation takes place, and the blebs become dry and crusted.

In strong subjects the constitutional symptoms of cutaneous erysipelas are often slight. In the old and debilitated the symptoms are typhoidal, there is a dry tongue, dyspnea and hebetude, delirium comes on, and death is usual. Possible complications are meningitis, pneumonia, septicemia, pleuritis, pyemia, myocarditis, endocarditis, arthritis, and albuminuria. Erysipelas neonatorum is very fatal. The mortality in infants is certainly 50 per cent. (Sir Watson Cheyne's "Weightman Lecture for 1908"). In some instances an attack of erysipelas will cure an old skin eruption, a new growth, an ulcer, or an area of lupus. This is the *érysipèle salutaire* of our French confrères.

*Treatment.*—Isolate the patient. Asepticize the wound, if there be a wound. Examine to determine if there is diabetes or Bright's disease. Administer a purge. Cases of cutaneous erysipelas occurring in a fairly healthy, young or middle-aged subject, tend to get well without treatment. The late J. M. DaCosta advocated the administration of $\frac{1}{5}$ to $\frac{1}{3}$ gr. of pilocarpin. Debility absolutely contra-indicates this drug. If a person is debilitated, free stimulation is necessary. Tincture of chlorid of iron is usually administered in doses of from 20 to 40 min. well diluted, and given three or four times a day. Tonic doses of quinin are also given. Nutritious food is given at intervals of three or four hours. For sleeplessness or delirium use chloral or the bromids; for very high temperature cold sponging is required. Early in an attack, when the area is limited, the application of Bier's cup may do good. To prevent spreading some have advised injection of the healthy skin near the blush with a 2 per cent. carbolic solution or with fluid containing $\frac{1}{16}$ gr. of corrosive sublimate. A band of iodin painted on the skin may arrest the progress of the disease, and so may a ring streaked around a limb or about an erysipelatous area by lunar caustic. Kraske has suggested a method of preventing the spread of cutaneous erysipelas which is often effective. The patient is anesthetized. At about 2 inches from the margin of the redness a series of cuts are made into the skin, to a sufficient depth to cause free oozing. Each cut is crossed by another cut and a ring of scarifications is made to surround the region of the erysipelas. After the oozing ceases the scarified area is soaked for one hour with a solution of carbolic acid (1 : 20) or corrosive sublimate (1 : 2000). The part is dressed with pads wet with carbolic acid (1 : 40) or corrosive sublimate (1 : 2000). This operation causes the formation of a protective barrier of leukocytes. For a number of years I have used with satisfaction a treatment taught me by my old master, Prof. S. W. Gross. It is as follows: Paint the part and well around the part several times a day with a mixture of equal parts of tincture of iodin and alcohol. If a wound exists, keep it open and disinfect it with the iodin once a day. Cover the part with lint wet with lead-water and laudanum, and, if it be an extremity, bandage it from the toes or fingers to well above the erysipelatous area. The iodin is germicidal and quickly enters the lymph-spaces. The lead-water and laudanum allays the burning pain. Saturated solution of Epsom salt is a useful preparation. It is applied on gauze which is kept constantly moist. It quickly allays the burning pain and seems to limit the spread of the infection. Some advocate a daily inunction of Credé's soluble silver. A good application is a 50 per cent. ichthyol ointment with lanolin. A very useful method is von Nussbaum's. The author applies it somewhat modified, as follows: wash the part with ethereal soap, irrigate with a solution of corrosive sublimate (1 : 1000), dry with a sterile towel, apply an ointment of ichthyol and lanolin (50 per cent.), and dress with antiseptic gauze. Some use iced-water cloths. Hot fomentations are distinctly harmful. Some apply borated talc or salicylated starch. Ringer advised painting every three hours with a mixture composed of 30 gr. of tannic acid, 30 gr. of camphor, and 4 dr. of ether. Antistreptococcic serum has been used in erysipelas, and most beneficial results have been claimed for it. It is asserted that under its influence the temperature soon becomes normal. My personal experience with the serum treatment has not convinced me of its value, although some cases seem to be benefited. Schorer studied 100 cases of erysipelas in Bellevue Hospital and determined the opsonic index and its relation to treatment by inoculation of dead streptococci. He concluded that a vaccine does not prevent migration or recurrence, but seems to shorten the duration of the disease ("Amer. Jour. Med. Sciences," Nov., 1907). Ross and Johnson regard treatment by a specific vaccine as very efficient ("Jour. Amer. Med. Assoc.," March 6, 1909).

**Cellulocutaneous** or **phlegmonous erysipelas** is characterized by high temperature (104°–106° F.), the rapid onset of grave prostration, irregular chills, sweat, and a strong tendency to delirium. The constitutional condition may be one of suppurative fever, sapremia, septicemia, or pyemia. The parts are red, as in cutaneous erysipelas, and the tumefaction is vastly greater. The swelling is brawny, comes on early, increases with exceeding rapidity, induces a high degree of tension, and frequently becomes associated with sloughing or even cutaneous gangrene. The lymphatic glands are swollen, but the inflamed lymphatic vessels are hidden by the tumefaction. In most cases suppuration occurs, and when this happens the parts become boggy and the pus is widely disseminated in the subcutaneous and intramuscular tissues, and even into muscle-sheaths and tendon-sheaths (*purulent infiltration*). When the disease abates sloughs form, which leave ulcers upon being cast off. In bad cases muscles, vessels, tendons, and fascia may slough away. The commonest complications are suppression of urine, bronchopneumonia, congestion and edema of the lungs, meningitis, congestion of the kidneys, and acute pleurisy. Septicemia or pyemia may occur. We sometimes meet with this form of erysipelas after extravasation of urine. It is not a pure streptococcic infection. There is a mixed infection with other pyogenic cocci, and often with organisms of putrefaction.

*Treatment.*—At once asepticize and drain any existing wound, and dress such a wound with hot antiseptic fomentations. If there are inflamed lymph-vessels or glands above the area of cellulocutaneous infection, paint the skin above them with iodin and smear it with blue ointment or rub in Credé's ointment of soluble silver. Make numerous incisions into the inflamed tissues. These incisions should be near together, and each cut should be 2 or 3 inches long. Spray the wounds with hydrogen peroxid by means of a atomizer, wash with a solution of acetate of aluminum (2 per cent.), and pack each wound with iodoform gauze. Dress with many layers of gauze wet with a hot solution of acetate of aluminum (2 per cent.). The gauze is covered with a rubber-dam and a hot-water bag is laid upon the dressing. If sloughs form, cut them away and employ hot antiseptic fomentations. Change the dressings often. In some cases it may be necessary to employ continuous irrigation with warm antiseptic fluid, or continuous immersion in a hot aseptic or antiseptic bath. It is not unusually necessary to operate for the removal of enlarged lymphatic glands. The Bier treatment is a valuable addition to our resources. In rare cases amputation is demanded. When granulations begin to form, treat as a healing wound. The constitutional treatment is that previously set forth as applicable to septicemia, viz., purgation, the use of diuretics and diaphoretics, the administration of strychnin, quinin, digitalis, alcoholic stimulants, and nourishing food. In severe cases employ hypodermoclysis or saline infusion into a vein. Antistreptococcic serum may be employed.

**Cellulitis** (Fig. 100) is a microbic inflammation of the cellular tissue. It may be due to staphylococci, to streptococci, to other pyogenic bacteria, or to mixed infection with two varieties of pyogenic organisms. The commonest form is streptococcic infection, and this is a variety of erysipelas. A streptococcic infection may be followed and replaced by a staphylococcic infection. Infection with the *Bacillus aërogenes capsulatus* causes *gangrenous cellulitis*. Cellulitis is prone to arise in damaged tissues, for instance, in a crushed part, a limb the seat of a compound fracture, or tissue containing extravasated urine. In tissue the resistance of which has been lessened by diabetes, Bright's disease, irritating discharges, or trophic lesions, cellulitis is rather apt to develop. In cellulitis of the subcutaneous tissue the microorganisms find entrance by means of a wound. Swelling precedes redness.

The swelling is not so marked as in phlegmonous erysipelas, and the redness is darker and is less distinct than in cutaneous erysipelas. The redness of cellulitis is about the wound; it spreads, but does not fade at the center as does ordinary erysipelas; red lines due to lymphangitis ascend the limb from the infected wound, and the anatomically associated lymphatic glands enlarge. In the wound and its neighborhood there is severe throbbing pain. The constitutional symptoms of infection develop rapidly. In trivial cases the lymphatics dispose of the poison and suppuration does not occur. In severe cases pus forms about the wound and lymphatic glands may suppurate. In staphylococcic infection pus is thick, in pure streptococcic infection it is thin. Phlegmonous erysipelas may develop, and septicemia or pyemia may arise.

**Treatment.**—Open, disinfect, and drain the wound. Paint iodin upon the skin over inflamed lymphatic vessels and glands and cover with ichthyol ointment, or rub Credé's soluble silver ointment into the skin over the inflamed lymph-glands and vessels. Dress the wound and the adjacent inflamed area

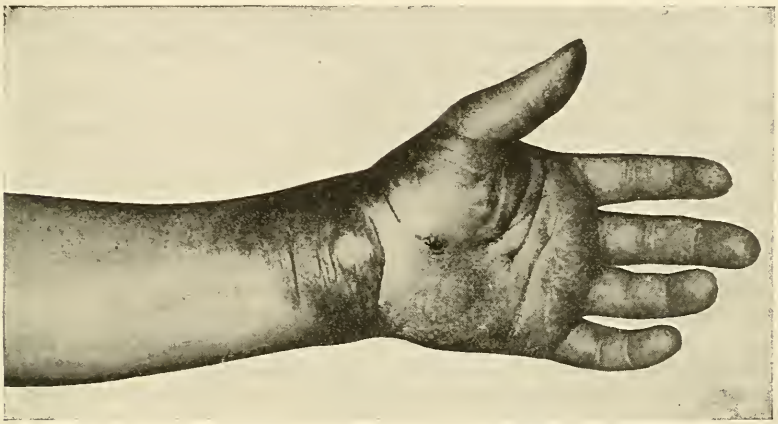


Fig. 100.—Acute cellulitis of palm and forearm following a slight wound.

with hot antiseptic fomentations. Secure rest of the part. It may be necessary to make incisions as in phlegmonous erysipelas. In some cases it is necessary to remove breaking-down glands. The constitutional treatment is that employed for septicemia.

## XII. TETANUS, OR LOCKJAW

Tetanus is a microbic disease invariably preceded by some injury and characterized by spasm of the voluntary muscles. The wound may have been severe, it may have been so slight as to have attracted no attention, it may have been inflicted upon the alimentary canal by a fish-bone or other foreign body, or may have been situated in the nose, urethra, rectum, vagina, or ear. It is possible that infection can occur through a mere abrasion of a mucous membrane. Sir David Semple has recently sought to demonstrate that tetanus does not of necessity depend on spores or bacilli introduced at the time of the injury. He believes that spores may be taken up from the intestine and deposited from the blood in the anaërobic area created by a wound or contusion ("Lancet," May 20, 1911, March 9, 1912). As yet this view lacks general acceptance. It has long been taught that so-called idiopathic tetanus is

either not tetanus at all, or the term expresses the fact that we have not found the traces of an injury which did exist. Sir David Semple maintains that spores may enter into trivial wounds and remain in the healed area for months, to possibly become active as a result of exposure to great heat or cold, to fatigue, or to bruising of the part. Semple's view would explain so-called idiopathic tetanus (see "Lancet," May 20, 1911). Tetanus arises most frequently after punctured and particularly after lacerated wounds of the hands or feet. In a surgical experience of over twenty-five years in connection with the Philadelphia Fire Department I have known hundreds of firemen to injure their feet by stepping on nails and not one developed tetanus. In fact, the only case of tetanus among them since 1871 arose in a man who lacerated his hand with glass. Before tetanus appears a wound is apt to suppurate or slough; but in some instances the wound is found soundly healed when the tetanus begins. The toy pistol produces a peculiarly dangerous wound. In the United States many cases of tetanus follow the celebration of the Fourth of July, a large percentage of the causative wounds being from the toy pistol. The Fourth of July, 1903, was responsible for 466 reported and no one knows for how many unreported cases in the United States. Since that date the prophylactic use of antitetanic serum has become the rule of practice in suspected injuries and there has been a notable diminution in the number of cases. In 1909 there were 150 cases; in 1910 there were 72 cases; in 1911 there were 18 cases; in 1912 there were only 7 cases ("Jour. Amer. Med. Assoc.," Sept. 7, 1912). Of the 7 cases, 6 died (86 per cent.). The fact that the bacillus of tetanus is anaërobic explains the comparative frequency with which punctured and lacerated wounds are attacked, for in such wounds the bacilli are deeply lodged in recesses or cavities into which air does not penetrate or are covered with discharges which exclude air. Suppuration favors the growth of tetanus bacilli because the pyogenic organisms consume oxygen. Occasionally, though fortunately very rarely, tetanus follows vaccination. It is essential that vaccine virus should be carefully selected and prepared. When care is taken, the operation is absolutely safe. When tetanus follows vaccination, it arises from infection of the wound either at the time of vaccination or, as is far more common, at a later period from scratching or some other fouling. The tetanus organism is not introduced in the vaccine, but obtains entrance during or subsequent to the operation of vaccination, because of utter neglect of the vaccine lesion and in consequence of the accumulation of filth upon and about it. In no reported case have the symptoms of tetanus appeared earlier than two weeks after vaccination (Wm N. Welch, "N. Y. Med. Jour.," Jan. 16, 1909). The organisms or its spores have never been discovered in tubes or in points, and, as Rosenau points out, the organism cannot grow and cannot form toxins on dry points or in glycerinated virus. The most scrupulous care is taken to prevent contamination of vaccine virus, and it is examined for tetanus toxin and tetanus bacilli before it is placed on the market. Tetanus has followed the injection of gelatin. Commercial gelatin often contains the bacilli and should never be used without careful fractional sterilization (see page 422). Roberts reports a case of fatal tetanus in a patient with chronic ulcer of the leg ("Lancet," vol. i, 1912). Evler infected himself while operating on a case of tetanus ("Berliner klin. Wochenschr.," Sept., 12, 1910). Tetanus has followed a burn, a frost-bite, a hypodermatic injection of quinin, child-birth, abortion, and the use in a wound of contaminated catgut. Most cases of postoperative tetanus depend upon infected catgut. The disease sometimes arises when no catgut is used (J. B. Smith's case, "Brit. Med. Jour.," May 6, 1911). Cases have arisen from such trivial operations as ligation of piles and hypodermatic injection of quinin. Peterson ("Jour. Am. Med. Assoc.," Jan. 8, 1910) collected 49 cases of postoperative tetanus reported since 1890. In 40 the peritoneal cavity had

been opened. In only 18 cases suture material was named (catgut, 3; catgut and kangaroo-tendon, 2; catgut with other material, 7; silk, 4; silk and silk-worm-gut, 1; silk and silver wire, 1). No case appeared before the fourth day, most on the ninth day. Drainage should tend to prevent it. Tetanus may appear within twenty-four hours after an accident, but it may not arise until many days or even several weeks have elapsed. Rose reported a case which began within twenty-four hours. Kuhn ("Berliner klinische Wochensch.," 1901) reports a fatal case of tetanus beginning twelve hours after an injection of gelatin. Such a rapid case could only be due to the gelatin having contained a large quantity of tetanus toxin (Schuckmann). Samuel D. Gross, in his "System of Surgery," speaks of 1 case occurring in a man five weeks after injury, and another in a girl four weeks after injury. Jacobson and Pease are of the opinion that "such cases as have been recorded with periods of incubation under three days must be accepted with considerable reserve" ("Annals of Surgery," Sept., 1906). Tetanus prevails more in certain localities than in others, but it is met with all over the world from the Arctic Zone to the Tropics, and may arise in either sex, in any race, and at any age. Colored people are very susceptible, and the disease may exist endemically, and does so in certain portions of New Jersey and of Cuba. In our country the greatest prevalence, according to Anders, is in Pennsylvania, northern New York, Long Island, Virginia, Georgia, and Louisiana. Anders collected 1201 cases and Pennsylvania stands first on his list with 224 cases ("Jour. Am. Med. Assoc.," July 29, 1905). It is stated that in certain districts of Nigeria the soil contains so many tetanus spores that the natives poison their arrows by sticking them into the ground (Allan C. Parsons, in "Brit. Med. Jour.," Jan. 23, 1909). Tetanus is due to the growth in a wound of a bacillus which was first described by Nicolaier and was first cultivated by Kitasato. It is the most widely distributed of all the pathogenic bacteria. It is very difficult to cultivate and cannot be cultivated at all unless air is absolutely excluded. Tetanus bacilli or their spores are found particularly in garden soil, in the dust of walls, walks and cellars, in street dirt, and in the refuse of stables. There is much suggestive evidence that virulent tetanus bacilli come from the intestinal canal of animals; that the bacteria lose their virulence when they have been long outside of the intestinal canal; and that the highest degree of virulence is obtained by those which have passed frequently through intestinal canals. The above view is known as the fecal theory and is strongly advocated by Somani.[1]

It is taught that in tetanus the bacilli do not enter into the blood, and toxic products produced by them are not directly absorbed by the blood or lymph. Porter and Richardson ("Boston Med. and Surg. Jour.," Dec. 23, 1909) in 2 cases obtained cultures of the bacilli from lymph-glands which received drainage from the wound region. This is a highly important observation. The toxic products alone without any bacteria enter the muscular end organs of motor nerves, ascend within the nerves, and reach the spinal cord and medulla (Brunner, Marie), become fixed in the nerve-cells of the spinal cord and medulla, and produce the symptoms of the disease. Metschnikoff found tetanus toxin in the cord. Emulsion made from the region of the masticatory nuclei of the floor of the fourth ventricle is peculiarly rich in highly potent toxin (Trosier and Georges Roux, quoted in "Lancet," Jan. 15, 1910). Tetanus is an intoxication and not an infection, and a drop of blood of an animal with tetanus, if injected into another animal, will not produce the disease. Tetanus toxin poisons the nervous system as would strychnin or some other vegetable alkaloid. It is probably the most powerful of known poisons. It has been estimated that $\frac{1}{278}$ gr. is sufficient to kill an adult weighing 165 lbs. ("American Medi-

[1] "Verhandl. d. X internat. med. Cong.," Berlin, 1890, Bd. v, Abth 15, p. 152.

cine," Nov. 30, 1901). The great power of the poison is shown by the report of Dr. Nicholas's case ("Comptes rendu de la Société de Biologie," 1893). Dr. Nicholas had been using a syringe to inject filtered cultures of the bacilli of tetanus and he accidentally pricked his finger with the needle. In four days tetanus began, and he barely escaped with his life in spite of the fact that the fluid was free of bacteria and the dose of toxin was extremely minute. The nature of the virulent poison which is produced at the seat of inoculation is uncertain. Some believe it to be alkaloidal, like the vegetable alkaloids; some, that it is a toxalbumin; others maintain that it is an enzyme or ferment (Nocard, Courmont, and others). In a very few instances the injection of perfectly sterile antidiphtheritic serum into human beings has caused death with all the symptoms of tetanus. The serum must have been obtained from horses in whom tetanus was incubating, and the blood-serum injected must have contained a fatal dose of tetanus toxin. In tetanus an ascending neuritis occasionally, though seldom, exists in the peripheral nerve near the lesion. The toxin is carried to the cord by the motor nerves only, and it is not only absorbed by the lymph-channels of the nerve, but ascends along the axis-cylinders of the nerve itself and reaches the motor cells of the spinal cord (Meyer and Ransom, in "Arch. exper. Path. u. Pharmakol.," 1903). On reaching the cord it attacks the motor nerve-cells, producing changes similar to those produced by certain infections, and ascends in the motor tracts of the cord to the medullary nerve-centers. While toxin is ascending the axis-cylinders a certain amount is taken up by the lymphatics, enters the blood, and reaches the spinal cord by other nerve-fibers (Jacobson and Pease, in "Annals of Surgery," Sept., 1906). The essential basis of tetanus is spreading irritation of the motor portion of the spinal cord accompanied by extreme reflex excitability which is due to poisoning of sensory neurones (Meyer and Ransom). The irritation of the motor cord produces tonic contraction of the muscles; the excitation of the sensory neurones is responsible for clonic convulsions. There are no instances on record of second attacks of lockjaw, but it is not believed that one attack confers any prolonged immunity.

**Local Tetanus.**—In some cases local symptoms precede widespread evidences of tetanus. Experimental tetanus in animals "exhibits almost without exception as its earliest manifestations those of a purely local character and which are at first restricted to the neighborhood of the inoculation. This is now understood to be due to the absorption of the toxin by the motor nerve of the part. The conditions favoring the local appearance of tetanus are a short motor nerve, as in head injuries; an injury to a nerve-trunk permitting the rapid absorption of a large amount of toxin; the production of a meager amount of toxin or the presence of something which prevents the admission of a large amount of toxin into the circulation (Nathan Jacobson and Herbert D. Pease, Ibid.). Cases with local symptoms in the beginning are apt to have had long periods of incubation, are apt to be cured, and usually endure a considerable time.

*Mortality.*—It is a very fatal disease. Acute tetanus has a mortality of from 75 to 90 per cent.; chronic tetanus, from 40 to 50 per cent.; postoperative tetanus, of over 85 per cent. (Peterson's estimate in "Jour. Am. Med. Assoc.," Jan. 8, 1910).

Local tetanus is apt to terminate in recovery. Tetanus produces death by overwhelming the patient with toxin by exhaustion resulting from repeated convulsions, by spasm of the glottis, or fixation of the respiratory muscles.

**Symptoms.—Acute tetanus** begins within ten days of an accident. The usual period of incubation is from three to five days. Evler had the rare and dreadful experience of contracting the disease from a victim of tetanus on whom

he was operating, and the extreme good fortune to recover. He reports his own case and says that various short and transient early symptoms occur which the patient is apt to attribute to the healing wound. Among these he mentions restlessness, sleeplessness, bad dreams, oppression of breathing, frequent and difficult micturition, headache, fatigue, vertigo, chilliness, darting pains in various regions, and perhaps pains about the injured part. If an extremity is the seat of wound, it may swell and remain swollen even when elevated, and it feels hot, but is not discolored. Before long the wound becomes tender and glands often swell. There may be painless contractions and tremors of injured extremity. Single groups of muscles may undergo tonic contraction ("Berliner klin. Wochensch.," Sept. 12, 1910). In most cases of tetanus the first symptom noted by the patient is stiffness of the jaw on opening the mouth. In some cases the first symptom is stiffness of the neck, and the patient believes he has "caught cold." In any case the neck soon becomes stiff, and finally both the neck and jaw become as rigid almost as iron. The fixation of the jaw is called *trismus*. The muscles of deglutition become rigid on attempts at swallowing. The muscles of the back, legs, and abdomen are thrown into tonic spasm, but the arms rarely suffer. If the infected injury is on the hand or foot, that extremity usually is found to be rigid. Spasm of the face muscles causes the *risus sardonicus*, or sardonic smile (contraction particularly of the *musculus sardonicus* of Santorini). The contraction of the muscles of the back is often so powerful as to bend the patient into a curve like a bow and allow him to rest only on his occiput and heels. This condition is known as *opisthotonos*. If he is bent forward, so that the face is drawn to the legs, it is called *emprosthotonos*. If his body is curved sideways, it is designated *pleurosthotonos*. An upright position is *orthotonos*. The spasm may be so violent as to cause muscular rupture.

The characteristic condition in tetanus is one of widely diffused tonic spasm, aggravated frequently by clonic spasms arising from peripheral irritations. These irritations may be drafts, sounds, lights, shaking of the bed, attempts at swallowing, contact of the bed-clothing, the presence of urine in the bladder or of feces in the rectum, or various visceral actions. The clonic spasms begin early in the case and become more frequent and more violent as the disease progresses. The muscles become more rigid and the attitude produced by the tonic contraction of the muscles is temporarily exaggerated. The forcible contraction of the jaw may loosen or break teeth. The spasms of the diaphragm, of the glottis, and of the muscles of respiration may produce death and always produce great dyspnea. The man laboring under a tetanic convulsion presents a dreadful picture; he is bent into some unnatural attitude, the face is cyanotic and wet with drops of sweat, the lips are covered with froth which is often bloody, the eyes bulge and are suffused, and the countenance expresses deadly terror and suffering. The agonizing "girdle pain" so often met with is due to spasm of the diaphragm. Each clonic spasm causes a hideous scream by the constriction of the chest forcing air through a contracted glottis. During the progress of the disease constipation is persistent, and retention of urine is the rule (because of sphincter spasm). The mind is almost invariably entirely clear until near the end—one of the worst elements of the disease. There is obstinate insomnia. Headache is common. Hearing, at first hyperacute, is later impaired. Deafness may arise. Pulse is slow and full, with high tension. In very rare cases delirium arises. I have seen it twice, due, I fancy, in each case to the drugs employed. It might, of course, be due to previous alcoholism. Swallowing in many cases is impossible. Talking is very difficult and it is impossible to project the tongue. The muscles throughout the body feel very sore. The temperature may be normal, but it is usually a little elevated, and always arises just before death. Hyper-

pyrexia sometimes occurs (108°–110° F.), and the temperature may even ascend for a time after death. An injection of serum raises the temperature several degrees. In about 80 or 85 per cent. of cases of acute tetanus death occurs within five days, and many of these patients die within two or three days. Very few puerperal cases recover and practically no cases which follow abortion recover. Of late years the mortality in acute tetanus has slightly diminished. If a patient lives a week, his chance of recovery is good. Death may be due to exhaustion or to carbonic-acid narcosis from spasm of the glottis or fixation of the respiratory muscles.

**Chronic tetanus** comes on late after a wound (from ten days to several weeks). The symptoms are not so severe as in acute tetanus. The muscular spasm is widespread, but it may not be persistent, intervals of relaxation permitting sleep and the taking of food. Chronic tetanus long had a mortality of 40 or 50 per cent., but modern methods of treatment, it has been claimed, have considerably reduced it. According to the report of Jacobson and Pease it is still from 35 to 50 per cent. ("Annals of Surgery," Sept., 1906). The disease may last for some weeks.

*Trismus neonatorum*, or *trismus nascentium*, the lockjaw of the newborn, is due to infection of the stump of the umbilical cord, and is practically invariably fatal. *Hydrophobic tetanus*, *head tetanus*, or *cephalic tetanus* is a condition in which the spasms are confined chiefly to the face, pharynx, and neck, although the abdominal muscles are usually also rigid, and in which there is palsy of the seventh nerve. It follows head injuries, and gives a better prognosis than does general tetanus.

Two other forms of tetanus have been produced in animals by experimenters. One is *cerebral tetanus*, produced by injecting tetanus toxin into the brain and characterized by mental symptoms (Roux and Borrell, in "Annals Inst. Pasteur," July, 1897). Another is *tetanus dolorosa*, produced by injecting toxin into the posterior roots of the spinal nerves, and characterized by violent spasms of pain without motor symptoms.

**Diagnosis.**—Tetanus may be confounded with strychnin-poisoning, with hysteria, with tetany, or with hydrophobia. Wood's table (see page 209) makes the diagnosis clear between tetanus, strychnin-poisoning, and hysteria.[1]

Tetany is distinguished from tetanus by the milder nature of the spasms, by the greater limitation of the rigidity, by the fact that spasms begin in the hands or feet, not in the jaw and neck, and in most cases by periods of distinct intermittence.

In hydrophobia tonic spasm does not exist, and if clonic spasms occur they are secondary to suffocative attacks.

**Treatment.**—Far better even than to treat tetanus well is to prevent it. Careful antisepsis will banish it as a sequence of surgical operations as thoroughly as it has banished septicemia. Every infected wound must be disinfected with the most scrupulous care. Every punctured wound is to be incised to its depths and thoroughly cleaned and drained. In a very suspicious wound, such as a Fourth of July injury or a wound from a dung fork, or the entrance into the tissues of a splinter from a stable floor, after the removal of foreign bodies and thorough antiseptic cleansing, dust the wound with antitoxin powder or, better, give hypodermatically 2000 or 3000 units of antitetanus serum. It seems reasonably certain that tetanus antitoxin has prophylactic power; in fact, Jacobson and Pease say that "as a prophylactive measure it merits our fullest confidence" (Loc. cit.). Obviously, this cannot be done for every wound. The procedure is not a certain preventive. Reynier injected antitoxin into a patient on whom he was about to operate because

[1] "Nervous Diseases," by Prof. H. C. Wood.

| Tetanus. | Hysteric Tetanus. | Strychnin-poisoning. |
|---|---|---|
| | Commences with blindness and weakness. | Begins with exhilaration and restlessness, the special senses being usually much sharpened. Dimness of vision may in some cases be manifested later, after the development of other symptoms, but even then it is rare. |
| Muscular symptoms usually commence with pain and stiffness in the back of the neck, sometimes with slight muscular twitching; comes on gradually. Jaw one of the earliest parts affected; rigidly and persistently set. | Muscular symptoms commence with rigidity of the neck, which creeps over the body, affecting the extremities last. Jaws rigidly set before a convulsion, and remain so between the paroxysms. | Muscular symptoms develop very rapidly, commencing in the extremities, or the convulsion when the dose is large seizes the whole body simultaneously. Jaw the last part of the body to be affected; its muscles relax first, and even when, during a severe convulsion, it is set, it drops as soon as the latter ceases. |
| Persistent muscular rigidity very generally, with a greater or less degree of permanent opisthotonos, emprosthotonos, pleurosthotonos, or orthotonos. | Persistent opisthotonos and intense rigidity between the convulsions and after the convulsions have ceased, the opisthotonos and intense rigidity lasting for hours. | Muscular relaxation (rarely a slight rigidity) between the convulsions, the patient being exhausted and sweating. If recovery occurs, the convulsions gradually cease, leaving merely muscular soreness, and sometimes stiffness like that felt after violent exercise. |
| Consciousness preserved until near death, as in strychnin-poisoning. | Consciousness lost as the second convulsion comes on, and lost with every other convulsion, the disturbance of consciousness and motility being simultaneous. | Consciousness always preserved during convulsions, except when the latter become so intense that death is imminent from suffocation, in which case sometimes the patient becomes insensible from asphyxia, which comes on during the latter part of a convulsion and is almost a certain precursor of death. |
| Drafts, loud noises, etc., produce convulsions, as in strychnin-poisoning; may complain bitterly of pain. | Crying spells alternating with convulsions. | The "slightest breath of air" produces convulsion. Patient may scream with pain or may express great apprehension, but "crying spells" would appear to be impossible. |
| Eyes open and rigidly fixed during the convulsion. | Eyes closed. | Eyes stretched wide open. |
| | Partial spasm in the leg, producing in Wood's cases crossing of the feet and inversion of the toes. If all the muscles were involved, eversion would occur, as the muscles of eversion are the stronger. | Legs stiffly extended with feet everted, as the spasms affect all the muscles of the leg. |

there was a case of tetanus in the wards, and yet this man developed tetanus ("Gaz. des Hôpiteaux," July 16, 1901). Thirty light cases have been reported in which prophylactic injections failed to prevent the disease. When in spite of such injections the disease does arise, it is apt to be mitigated in violence. Nevertheless it is sure that animals can be rendered immune to tetanus, and the prophylactic power of antitoxin is warmly advocated by many eminent men. It is extensively and most successfully used by veterinarians to prevent tetanus after castration of horses, and this success is a guide-post to us. The following table is most suggestive (quoted by Heineck in "Surgery, Gynecology, and Obstetrics," Jan., 1909, from Scherck's article in "Jour. Am.

Med. Assoc.," 1906, vol. xlvii, p. 500). It sets forth the Fourth of July injuries treated in St. Louis dispensaries:

| Years. | No. of cases. | Antitetanic serum as a preventive. | Death from tetanus. |
|---|---|---|---|
| 1903 | 56 | no | 16 |
| 1904 | 37 | yes | none |
| 1905 | 84 | yes | none |
| 1906 | 170 | yes | none |

Puerperal tetanus is prevented by antiseptic midwifery, and tetanus neonatorum by the antiseptic treatment of the stump of the cord. In order to obviate all danger of the development of tetanus after vaccination perform the little operation with cleanliness and care properly for the wound and for the pustule. The skin should be cleansed with soap and water, rubbed with alcohol, and washed with boiled water. It should be gently scraped with a knife (which has been boiled) until serum exudes. The virus, taken from a hermetically sealed tube, is applied to the raw surface and allowed to remain exposed to the air until dry. A piece of sterile gauze is laid over the part and is held in place by a bandage. This dressing is changed as may be necessary, and is used until granulation begins, at which time the use of any simple ointment is admissible. Do not apply a shield. The evil of shields is pointed out by Robert N. Willson ("American Medicine," Dec. 7, 1901).

When tetanus exists, always look for a wound, and if one is found, open it; if there are sloughs, cut them away, wash the wound with peroxid of hydrogen and then with hot normal salt solution, dry the wound with gauze, paint the surfaces of the wound with bromin, and secure drainage by packing with iodoform gauze. Dennis disinfects the wound with a solution of trichlorid of iodin (0.5 per cent.).

Surgeons of a former day were accustomed to amputate for tetanus if the wound was upon an extremity. When we reflect that the poison-producers are in the wound and not in the circulation, it seems a reasonable treatment. As a matter of fact, it never does any good, because, when the symptoms begin, the toxin has already entered into the nerve-cells and become fixed. Kitasato has shown that if a mouse is inoculated with tetanus near the root of the tail, excision of the tail and cauterization of the stump will not prevent tetanus unless it is performed within one hour of the inoculation. Nocard inoculated sheep near the root of the tail with tetanus spores, and although the moment symptoms appeared he amputated well above the point of inoculation, the animals died of the disease. We must regard amputation as a useless method of treatment. The cases of Porter and Richardson in which bacilli were found in adjacent glands suggest the wisdom of removing any enlarged glands which chould have received lymph from the wound.

Keep the sufferer from tetanus in a darkened, well-ventilated, and quiet apartment, so as to exclude as far as possible peripheral irritation. Watch for the occurrence of retention of urine, and use the catheter if necessary. Secure movements of the bowels by administering salines, castor oil, croton oil, or enemas. Stimulate freely with alcohol, and give fluids by hypodermoclysis. Give plenty of concentrated liquid food unless swallowing causes convulsions, then feed by the rectum. If swallowing causes convulsions some surgeons give an inhalation of nitrite of amyl before an attempt is made to swallow. If this treatment does not make swallowing possible, then partially anesthetize the patient and feed him by means of a pharyngeal tube passed through the nose. It may become necessary to abandon mouth feeding. Large doses of the bromid of potassium, or of this drug with chloral, give the best results, as far as drug treatment is capable of giving results. If bromid is used, give about 1 dr. every four to six hours. Chloretone has warm advocates.

It is given in large doses. It abates rigidity and diminishes the number and severity of clonic convulsions. Other drugs that have been used with some success are gelsemium, morphin, curare, infections and fomentations of tobacco, physostigmin, anesthetics, cocain, and cannabis indica. An ice-bag to the spine somewhat relieves the girdle pain. Hot baths have been advised. It is said that venesection followed by the intravenous infusion of saline fluid does good. This procedure is followed by a free flow of urine and by lessening of the number of the paroxysms. It may be repeated several times during a few days (E. J. McOscar, in "American Medicine," Sept. 14, 1901; A. V. Moschcowitz, in "Med. News," Oct. 13, 1900).

Yandell says, in summing up Cowling's report on tetanus:[1] "Recoveries from traumatic tetanus have been usually in cases in which the disease occurs subsequent to nine days after the injury. When the symptoms last fourteen days recovery is the rule, apparently independent of treatment. The true test of a remedy is its influence on the history of the disease. Does it cure cases in which the disease has set in previous to the ninth day? Does it fail in cases whose duration exceeds fourteen days? No agent tried by these tests has yet established its claim as a true remedy for tetanus."[2]

It is now claimed by some observers that we have a remedy which fulfils the requirements of Yandell in the tetanus antitoxin serum. Behring's serum is said to be six times as strong as Tizzoni's, but it is difficult or impossible to estimate the exact power of either. Behring and Kitasato succeeded in immunizing animals, and Tizzoni and Cattani assert that the antitoxin is an enzyme. The antitoxin destroys the activity of the toxin and is obtained from an immunized horse.

If injected subcutaneously it is absorbed very slowly, and even twenty-four hours or more after such an injection a considerable amount remains unabsorbed in the tissues. It is not absorbed at all by the nervous structures. It is eliminated rapidly and unaltered in the urine, feces, and sweat. It seems to be harmless and its immunizing powers are certain. Its curative power is very much less certain. Hypodermatic injections are practically useless. Intravenous injections are of more service, but even then the antitoxin only grasps the toxin in the blood and fails to reach that in the nerves, nerve-cells, and nerve-tracts. Some practise intramuscular injections, but 7 acute cases so treated died, a mortality of 100 per cent. (Jacobson and Pease, "Annals of Surgery," Sept., 1906). Injection into the theca of the cord (intraspinal injection) by means of lumbar puncture is an attractive method, but the inability of nerve-elements to absorb antitoxins when the pia intervenes is an argument against it, though in 1 violent acute case of my own, occurring in a boy, recovery followed this method. In 7 acute cases treated by this method the mortality was 57.1 per cent. (Jacobson and Pease, Ibid.). John Rodgers injected antitoxin into the cauda equina and nerves and apparently hopeless cases recovered ("Med. Record," July 2, 1904). Injection into a nerve (intraneural injection) is a more rational method, but even this plan is only of service in localized tetanus, the main nerve about the part tetanized being injected (Küster, in German Surgical Congress of 1905). However antitoxin is given, the dose must be large if any good is to be done. Serum is usually prepared as follows: A horse is injected repeatedly with the toxins obtained from cultures of tetanus bacilli, the strength of the injections being gradually increased. Eventually the animal becomes immune to tetanus. Some days after the final injection a cannula is placed in the jugular vein of the immunized animal, blood is drawn into a sterile vessel and is permitted to coagulate during twenty-four hours, and at the end

[1] "American Practitioner," Sept., 1870.

[2] Quoted by Hammond, in his "Diseases of the Nervous System."

of this period the serum is separated from the clot, is evaporated to dryness in a vacuum over sulphuric acid, and the powder is placed in hermetically sealed glass tubes. In order to use the serum, dissolve the powder in sterile water, in the proportion of 1 gm. to 10 c.c. The fluid serum sold in the shops bears this proportion to the powder. The serum can be given subcutaneously or intravenously, or can be injected into the brain or under the cerebral dura or the spinal arachnoid, or into a nerve. If used subcutaneously, from 20 to 30 c.c. of the fluid serum should be injected into the abdominal wall, and this dose should be given every six or eight hours until there is improvement. Then from 5 to 10 c.c. should be given every six or eight hours. As the symptoms abate the dose is lessened and the intervals between the doses are increased. In a violent cases of tetanus the first dose should consist of 40 to 50 c.c., and this can be repeated in four or five hours. In a case of tetanus which recovered, reported by Mixter, enormous doses were given. This patient received in the aggregate 3400 c.c. of serum, or 285 c.c. a day.[1] In 47 acute cases treated by subcutaneous injection the mortality was 82.6 per cent. In 30 acute cases treated by a combination of either subcutaneous, intraspinal, intravenous, or intracranial injections the mortality was 93.1 per cent. (Jacobson and Pease, in "Annals of Surgery," Sept., 1906). Roux and Borrel maintain that the toxins of tetanus pass from the blood into nervous tissue and are fixed in the nerve-cells. As the antitoxin when given hypodermatically or intravenously remains in the blood, it can only antidote the poison in the blood and not that in the nerve-cells. These observers advise that the antitoxin be placed where the toxins are active—that is, that it be thrown into the cerebrum (intracerebral injections). The skull is trephined or opened with a small drill, a blunt needle is passed to the depth of $1\frac{1}{2}$ inches into the frontal lobe, and the serum is slowly injected. Abbe follows Kocher; uses a local anesthetic and bores a very small hole through the skull midway between the outer angle of the orbit and the middle of a line running across the head from one external auditory meatus to the other. The serum should be concentrated. One gram of dry antitoxin is dissolved in 5 c.c. of water, and this amount is the proper dose. The opposite frontal lobe should also be injected either at once or the next day. Even when serum has been injected into the cerebrum it should also be given subcutaneously. Abbe employed intracerebral injection in 5 severe cases and 3 of them recovered. He is a strong believer in the method (Ibid.). Moschcowitz has collected 38 cases so treated and claims that one-half of them recovered. Cerebral abscess followed in 1 case ("Med. News," Oct. 13, 1900). Tuffier has reported a successful case in which he injected 10 c.c. of serum into each frontal lobe ("Gaz. heb. de Med. et Chir.," July 4, 1901). The method has of late been practically abandoned in spite of the early favorable reports.

The value of the tetanus antitoxin in acute tetanus is more than doubtful. In the Russo-Japanese War its use was abandoned, and d'Autma found it of no value in the tetanus cases which followed the Italian earthquakes of 1908. A serum injection in tetanus raises the temperature and may cause depression of the circulation, severe headache, vomiting, diarrhea, perhaps, in very rare cases, death. Under its use the mortality from acute tetanus is said to fall from nearly 90 to 75 per cent., but the figures above given do not sustain this contention. Neither do the figures indicate that the mortality in chronic tetanus has been greatly influenced by it.

Kitasato has shown that injections of iodoform render animals immune, and Sonnani has maintained that this drug placed in a wound prevents the disease. If antitoxin is not obtainable, give hypodermatic injections of iodoform, 3 to 5 gr. *t. i. d.*

[1] "Boston Med. and Surg. Jour.," Oct. 6, 1898.

*Bacelli's treatment* was introduced by Bacelli, of Rome, in 1892. It consists in the hypodermatic injection of carbolic acid, which is thought to grasp tetanus toxin and mitigate its virulence or even make it inert. The drug is also thought to be sedative and to lower temperature. Tetanus toxin is destroyed by carbolic acid (shown by Tizzoni and Cattani in 1890). Kitasato pointed out in 1891 that cultures of tetanus bacilli can be rendered sterile in thirty minutes by a 5 per cent. solution of carbolic acid. The usual dose is 15 minims of a 3 per cent. solution every two hours. Three times this amount can be given in a day. To avoid irritation Maragliano uses a 5 per cent. solution in sterile olive oil. Favorable results are claimed for the plan. Bacelli has collected 190 cases (all severe or very severe). There were 94 severe cases and 92 recovered (2½ per cent. mortality). There were 38 very severe cases and 22 recovered (see Bacelli, in "Berliner klinische Wochenschrift," June 5, 1911). Even the occurrence of carboluria does not cause the surgeon to suspend the treatment. These results are the best ever given in tetanus and will cause us to try the treatment.

The hypodermatic injection of an emulsion of fresh brain-matter has been advocated on the ground that brain-matter and tetanus toxin have a mutual affinity (Krokiewicz). The results are not conclusive

Mathews reports cure in 2 cases following the very gradual introduction into a vein of a solution containing sodium chlorid, sodium citrate, sodium sulphate, and chlorid of calcium ("Jour. Am. Med. Assoc.," August 29, 1903). Cure of acute tetanus has followed the intraspinal injection of a solution of magnesium sulphate, which drug, Meltzer has shown, strongly stimulates inhibition. Blake has reported such a case ("Jour. of Surgery, Gynecology, and Obstetrics," May, 1906). If magnesium sulphate is used, 2 c.c. of a 25 per cent. solution are injected into the subarachnoid space of the cord, after first removing an equal quantity of cerebrospinal fluid. Some hours after such an injection there is marked muscular relaxation lasting a number of hours. Not unusually there is lowering of respiratory rate. When the improvement ceases another dose is given. Heinecke (Ibid.) reports a successful case and collects 12 other cases so treated, 7 of which died. Smithson used this drug and, although the patient died, there was not a convulsion after the injection.

Murphy reports the recovery of a case after spinal puncture and injection of morphin and eucain into the theca of the cord ("Jour. Am. Med. Assoc.," August 13, 1904). The only case of acute tetanus I have ever had recover was treated by intraspinal injection of antitoxin. I have known 3 cases of chronic tetanus to recover: 1 was treated by chloral and bromid only, 2 were treated by chloral and bromid and antitoxin subcutaneously.

## XIII. SURGICAL TUBERCULOSIS

Tuberculosis is an infective disease due to the deposition and multiplication of tubercle bacilli in the tissues of the body. The term *surgical tuberculosis* is applied to all of those numerous tuberculous lesions that may demand surgical treatment. Such lesions may exist in different structures, often seem clinically to be strictly localized processes, and in many instances may be extirpated, drained, or sterilized. Among the conditions placed under the heading of surgical tuberculosis are: Tuberculosis of glands, of bones, of joints, and of the skin. These lesions are most common in children, the majority of cases are curable, and they are not so often associated with or followed by pulmonary phthisis as are some other tuberculous lesions. They tend in many cases to remain local and, beyond doubt, a considerable number

of them are due to infection with bovine bacilli. Tuberculosis is characterized either by the formation of tubercles or by widespread cellular proliferation (diffuse tubercle) or by fibrinous exudation, which is very rich in cells. Tuberculous conditions tend to caseation, sclerosis, or ulceration.

A *tubercle* is a non-vascular infective focus, appearing to the unaided vision as a semitransparent gray or yellowish mass the size of a mustard-seed. The microscopic tubercle is the most characteristic evidence of the disease. The microscope shows that a gray tubercle consists of a number of cell-clusters, each cluster constituting a *primitive tubercle.* A typical primitive tubercle shows a center consisting of one or of several polynucleated giant-cells surrounded by a zone of epithelioid cells which are surrounded by an area of leukocytes. When the bacillus obtains a lodgment the fixed connective-tissue cells multiply by karyokinesis, forming a mass of nucleated polygonal or round cells. These cells are connective-tissue cells and are derived particularly from endothelium and are called *epithelioid cells* from their resemblance to epithelial cells. Early in the development of a tubercle blood channels lined with epithelioid cells exist, but continued cell proliferation blocks the channels and at the same time the blood-supply of the growth is further limited by the pressure of proliferating perivascular cells and the proliferation of the endothelial cells of adjacent vessels. Some of the epithelioid cells proliferate, and others attempt to, but fail for want of blood-supply. Those which fail to multiply succeed only in dividing their nuclei and enormously increasing their bulk (giant-cells). Giant-cells, which may also form by a coalescence of epithelioid cells, are not always present. Giant-cells are not certain evidence of tuberculosis, for they occur in syphilitic lesions. The presence of irritant bacterial products induces surrounding inflammation and numbers of leukocytes gather about the epithelioid cells (Fig. 101).

Fig. 101.—Synovial membrane, showing giant-cells (Bowlby).

The bacilli, when found, exist in and about the epithelioid cells, and sometimes in the giant-cells. When bacilli enter the tissues they are often killed. If they enter in large numbers or are peculiarly virulent they induce chronic inflammation, granulation tissue forms, and the cells of the focus often have the characteristic arrangement described above. The bacilli are not pyogenic and suppuration means secondary infection. If mixed infection of any considerable area occurs, the patient is apt to develop fever and to perish from exhaustion, amyloid disease, dissemination, or a terminal infection. In rare cases the tuberculous area is destroyed and cure is brought about. A tuberculous focus tends strongly to degenerative changes because of the local anemia and the presence of bacilli. If numerous active bacilli are present *caseation* takes place. This is coagulation necrosis due to the action of bacteria upon a non-vascular area. It starts at the center of a tuberculous focus and spreads toward the periphery and finally forms masses like cheese. When caseated material is mixed with serum *tuberculous pus* is formed.

A caseated focus may be surrounded or encapsulated by fibrous tissue. When this happens the tuberculous process may remain latent for months or years, perhaps awakening into activity as the result of a traumatism or lowered general resistance. A caseated focus may be cured by growth of fibrous tissue which replaces the tuberculous focus. This is cure by sclerosis. A caseated area may calcify. Even when tuberculous pus forms encapsulation

may occur, the fluid being absorbed, and the remains being surrounded by fibrous tissue. Whenever tubercle bacilli consume all available food they die or remain latent. If they die the granulations are converted into fibrous tissue and the part is healed. If they remain latent they may at any time become again active. Infiltrated tubercle is due to the running together of many minute infective foci, or to widespread infiltration without any formation of foci. Infiltrated tubercle tends strongly to caseate. The description of a tubercle previously given relates to the common *reticulated tubercle*. Two other varieties exist.

The *fibrous tubercle* is much richer in dense connective tissue than is the ordinary tubercle. It forms when bacilli are greatly weakened or killed. When this happens embryonal cells cease to degenerate, and ordinary inflammation results in fibrous tissue formation. Fibrous tubercle is evidence of an effort at cure.

*Hyaline tubercle* results from hyaline degeneration of the reticulum of an ordinary tubercle and is the early stage of coagulation necrosis.

Knowledge of recent years proves that the bacillus of tubercle may fail to cause the supposed essential lesions outlined above, but may induce instead tissue changes identical with those due to various other organisms. Some of these changes are acute, some are chronic. In some there is hyperemia, in some serous exudation, in some fibrous exudation, in some cellular hyperplasia, perhaps with parenchymatous degeneration, or perhaps with sclerosis causing cirrhosis (Rudolph Matas, in "Southern Med. Jour.," Oct., 1911). Matas says that this group is non-tuberculomatous, atypical, and non-specific.

**The Incidence of Tuberculosis.**—Tuberculosis is the most widespread of diseases, being particularly common in northern countries, in civilized regions, and in great cities. Both men and domestic animals suffer from it, and it is occasionally met with in captive wild animals. It may even occur in cold-blooded animals. It is rare in savage races and extremely rare in wild animals dwelling under natural conditions.

Many people possess lesions of tuberculosis who present no clinical evidence of it. Careful necropsies, with microscopical studies and observations with the aid of tuberculin, prove this. The greatest death-rate of those infected is in childhood and early adult life (Burkhardt, in 1903, quoted by Hamman and Wolman in their book on "Tuberculin").

How many persons die of tuberculosis is a much debated point. Some writers claim that consumption of the lungs alone kills one-third of all that die; and if the deaths from various other tuberculous lesions are added to this, it will be seen what an enormous part the disease plays in the mortality tables. Many observers hold that one-third of the human race suffer from tuberculosis, and that in every country the remaining two-thirds free from the disease are every moment in danger of acquiring it. Evans has maintained that of the 35,000,000 deaths that occur yearly in the world, 5,000,000 are the result of tuberculosis. Pflügge thinks that one-seventh of the race die of tuberculosis. Sherman G. Bonney, in his work on "Pulmonary Tuberculosis," asserts that "from 85 to 95 per cent. of the human race have been at some period of life the subject of tuberculous infection" and that 1 person in 7 dies of the disease.

This enormous incidence of the disease, however, is disputed by some authorities; notably, by G. Cornet (Nothnagel's "Encyclopedia of Practical Medicine"). This observer states that apparently one-seventh of all deaths result from tuberculosis, and that some pathologists have reported that in one-third of all necropsies tuberculous lesions are found; but that these statistics are obtained from institutions where only the very poor are cared for, and that the percentage of tuberculosis is vastly lower in the better classes of

the community. The exact figures, however, are difficult to determine. It is certain that enormous numbers of people are affected with tuberculosis. I believe many affected ones recover, for Naegeli points out that almost all who perish after thirty from non-tuberculous conditions show healed lesions of tubercle. Of 420 adults, 391 (93 per cent.) showed signs of tuberculosis. Spengler claims that every human adult was at some period of life a host for tubercle bacilli. Bollinger stated that in one-fourth of all postmortems upon adults evidences of tuberculous disease are found at the pulmonary apices. Baumgarten asserts that one corpse out of every three showed a tuberculous focus, latent or healed. Tuberculin tests confirm the views of Burkhardt and Naegeli and contradict the opinions of Cornet. Franz injected 400 recruits who had passed their physical examination for the army and 61 per cent. reacted. Von Behring maintains that all of us are "a little tuberculous" (Jonathan Wright, in "New York Med. Jour.," April 2, 1904). Pflügge maintains that from 50 to 70 per cent. of the human race are predisposed to tuberculous infection, and if infected will die of it unless an intercurrent malady destroys them.

**The Bacillus of Tuberculosis.**—The tubercle bacillus was discovered by Robert Koch in 1882. It is a little rod with a length about equal to one-half the diameter of a red corpuscle. It is non-motile, does not form spores, and requires oxygen in order to grow, but it may obtain oxygen from body-cells or fluids. Tubercle bacilli exist in all active tuberculous lesions, and the more active the process the greater their numbers. They may not be found in a tuberculous area, having once existed, but having died for want of nourishment. For instance, in a cold abscess they are frequently absent. Bacilli may be destroyed by a secondary infection, for example, by a pyogenic infection. Even when present, tubercle bacilli may be overlooked. Differential staining may exhibit the bacilli. In the material from an active tuberculous lesion, even if bacilli are not found, injection of the tuberculous matter into a guinea-pig will be followed by the production of the disease, and in these lesions bacilli can be demonstrated. Bacilli may be widely distributed throughout the body. It has long been taught that they may occasionally though seldom be demonstrated in the blood in cases of acute miliary tuberculosis. We have discussed the tubercle bacillus on page 52. The bacillus of leprosy, the smegma bacillus, and the tubercle bacillus are similar, but not identical. Each is an acid-fast bacillus; that is, if stained with an anilin color, mineral acids will not wash out the stain. All acid-fast bacilli are capable of producing lesions that, to some extent at least, resemble tuberculous lesions; but the lesions produced by all, except the tubercle bacillus and the leprosy bacillus, tend to recovery. It is possible that all acid-fast bacilli are branches from a common stem.

The tubercle bacilli obtained from different animals differ considerably, both in morphology and in virulence. Koch asserted, in 1901, that the bacilli of human tuberculosis differ radically from those of bovine tuberculosis, that human tuberculosis cannot be given to cattle at all, and that it is so difficult to transfer bovine tuberculosis to the human being that the danger from infected cattle is utterly trivial and may be disregarded. Ravenal and others have positively opposed this view of Koch's, and there have been reported what appear to be undoubted cases of the transference of tuberculosis from animals to man. There is still dispute upon this point, but most writers believe that bovine tuberculosis and human tuberculosis are essentially the same, although the bacilli present temporary differences due to altered environment. The bacilli of bovine tuberculosis are certainly far less dangerous to man than are the bacilli of human tuberculosis, and the bacilli of human tuberculosis are vastly less dangerous to cattle than are the bacilli of bovine tuberculosis. Human bacilli introduced into cattle may produce chronic

lesions, but they are always non-progressive. The histologic lesions seen in man and cattle are identical and so are the degenerative changes, and, as Baumgarten showed, cattle react to tuberculin obtained from human bacilli.

Nocard reports 2 cases of individuals who wounded themselves while cutting the flesh of tuberculous cattle. Both developed generalized lesions and died. Ravenal strongly opposes the view of Koch and maintains that the bacillus of bovine tuberculosis is highly pathogenic for man ("University of Penna. Med. Bull.," xiv, 238, 1901). The same author has reported 4 cases of undoubted inoculation tuberculosis in the hands of veterinarians. Similar cases have been placed on record by other observers. The entire question is one of immense importance. If Koch is right, there is practically no danger to man in eating tuberculous meat or in drinking tuberculous milk. Most observers endorse the words of the report of the British Commission of 1904. This commission positively maintained that bovine tuberculoiss can be communicated to man.

The bacilli of bovine tuberculosis, when they find lodgment in human tissues, are apt to produce local lesions and seldom disseminate, and vice versâ. It has been proved that many cases of tuberculous cervical adenitis in children, but only 3 per cent. of cases in adults, are due to bovine bacilli. Fifty per cent. of cases of abdominal tuberculosis in children and 20 per cent. in adults are due to bovine bacilli. Some bone cases and a considerable number of joint cases in children depend on bovine bacilli, but very few in adults are so caused. Pulmonary tuberculosis very seldom depends upon bovine organisms. It is thus clear that human infection with bovine bacilli is most common in the young, and that surgical tuberculosis is far more apt to have such origin than other forms. Such infections frequently tend to spontaneous cure.

**Distribution of the Bacilli.**—These bacilli are parasites, and not saprophytes; and the real source of infection is a tuberculous person or animal. Wherever there are tuberculous men or animals the bacilli get into the air. The number that get into the air depends upon the number of animals affected, the seat of the tuberculous lesion in each, the care taken by the victims, and the control exercised by the community.

Tubercle bacilli from an infected individual may get into the atmosphere from the urine, the sputum, the feces, the sweat, the milk, or caseous or purulent material. The bacilli from dried sputum enter the dust, in which, fortunately, they are usually destroyed quickly by the complete dryness, the oxygen of the air, and the sunlight; but under some circumstances they may retain their virulence for weeks or even for months. The infected area itself is usually the direct source of the bacteria from a given case of tuberculosis, but this is not invariably so; for a tuberculous woman with a healthy mammary gland may secrete milk containing tubercle bacilli, a consumptive free from genito-urinary tuberculosis may occasionally pass urine containing bacteria, a cow may give tuberculous milk when the udder is not diseased, and tubercle bacilli may enter the bile of a tuberculous patient. The Third Interim Report of the Royal Commission on Tuberculosis states positively that the milk of tuberculous cows may contain bacilli even when the udder is not diseased. It is probable that flies and insects may transmit infection (Lord, in "Boston Med. and Surg. Jour.," 1904, cli); and it is certain that putrefaction does not surely destroy tubercle bacilli. This is proved by the fact that living bacilli may be passed in the feces of an animal that has been fed on tuberculous meat, and that they may be found in the feces of an individual suffering from intestinal tuberculosis. We are thus justified in concluding that slaughter-house waste, if improperly disposed of, is a danger to the community.

**Routes of Infection.**—An individual may acquire tuberculosis by inhaling tuberculous material (*inhalation tuberculosis*), by swallowing tuberculous

material (*ingestion tuberculosis*), and by inoculation with tuberculous material (*inoculation tuberculosis*). Infection of the lungs may be brought about by the inhalation of dried tuberculous sputum or dust carrying tubercle bacilli. Ingestion tuberculosis may follow the eating of tuberculous meat, the drinking of tuberculous milk, or the consumption of uncooked articles on which tubercle bacilli have gathered. It has been shown that the lacteals may take up tubercle bacilli from the intestine, even if there is no intestinal lesion; and that bacilli can pass through the thoracic duct and into the blood, and lodge in some tissue, particularly the pulmonary tissue, so inducing tuberculosis. They are apt to lodge at any point of least resistance; and if not caught up in the lungs, will tend to be arrested in an irritated gland or in some region that has been the seat of a trifling injury—for instance, in an epiphysis that has been strained. It is a peculiar fact that a trivial injury constitutes a point of least resistance; but a severe injury, such as a fracture of a bone, does not do so. Baumgarten was a strong believer in the idea that bacilli enter the organism with the food and von Behring now warmly advocates the same view, teaching that bacilli enter the organism of every person in early life. They may be destroyed by tissue resistance, but if not destroyed have a period of latency and, finally, perhaps after years, become active and cause the disease ("Deutsche Med. Woch.," Sept. 24, 1903). Calmette and Vanstenberg ("Annales de l'Institut Pasteur," 1906) have long insisted that infection is chiefly by the alimentary canal and that inhalation infection is rare.

It is certain that inoculation may be followed by tuberculosis. The inoculation of tubercle bacilli in the intestine may produce intestinal ulceration. It has been shown experimentally that rubbing the bacilli into the nasal mucous membrane may produce a local area of disease. Inoculation of the skin may result from a wound, the bacilli being carried into the wound itself. The victims of cutaneous inoculation are usually butchers, veterinary surgeons, physicians who have made postmortem examinations, and workmen who handle hides. In these cases, as a rule, an ulcer promptly forms at the point of inoculation; but in some few cases the wound heals soundly and tuberculous lesions develop in several or many weeks later in the wound area or in the neighborhood. In still rarer instances no apparent inflammation or ulceration occurs in or around the seat of inoculation, but the anatomically related lymph-glands become tuberculous. In other cases adjacent bone or an adjacent joint becomes tuberculous. Disease of the lungs may follow cutaneous inoculation.

A number of cases of inoculation tuberculosis have been reported. Not a few pathologists have developed anatomic tubercle (see page 247). Schmidt records a case of tuberculous ulcer on a woman's lip due to a bite from her tuberculous husband. It is recorded that a tuberculous person inoculated others while tattooing them, the needle having been moistened with saliva. Letulle's case of inoculation tuberculosis was a woman, who, while scrubbing the floor of the room in which her tuberculous husband had died, ran a splinter into her hand. The wound became tuberculous. Bosanquet ("Lancet," Jan. 13, 1912) refers to a laundress who infected a whitlow with tuberculosis while washing infected linen. I have treated a physician who inoculated his finger while making culture studies with tuberculous material. In this case the axillary glands became tuberculous and were removed. I have also seen a tuberculous ulcer of the forearm in an attendant of a lunatic asylum who had been bitten by a tuberculous patient. Inoculation tuberculosis occasionally follows circumcision, as practised by an orthodox rabbi, the operator being tuberculous. A ritual operator (as Bosanquet calls him) stops bleeding either by applying his mouth to the wound, or by squirting wine from his mouth upon the wound. There have been reported apparent cases of direct inoculation of the genito-urinary tract during sexual intercourse.

If there has been some definite injury of the tissues, inoculation may follow a simple rubbing of tubercle bacilli into a part.

When the mother's ovum is tuberculous, the disease may be directly transmitted to the fetus, producing the condition known as congenital tuberculosis. Rosenberger found bacilli in the blood from the umbilical cord of the placenta of a tuberculous mother. This proves that congenital tuberculosis may exist even when the ovum is not known to be tuberculous, and also that a child born of a tuberculous mother is, if not immune to the bacteria, tuberculous from the moment circulation is established between embryo and mother. Baumgarten believes that bacilli may pass the placenta, enter the fetus, and remain latent for years. Latent bacilli have been found in normal lymph-nodes (Harbitz, in "Jour. Infect. Diseases," vol. ii, 1904); this proves that latency is possible. However common the direct transmission of bacilli may be, the direct transmission of the disease is unusual, but the transmission of an hereditary predisposition to infection is not unusual. In spite of recent assertions to the contrary, I believe that there is such a thing as hereditary predisposition to tuberculosis. The experience of the human race uniformly confirms the belief in predisposition. In some cases of tuberculosis we can satisfy ourselves clinically as to the cause of the infection. For instance, when an individual is injured with an object known to carry tubercle bacilli, if an ulcer of the skin forms, and the adjacent lymphatic glands enlarge, the deduction is obvious. In other cases it is impossible to make up our minds as to the cause of a tuberculous lesion. For instance, we can only guess that a person has inhaled tuberculous material or has eaten tuberculous food. If in inoculation tuberculosis no lesion arises at the point of entry, the opinion as to the causation will be founded merely upon guesswork.

It seems certain that when the bacilli of tuberculosis enter into the body, if they are not destroyed by the body-resistance, they may produce a local lesion at the site of inoculation, or pass to the nearest lymphatic glands or to some point of least resistance, and there establish disease. The first lesion is known as the primary focus, and from this focus disease may be disseminated to the most distant parts. The bacilli enter readily if there is a wound or an abrasion; but in exceptional circumstances they may enter through unbroken skin and undamaged mucous membrane. Any structure may become tuberculous, but some structures are much more liable to do so than others. The lungs are very liable; the conjunctiva is very resistant.

The bacilli are generally distributed by the lymph, but may enter the blood. Those which do enter the blood may pass out in the urine or feces, may produce local lesions, or may induce advancing and widespread tuberculosis. Dissemination by the lymph-stream is known clinically to occur, and it means slowly advancing tuberculosis with localization of lesions. In dissemination by the lymph-stream, the dissemination is usually in the normal direction of the lymph-current; but if the lymph-vessels become blocked, lymph-regurgitation may occur, and then the dissemination takes place in a direction opposite to the normal flow of the lymph-current.

**Latent Lesions.**—By a latent lesion we mean a non-progressive or a healing lesion which gives no clinical evidence of its progress. Such a lesion is most apt to be in a lung or in a gland. It may serve to furnish bacilli to distant parts and hence be responsible for secondary lesions. It may give toxin to the blood and thus induce distant trouble. The frequency of latent lesions becomes evident when we test apparently healthy adults with tuberculin. Although we state that a latent lesion causes no symptoms, we had better say presents no symptoms to suggest tuberculous involvement of the part. Hollos, of Hungary, insists that such an area contains toxin-forming bacilli, and that the poisons taken up from it by the circulation cause a chronic toxemia productive of

numerous symptoms. Such symptoms are usually thought to be due to anemia or neurasthenia. This subject has been brilliantly discussed by Matas in the "Southern Med. Jour.," Oct., 1911.

**Products of the Tubercle Bacilli.**—A great variety of products are formed by the tubercle bacilli, and among them we may mention alkaloids, toxalbumins, fatty acids, and ferments. Experimental injection of the toxalbumins produces inflammation; and of the alkaloids, fever. It has been shown by Maragliano that injection of the toxalbumins actually lowers the temperature. Beyond any doubt, the culture material in which tubercle bacilli are growing contains poison; and the bodies of the bacilli themselves contain poison. The poisons in the culture-medium are called *extracellular poisons*, and those within the bacilli are called *intracellular poisons*. It is quite probable that the former poisons are identical with the latter, and have merely passed from the bacilli into the culture-medium.

*Tuberculin.*—It was proved some time ago that dead bacilli are toxic and, if experimentally injected, induce a toxic condition in the animal, cause inflammation of the kidneys, and sometimes produce subsequently cold abscess at the seat of injection. In 1890 Koch collected the poison from dead bacteria in the form of a liquid, which he called tuberculin.[1] A number of different methods of extracting such poison have been suggested; hence, there are a number of different tuberculins, not one of which contains living bacilli. Koch has made several himself. His early tuberculin was made by making a glycerin-bouillon culture of tubercle bacilli, evaporating on a water-bath to one-tenth of its volume, and filtering out the dead bacilli. The filtrate contained tuberculin mixed with glycerin. It is now known as original or old tuberculin or OT. Later Koch prepared tuberculin from virulent cultures of bacilli, dried, ground up, and mixed with water, the fluid being centrifuged for forty-five minutes. When centrifuged, two layers separate. The upper layer, which is white and opalescent, contains the elements soluble in glycerin, is like the old tuberculin, and is called TO. The lower layer contains an emulsion of insoluble elements of high immunizing power, and is called TR. In 1901 Koch presented another tuberculin of dried bacilli in equal amounts of glycerin and water. It is called bacillary emulsion or BE. This is really a vaccine.

It was discovered by Koch that tuberculous animals are much more strongly affected by an injection of tuberculin than are healthy animals. The most positive reaction is noted in the tuberculous area; but, as a rule, there is also a reaction in the area where the injection is made. We get no reaction from the administration of tuberculin by the stomach,[2] but occasionally can obtain it by the inhalation of the dried material. If a moderate dose of tuberculin is injected into a non-tuberculous animal, there may be a trivial redness at the point of injection and a slight and temporary rise of temperature; or there may be no evidence of reaction whatever. An injection in a tuberculous animal, however, is followed by distinct inflammation at the seat of injection, and a positive reaction in the tuberculous area. This area undergoes congestion or inflammation, leukocytes collect around it, and the part tends to necrosis and is liable to break down. It is not that the bacilli are killed, but, rather, the tissues containing the bacilli die.

In addition to the changes already mentioned there is elevation of temperature. If the dose has been small, there may be only a slight feeling of coldness to usher in the rise of temperature; but if the dose has been large, there is usually a distinct chill. This chill comes on eight to twelve hours after the

[1] "Deutsch. med. Wochenschr.," 1891, xvii.

[2] Latham and S. Solis-Cohen claim results from oral administration. Moller and Heinemann seem to demonstrate that those clinicians are mistaken ("Deutsche med. Woch.," Oct. 5, 1911.

injection and is accompanied and followed by elevated temperature. The fever lasts from four to twenty-four hours, and the temperature may be elevated from 2° to 5° F. The febrile condition is accompanied by pain in the head, limbs, and back, with increased rapidity of the circulation, restlessness, weakness, and usually nausea. As the temperature passes to normal all the symptoms disappear. The slight elevation of temperature which may be noted after tuberculin has been injected into a non-tuberculous animal is not ushered in by a chill, and does not exceed 1° F. unless a very large dose has been given. We thus note that the injection of tuberculin may be of the greatest possible value in diagnosis.

A person with a thoroughly healed lesion does not, and a far-advanced case may not, react to tuberculin.

Much has been written on the reaction of non-tuberculous human beings to tuberculin. Such reaction is said to occur in leprosy, in convalescents from acute illnesses, in syphilis, and in actinomycosis. Many supposedly healthy people react to tuberculin. Some react to a moderate dose, some only to a large one. We are now convinced that the tuberculin reaction is specific and that any one who exhibits it possesses a tuberculous focus, active, latent, or healing. Reaction means infection, but not of necessity disease with clinical evidences. Young children, especially infants, are very refractory to tuberculin because young children are seldom tuberculous.

A real, complete reaction to tuberculin has three elements:

1. A constitutional reaction manifested by fever.

2. A local reaction manifested by the indication of swelling and redness, a nodule, or an infiltrating area in the region of puncture (stick reaction). Sometimes adjacent glands swell.

3. A focal reaction, that is, inflammation about the lesion. ("Tuberculin," by Hamman and Wolman.)

A good many observers have grown fearful of injecting tuberculin, believing that it is liable to cause the tuberculous focus to spread, or actually to lead to the development of disseminated tuberculosis. Virchow was of this opinion. That such a condition may follow the use of large doses seems certain, but moderate or small doses appear to be entirely safe. Flick has pointed out that if a blister is applied to a tuberculous person a distinct febrile reaction appears a number of hours after the application. This is due to the absorption of toxic material, probably tuberculin, from the blister. It is known that in a tuberculous animal certain excretions (urine) and serous exudates contain tuberculin. Mérieux and Baillon show that if a tuberculous person is blistered the fluid of the blister, injected into a tuberculous animal, produces a definite reaction. This proceeding is of diagnostic value. A fluid containing tuberculin comes from the blister upon the tuberculous person and he is proved to be tuberculous by injecting the blister fluid into another tuberculous animal.

**Resistance of Bacilli.**—Among the antagonistic elements we have mentioned oxygen, dryness, and sunlight. Moist heat, at the temperature of boiling water, is rapidly fatal. A 5 per cent. solution of carbolic acid is one of the most powerful of germicides. Full-strength alcohol is next in point of power. Corrosive sublimate is not a satisfactory germicide. Formaldehyd is fatal only after long exposure. Iodoform and ether is a reasonably powerful mixture.

That the virulence of tubercle bacilli varies under different circumstances is sure. Under some circumstances they may be extremely powerful; under others, nearly innocuous. The liability to infection depends, probably, in part, on individual predisposition, and certainly, to a great extent, on the number and the virulence of the bacteria.

**Immunity.**—It seems likely that some persons are immune to tuberculosis. Such persons may come from an ancestral line in which all the predis-

posed died of tuberculosis, so that the immediate ancestors of the line were non-susceptible. The tendency to immunity may be strengthened by proper marriages, and may be weakened by improper marriages; or immunity in a line may be destroyed by the continuance of unfavorable conditions. Spengler claims to find in red blood-corpuscles of healthy men an immunizing body. It is sometimes noticed that during the progress of a localized tuberculous infection a deep-seated tuberculosis (for instance, phthisis) improves. This exhibits a progressive development in the powers of the organism to resist infection by stimulation of the apparatus for opposing infection. That numbers of people get entirely well of tuberculosis is certain and that many such people have secured prolonged immunity is probable. Paretic dements seem to possess a high degree of immunity to tuberculosis (Rosanoff, in "Jour. Amer. Med. Assoc.," February 13, 1909). Of course, the term "immunity" is only relative. No one can be absolutely immune; for when subjected to extremely unfavorable circumstances, or when a number of virulent bacilli are introduced, any one may become tuberculous.

**Predisposition.**—Personally, I believe that there is such a thing as a predisposition toward tuberculosis, just as there is toward many other diseases. Such a predisposed individual possesses temporarily or permanently a condition of the body-cells, body-fluids, or both, that either makes easy the entrance of the bacilli or prevents strong opposition to their multiplication when they have entered. A person is predisposed to an infectious disease when the opsonic index is low, for this indicates lack of phagocytic power in the leukocytes. Predisposition may be increased by some extraneous circumstance, such as occupation, residence, etc., that brings the individual into frequent or prolonged contact with virulent bacteria.

There is certainly such a thing as congenital tuberculosis, and any tissue may be involved in the congenital trouble. Rosenberger showed that blood in umbilical veins from the placenta of a tuberculous mother contained bacilli ("Amer. Jour. Med. Sciences," 1909). Young children are very liable to tuberculosis of the acquired form. According to Professor Behring, many children become infected with tuberculosis in their early years by eating tuberculous food; but such a tuberculosis often remains latent for a considerable length of time, and then develops. This liability depends probably upon the fact that the digestive organs of the child are not so strongly protective against bacteria as are those of the adult.

Do certain individuals possess a special predisposition to develop tuberculosis, and is this hereditary? Hereditary predisposition was once regarded as practically the only cause of the disease, but many thinkers now regard it as of slight importance, although I do not see how we can deny its existence. To do so is to run counter to the experience of the human race in all countries and at all times. We all see how common tuberculosis is in the descendants of tuberculous persons. Hutley studied 432 cases of tuberculosis. In 23.8 per cent. one or both parents had the disease (the father alone in 11.5 per cent., the mother alone in 9.9 per cent., and both in 2.4 per cent.). Some maintain that in 30 per cent. of consumptives, one parent or both parents have been consumptives, and in 60 per cent. a parent or a grandparent has suffered from tuberculosis. Of course, the above statements do not prove that the cases in a family are due to heredity; but that there must be such a thing as hereditary predisposition is indicated by the fact that there are many families living under similar conditions to the tuberculous families, without there having occurred, through several generations, a single case of tuberculosis among their members. A feature that makes us unable to reach a certain conclusion is that tuberculosis is contagious and several members of a family may be infected from one member, even when there is no predisposition to the trouble

by heredity. The mere living in one house may account for the infection. A fact strongly in favor of the hereditary influence is that in a family whose ancestors have been tuberculous and whose members have not lived together, but have been scattered widely over the earth, member after member may die of the disease.

Unhealthy environment particularly predisposes to tuberculosis; and the element of poverty—leading as it does to taking improper or insufficient food, dwelling in an unhygienic room or in an overcrowded building, pursuing an exhausting occupation, working for long hours, and obtaining insufficient amusement and outdoor exercise—also has a most powerfully unfavorable effect. As a class the poor dislike ventilation, take insufficient exercise in the open air, do not get enough sunlight, work in a dusty atmosphere, take insufficient nourishment and eat improper food, live in damp and dirty rooms, are subjected to grinding competition and cruel anxieties, and many of them drink quantities of whisky. City life is a predisposing cause of tuberculosis for many of the foregoing reasons, and particularly because many city workers follow an indoor occupation. The enemies of tuberculosis are sunlight, fresh air, nourishing food, and outdoor exercise, and the limiting of any of these factors favors the development of the disease.

Tuberculosis may occur in any region that man inhabits; although in some regions it is rare, and in others it is excessively common. Its great frequency in some regions is probably due less to climate than to environment, occupation, and heredity; and the greatest predisposition is found in the town dweller. There is much more tuberculosis among males than among females.

Many diseases and conditions predispose to tuberculosis. It is very common in chronic drunkards, in the insane, in the occupants of prisons, almshouses, and reformatories; among negroes in the North, particularly those engaged in indoor occupations; among American Indians subjected to the blighting influences of civilization by formula and routine; and in the sufferers from tertiary syphilis, diabetes, and Bright's disease. Any exhausting malady may be followed by tuberculosis.

**Relation of Trauma to Tuberculosis.**—(Inoculation Tuberculosis is discussed on page 218.) This question is often in dispute and has become of much medicolegal importance. Several times of late, in the courts of Philadelphia, it has been the subject of acrimonious controversy. Suitors affirm the relationship, corporations deny it, and experts wrangle till judge and jurymen do not know what to believe.

There can be no doubt that tuberculosis often becomes manifest in a part after that part has been subjected to traumatism. No one denies this. In fact, in over one-sixth of all cases of bone and joint tuberculosis traumatism is set down as causal.

I do not mean that trauma causes the tissue changes characteristic of tuberculosis. Such changes are always and only produced by the action of tubercle bacilli. I do mean that the injury puts the part in such a condition that the bacilli of tubercle attack the injured tissue, having been unable to attack it when it was in a state of health. The injury creates an area of least resistance. In such an area the cellular activities are no longer able to withstand the action of bacteria. Without an injury it is highly improbable that tuberculosis would ever have arisen in the part. At least the injury determined the localization and multiplication of bacilli and the origin of an active tuberculous focus, and to this extent the injury was causal.

Osteomyelitis is due to pyogenic cocci. It may arise in a bone subjected to traumatism. Traumatism is stated to be a cause. Why should a tuberculous process be placed in a different category? Where do the tubercle bacilli come from? Some maintain that if tuberculosis follows traumatism of a part, there

was a latent and undiscovered tuberculous process in the part before the accident, and that all the accident did was to light up a latent focus into activity; in other words, to precipitate an inevitable event. This contention is true in some few cases; we believe it to be untrue in a large majority of cases.

Some believe that though there may have been no local latent focus, there is, at least, somewhere in the body an area of tuberculosis to furnish the bacilli. This view is true of many, but we do not believe of all, cases. It gains in probability from the established fact that tuberculous bone or joint disease is most apt to arise in those known to have tuberculous infection somewhere about them. It is certain that in many cases there is no demonstrable focus of tubercle anywhere to be found except in the injured part. It is hard to prove a negative, and it is impossible in any case to deny arbitrarily that an unrecognizable distant latent focus may exist.

We know, however, that many cases never give any sign of distant tuberculosis before the accident, and never give any sign of it afterward. We do know that bacilli can exist for a considerable time in blood or lymph when there is no demonstrable lesion of tuberculosis. From such blood or lymph bacilli may be deposited in the injured part and become active for harm.

We do know that bacilli may live for a long time in glands or bone-marrow without producing any evidence of a lesion of bone or of gland (Petrow, Lannelongue). If bone-marrow or gland containing bacilli is injured, these bacilli become active and establish an area of disease. Without the injury it is improbable that there would have been disease.

Local tuberculosis follows slight rather than severe injury. I have seen it after a strain of an epiphysis, never after a fracture. I have seen it after a sprain of a joint, never after a dislocation. After a severe injury tissue reaction is so marked that bacilli are destroyed. It is particularly in bone and joint tuberculosis that traumatism is held to be causal. Whitman tells us that out of nearly 3400 cases occurring in the clinics of Bruns, Koenig, Mikulicz, and Hildebrand over 500 were attributed to trauma. I have seen a number of such cases, most of them involving the knee, foot, or wrist. I have seen tuberculosis of the glands of the groin arise after a bruise, tuberculous pleuritis and tuberculosis of the chest-wall follow a chest contusion, and sacro-iliac tuberculosis follow a sprain. Tuberculous meningitis has followed head injury. I have seen several cases of tuberculosis of the testicle after contusion. When injury is followed by a tuberculous lesion, the definite signs and symptoms of that lesion do not appear for from three to six weeks after the accident. We may conclude that tuberculosis may arise at the seat of an injury; that in some cases there may have been an antecedent lesion at that point; in some cases there is a distant active lesion; in some cases a distant latent lesion; in some the bacilli must have been lying inactive in the part or must have lodged there from blood or lymph, after the accident and because of it. R. L. Dixon reported a number of such cases ("Physician and Surgeon," Jan., 1909). Hueter and Schüller insist on the tendency of trauma to localize tuberculosis. Ribera Y Sans (quoted in "Practical Medicine Series," vol. in Surgery, 1912, edited by John B. Murphy) states that in 45 per cent. of cases of tuberculosis of the larger joints the condition is preceded by trauma. In an appended note by Murphy we learn that that surgeon believes that tubercle bacilli tend to escape from the circulation at the site of a slight trauma and to light up disease. Bosanquet ("Lancet," Jan. 13, 1912) says that in expressing an opinion "as to the probability of a tubercular lesion being due to a preceding accident, I think we must put disease of joints in a class by itself. There is a considerable mass of evidence, that in some way or other injury does lead to tubercular arthritis, and if the occurrence of the accident is clearly established and it is followed at a reasonable interval by the tuberculosis, we may accept the causal connection as probable." He

regards "a reasonable period of time" as not over three months. During the interval there should have been some pain in or stiffness of the joint.

**The Term "Scrofula."**—Many surgeons positively oppose the use of the term scrofula, but I believe that there is clinical value in retaining it. The surgeons that have entirely abandoned it think that, after all, it is exactly synonymous with tuberculosis. I use it to designate the persons that are predisposed to tuberculosis through possessing a type of tissue of low resisting power. These tissues fall a ready prey to the bacteria of tuberculosis. Such tissue vulnerability is usually hereditary; and, as a rule, one, or even both parents are tuberculous, are in ill health, or are themselves predisposed. Occasionally this type of tissue is acquired, a child having at first been apparently entirely healthy, and later, owing to poor food, insufficient air, and bad hygienic surroundings, developing scrofula.

That scrofula is not simply osseous, articular, or glandular tuberculosis is proved by the fact that a person that we recognize as scrofulous may never throughout his life develop a recognizable tuberculous lesion. Some surgeons think that scrofula is latent tuberculosis, and will, under the influence of an exciting cause, burst into activity. This is possible, but unproved. We do know that some so-called scrofulous lesions are not tuberculous; for instance, facial eczema, corneal ulceration, granular lids, and mucous catarrh. These lesions are rather expressive of poor health, improper food, and deprivation of fresh air.

The subjects of scrofula, besides being prone to the non-tuberculous lesions above mentioned, are particularly prone to develop tuberculous lesions; and such a lesion may arise in any part that has been the seat of a slight injury or of a non-tuberculous inflammation. The parts most apt to become tuberculous are the bones, the joints, and the glands.

There are two types of the so-called scrofulous, that is, two types of those that are predisposed. The common type is known as the *phlegmatic*, or *lymphatic;* and it is this type that is particularly described by our surgical forefathers. In the phlegmatic type the individual is stolid of expression, and has thick, coarse skin, a muddy complexion, dark, coarse hair, a thick neck, thick lips, a thick nose, and a heavy lumbering gait. He is dull of apprehension, with feeble emotional reaction, and but little capacity for concentration or interest. The other type is much more seldom met with. It is what is called the *sanguine type*, or what the elder Gross spoke of as the *angelic type*. Such a child is frequently beautiful and graceful in its movements. Its skin is transparent and clear, and the color comes and goes. The eyes are blue, the lashes long, and the hair silky. The tendency is to thinness, rather than fat; the mind is not dull, but precocious, and the temperament is nervous. In both these types of scrofula the condition of lymphatism exists.

**Lymphatism, or the Lymphatic Constitution (Status Lymphaticus).**—The term was introduced by Potain to designate a condition in childhood in which there is a very strong disposition to the development of disease of the lymphatic structures, or in which at birth there was excessive development of these structures. As a matter of fact, the condition, though most common in infancy and childhood, can exist in adults. The enlarged glands may be tuberculous from the beginning; but, as a rule, they are not so in the beginning, but tend to become so. Inflammation of a mucous membrane is followed by enlargement of the anatomically related lymphatic glands. These enlarged glands are frequently met with in the neck. We find them associated with enlarged tonsils and pharyngeal adenoids.

Usually lymphatism is congenital, but it may be acquired when children are placed under unfavorable conditions. Lymphatic children frequently have rickets and are invariably anemic, yet there is considerable or much sub-

cutaneous fat (Escherich). In infancy it is the bronchial and mesenteric glands that are particularly apt to enlarge; in childhood, it is the glands of the neck. Usually the tonsils are enlarged and the nasopharynx contains adenoids. The spleen is usually palpable. In lymphatic children it is not uncommon to have a persistent and hypertrophied thymus gland. The gland, however, is not obvious, but rather hides at the root of the neck (Humphrey, in "Lancet," Dec. 26, 1908), and may be missed at necropsy. During life the thymus is sometimes observed as a pulsating mass at the root of the neck. Even if it cannot be seen its presence can usually be determined by percussion and by the *x*-rays (Hochsinger) (see page 1247). In about half the cases a goiter is obvious, and, as pointed out by Berg, many of those with goiter have symptoms of Graves's disease. As the child increases in age, the lymphatic enlargements are likely to disappear unless tuberculous infection has occurred. After a child has reached the age of seven or eight years non-tuberculous glands of the neck cease to enlarge, and by the time of puberty they have usually disappeared. Buxton ("Lancet," Aug. 6, 1910) states that young persons suffering from this condition are tall and thin; possess clear, fair, and pale skins; the temperament is blended, there is mental slowness yet intelligence, there is shyness and strong self-feeling. They cannot withstand cold, are very emotional, and yet may be able to appear calm. The pulse is normally 50 to 60, but is made rapid and irregular by trifling excitement. I do not believe that the condition can be certainly diagnosticated during life. McCardie regards hypertrophied lingual follicles, and the existence of lymphoid masses in the wall of the pharynx, the pyriform sinus, and in the anterior surface of the epiglottis as significant of the existence of lymphatism.

If an operation is performed on the victim of lymphatism the wound is very liable to become infected, and the bleeding from the wound is very trivial. The victims of lymphatism are more apt than other persons to die under a general anesthetic, and occasionally one of them dies during natural sleep. (See Dr. Geo. Blumer, in the "Bulletin of the Johns Hopkins Hospital," Oct., 1903.) Cases have died from injection of diphtheria antitoxin (see page 46). This disease accounts for most otherwise inexplicable cases of sudden death in children and young persons, and such deaths are respiratory and not cardiac (Humphrey, in "Lancet," Dec. 26, 1908).

**The Diagnosis of Tuberculosis.**—Whenever he sees a persistent area of chronic inflammation in any structure of the body the surgeon must think of the possibility of its being tuberculous. A thorough investigation must be made into the local disease and the body generally; and it is of particular importance to determine whether there is any other diseased locality, and whether there is any evidence of tuberculous disease anywhere in the body. The patient's history must be investigated, and any possible tendencies or predispositions inquired into. Tuberculosis does not cause leukocytosis except, perhaps, occasionally and moderately in tuberculosis of serous membrane, and even in this condition there is no increase in polymorphonuclear cells. A mixed infection causes only a trivial increase in polynuclear leukocytes.

In many cases of tuberculosis the diagnosis can be made from purely clinical investigation. This is the case, for instance, in many tuberculous ulcers, abscesses, and glands. In some cases the diagnosis can be made only by making differential stains of material obtained from the suspected focus, or by removing a section of the inflammatory area by Mixter's cannula, and studying it carefully under the microscope. Cultures may be taken from any material obtained from the suspected focus.

In doubtful cases animal inoculation is necessary to make a diagnosis. The material is injected into a guinea-pig; and if it be tuberculous, the animal will develop miliary tuberculosis within a few weeks. With apparently sterile

fluid obtained from a tuberculous focus the disease can be induced in guinea-pigs by inoculation. Blistering a tuberculous person causes elevated temperature (see page 221). If the fluid of the blister be injected into a tuberculous animal a distinct reaction occurs (see page 221).

In a suspected case of tuberculous meningitis of the brain or of tuberculous disease of the membranes of the cord, the theca of the cord should be tapped (lumbar puncture), and the fluid obtained should be carefully examined. Of course, if, in a case of tuberculous cerebral meningitis, the foramina in the floor of the fourth ventricle have been blocked by exudate, no characteristic fluid will be obtained by tapping. It is usually found, however, that even in tuberculous cerebral meningitis there is increased tension of the fluid in the subarachnoid space of the cord, that this fluid is present in unnaturally large quantity, and that it is turbid through the presence of pus and lymphocytes. Sometimes it contains bits of fibrin and sometimes blood; and in many cases the bacilli of tuberculosis. Exploratory abdominal incision is sometimes necessary to determine the existence of tuberculous peritonitis.

The *x*-rays are of great aid in making a diagnosis of osseous, articular, and, perhaps, certain forms of pulmonary tuberculosis. The area of tuberculosis is lighter than the surrounding healthy structures when seen in a skiagraph.

**The Tuberculin Test.**—This test may sometimes be used to advantage in making an early diagnosis of recent lesions. Some physicians will not use it, believing that it is very dangerous. However tuberculin is used, it is much more reliable diagnostically in children than in adults. Apparently healthy infants never react. Apparently healthy children under six or seven years of age seldom react. In many adults free from demonstrable signs of tuberculosis, tuberculin gives a distinct reaction because many adults have encapsuled, quiescent, or retrogressive lesions of tuberculosis. I have already expressed the belief that if given in moderate doses it is safe; that is, it is safe if the disease is not too far advanced. Very large doses, or the giving of the remedy at all in greatly advanced tuberculosis, would not be safe. Some conditions contra-indicate its use, among them are the following: Addison's disease, recent pulmonary hemorrhage, and suspected bilateral renal tuberculosis (Howes and Floyd, in "Publications of Mass. Gen. Hospital," 1908). A contra-indication is the finding of cocci in the sputum. It should only be given when other diagnostic methods fail to give certain information, and is only to be used by a man trained in its use. Too large a dose may cause a severe chill, high fever, and great exhaustion, may arouse a latent focus to activity, and may actually cause dissemination of the disease.

The elements of the reaction following a tuberculin injection are:

1. Constitutional (fever, etc.).
2. Local (redness or nodule at the point of puncture).
3. Focal (inflammation at the seat of lesion).

If fever exists we never seek to obtain the constitutional reaction by an injection of tuberculin. The result would be misleading. We never inject tuberculin when there is mixed infection.

A temperature of 99.5° F. or over, when the patient is quiet in bed, contra-indicates the employment of the test. The test should be used as directed by John B. Howes and Cleveland Floyd (Ibid.). These rules are as follows: Koch's old tuberculin is used and the preparation must not be over two months old. One c.c. (100 mg.) of the material is drawn up in a pipet and is dropped into 10 c.c. of a ½ per cent. solution of carbolic acid. Each cubic centimeter of this solution contains 10 mg. of tuberculin; 1 c.c. of solution No. 1 is mixed with 9 c.c. of a ½ per cent. solution of carbolic acid. Each cubic centimeter of solution No. 2 contains 1 mg. of tuberculin.

One c.c. of solution No. 2 is mixed with 9 c.c. of the very dilute carbolic solution. Each cubic centimeter of solution No. 3 contains .1 mg. of tuberculin.

The patient is kept in bed for three days before beginning the test, and also during the test, and during all of this time the temperature is taken every two hours. The injection is to be made at an indifferent point. The first dose is .1 mg. of tuberculin (1 c.c. of solution No. 3). If there is no reaction, wait for three days and then give 1 mg. (1 c.c. of solution No. 2). If No. 2 gives no reaction, wait three days and give 10 mg. (1 c.c. of No. 1 solution); if No. 2 gives a slight reaction, inject 5 mg. ($\frac{1}{2}$ c.c. of No. 1 solution).

We have previously described the tuberculin reaction; that is, the temporary local congestion or inflammation in the tuberculous area, and the chilly sensation or chill, followed by marked elevation of temperature (see page 221). The constitutional signs of reaction are chilly sensations or chills, sweats, skin eruptions, headache, pain in the back and joints, diarrhea, nausea, malaise, cardiac palpitation, and dyspnea. Howes and Floyd regard even $\frac{1}{2}^{\circ}$ F. of fever as significant of reaction if there are also constitutional symptoms and local signs. The focal reaction is the most important. In certain tuberculous lesions we can see the focal reaction; for instance, in lupus. In lupus the diseased skin begins to swell and redden a few hours after the injection. The reddened tissue may actually necrose. The ulcerated area becomes crusted. The swelling and redness disappear in a few days. In joint tuberculosis the skin over the joint becomes red. In a tuberculous ulcer of the mouth we can see the changes; and in a lesion of the larynx the laryngologist can observe them with the laryngoscope. By means of a cystoscope the local reaction can be seen in a tuberculous ulcer of the bladder.

Epstein in 1891 pointed out that redness and swelling at the seat of injection constitute a specific reaction.

The tuberculin test should not be used in advanced pulmonary tuberculosis because it is unsafe. In advanced cases it fails to cause any reaction because the tissues are unable to produce antibodies when acted on by toxin (Howes and Floyd, in "Publications of the Mass. General Hosp.," 1908). As a matter of fact, there is never any need of using the test in an advanced case, because the diagnosis is perfectly clear without it. We should never give extremely large doses in making the tuberculin test. If, after the careful use of tuberculin, there is no reaction, it is usually a safe conclusion that there is no tuberculosis. The tendency is more and more to use as a diagnostic test the local rather than the constitutional reaction.

Various methods have been devised for obtaining a local reaction (ophthalmo-tuberculin reaction, von Pirquet's reaction, Moro's reaction). The local reaction is obtained without danger of dissemination of infection and can be used even if fever exists.

**Calmette's Ophthalmo-tuberculin Reaction.**—It was pointed out that when tuberculin is injected into a tuberculous individual a reaction arises at the seat of injection. It has been shown that if tuberculin is placed in the conjunctival sac of a tuberculous individual a reaction occurs, and this method is valuable because even a trivial reaction is easily observed. The introduction of tuberculin into the conjunctival sac is usually spoken of as Calmette's method. This test can be used even if fever exists and even if a skin eruption exists. It is not as satisfactory in surgical as in medical cases. The old tuberculin of Koch is used. It is carefully freed from irritant materials, a 1 per cent. solution is made in normal salt solution, and 1 drop of this is placed in the eye. Baldwin regards 1 per cent. as dangerously strong and uses $\frac{1}{2}$ per cent. No constitutional symptoms develop, but in four or five hours, if the subject be tuberculous, the conjunctiva of the lids becomes injected, the corneal vessels distend, lacrimation arises, and the lids may swell (Howes and Floyd, Ibid.).

The reaction attains its height in from twelve to twenty-four hours and disappears in from forty-eight to seventy-two hours after its first appearance. In a non-tuberculous person no redness, or only a trivial and temporary redness, is noted. There is never a constitutional reaction even in a case of advanced tuberculosis. Of course, this test is contra-indicated if there is a tuberculous lesion of the lids or eye, if there is ulceration of the cornea, or if conjunctivitis exists. I have never become convinced that the method is entirely free from danger to the eye, and I own that I rather fear to use it. Cases of permanent ocular injury are on record. Baldwin ("Jour. Am. Med. Assoc.," February 20, 1909) made over 1000 tests. He says that the test has some value in diagnosis, no value in prognosis, and as yet cannot distinguish "active latent" from healed tuberculosis. He says danger to the eye is slight.

**Von Pirquet's Cutaneous Tuberculin Reaction.**—After the skin has been cleansed with alcohol 2 drops of old tuberculin are applied a short distance apart. The skin is then abraded or scarified between the drops and through each drop. The abrasion between the drop is a control experiment. The tuberculin is permitted to remain for ten minutes and is then wiped off. In a tuberculous individual local redness will appear in ten or twelve hours, and in twelve hours more the area will be swollen and perhaps edematous. This condition disappears in a few days, leaving, perhaps, as a legacy a trivial induration. There is very seldom a febrile reaction.

**Moro's Cutaneous Tuberculin Reaction.**—The material used is 5 c.c. of old tuberculin and 5 gm. of lanolin. It is rubbed into the abdominal skin and if the individual is tuberculous red papules or nodules or numerous vesicles appear in the area where the inunction was made. A severe reaction or a moderate reaction will be noted within twenty-four hours. A slight reaction appears in from twenty-four to forty-eight hours. The eruption of a slight or a moderate reaction disappears in a few days. After a severe reaction the skin may remain red for several weeks. In suspected surgical tuberculosis Moro's reaction is generally used. There is no febrile reaction.

**Blistering a Tuberculous Person.**—(See page 221.)

**Injecting a Tuberculous Animal with Blister Fluid from a Person Suspected to be Tuberculous.**—(See page 221.)

**Massage of a Tuberculous Focus.**—Wright has shown that gentle massage of a tuberculous focus may be followed by a reaction like that which follows the diagnostic use of tuberculin. In such a case the massage drives tuberculous products into the blood and, perhaps, if the massage is frequently repeated, auto-immunizes the individual.

**Animal Inoculations.**—This method of diagnosis is seldom employed and only in unusually obscure cases.

**The Agglutination Test.**—This test, as applied to the blood-serum of a tuberculous individual, is decidedly uncertain.

**Prognosis.**—Many cases of tuberculosis are cured. This is indicated by the frequency with which we find healed tuberculous lesions in necropsies on individuals dead of other diseases. We reach the same conclusion from the clinical study of many cases. The prognosis of a single tuberculous focus, especially if it can be extirpated or sterilized, is very good; provided that the general health is good, that there is not much anemia, that the digestive processes are well performed, that mixed infection is absent, that there are no albuminoid changes in the viscera, and that the patient is able and willing to live the life that is necessary for his welfare. Unfavorable prognostic indications are inability to eat, disturbance of digestion, deepening anemia, progressive loss of weight, high fever, and sweats. Of course, the prognosis is influenced by the patient's temperament, his willingness to brook control, his monetary status, and his habits. The danger is greatly increased by multiple lesions.

The dangers of mixed infection and of albuminoid disease have been previously discussed.

In very young children the prognosis is most unfavorable; but in older children it is very much better; in fact, it is better in them than in adults.

Tuberculosis of the skin gives a very fair prognosis; and glandular, bony, and articular tuberculosis are frequently recovered from; but, of course, any tuberculous lesion, however limited in area, is a profound menace.

Another fact to be borne in mind is that many cases apparently cured are not really cured; and that the disease strongly tends to reappear in the same region or in a nearby region, or to reappear later in another part of the body. We should, further, remember that in many cases in which there is apparently one lesion only, there are, in reality, distant lesions undiscoverable by clinical methods. In any case of tuberculosis the higher the opsonic index the better the prognosis, the lower the opsonic index the worse the prognosis.

Another important fact is that when an individual has a latent focus of tuberculosis, especially if this latent focus is in the lungs, should a surgical operation be performed for some other purpose, and the patient be kept in bed for a considerable length of time, the latent focus may become active. I have always believed that in latent pulmonary tuberculosis the administration of ether or chloroform might awaken the disease into activity. It therefore becomes evident that in such persons operations of necessity are the only ones that should be undertaken. Such an operation, if possible, should be done under nitrous oxid or a local anesthetic; and the patient should be got about again at the earliest possible moment.

**Tuberculin in Prognosis.**—Wolff-Eisner maintains that advanced and rapidly advancing cases fail to show an ophthalmo-tuberculin reaction, hence, when the existence of tuberculosis is proved clinically, a negative ophthalmo-tuberculin reaction indicates a bad prognosis. Most observers reject this contention. "Of two individuals with moderately advanced disease and in equally good general condition, tuberculin cannot predict with more assurance than other clinical methods the state of affairs a year hence" ("Tuberculin," by Hamman and Wolman).

**Treatment.**—One of the first thoughts of the surgeon is to provide against the contamination of healthy individuals by the infected. Any infected excretion or suspicious discharge from the patient must be disinfected at once and dressings that are removed from the patient should be burned.

We are not in this section discussing the treatment of tuberculosis of the lungs, which belongs to the medical man, and in which climate is of great importance. In cases of surgical tuberculosis, however, the patient may do better in some climates than in others; and the change, by stimulating the appetite and causing sleep and giving renewed hope, will be beneficial. In surgical tuberculosis climate is not the factor that it is in tuberculosis of the lungs; but if there is pure atmosphere, an equable temperature, and plenty of sunlight, the climate will lure the patient out-of-doors, and will thus be greatly to his advantage.

A life in the open air is the most essential thing in the treatment of surgical tuberculosis; but, as Professor Halsted points out, it is not of much use to tell a great many persons to live in the fresh air. They will not do it unless they are made to; and it is hard to make them unless they live in quarters especially built with this object in view. Therefore, other things being equal, if patients with surgical tuberculosis have the means, it is a good plan to send them to a sanatorium in the mountains or at the seashore, where they can obtain the persistent, unbroken life in the open air that is the cure of the disease. The patient should spend his days in the fresh air, and he should sleep at night directly exposed to the air; and if the atmosphere is free from dust and foul

odors, so much the better. The poorer patients must get the fresh air at home, if they cannot be sent to some camp or colony. In large cities adjacent to the seaside resorts poor people can usually be sent for a short time, at least, to the seaside; and I am a very great believer in the beneficial effects of Atlantic City and other seashore resorts.

It is frequently necessary to do an operation in a great city, although we operate much less than formerly for these conditions. If an operation is done in a great city, the patient is kept in the fresh air as much as possible during his convalescence. If it is feasible, he is sent away to a colony or sanatorium to recuperate. It would be an excellent thing if, in many of those cases in which operation is necessary, the operation could be performed at the camp or the sanatorium. One advantage of the camp or sanatorium is that the patient is watched and regulated daily, and is led to do things that otherwise he would neglect. Many patients endeavor to avoid going out when they should go out because they are afraid of taking cold; and many of them are simply neglectful and do not want to take the trouble to do it.

It cannot be soo strongly insisted on that in surgical tuberculosis fresh air is of as much importance as in tuberculosis of the lungs. It increases the vital resistance, it stimulates opsonic power, and it causes the patient to eat more nourishing food and to sleep better at night. Frequently we see children that have had sinuses for months get rapidly well when they adopt an open-air life; and, although albuminoid changes, when they once exist, will never pass away, further albuminoid changes may not take place if the patient lives properly.

A patient with surgical tuberculosis can have no more injurious environment than a dark, damp room, especially if it is in a crowded tenement and up a narrow court. The value of sunshine is also beginning to be appreciated (*heliotherapy*). We know that it limits the growth of tubercle bacilli. It is not the heat that benefits the person, but the chemical rays of sunlight. These rays have some germicidal influence, have considerable penetrating power, and seem to influence decidedly the nutritive processes. Tuberculous joints, even when sinuses exist, are often much benefited by exposure to the direct rays of the sun. It is often advisable to expose the entire body except the head. Leysin begins with a three-minute exposure and gradually increases it up to two or three hours. During exposure traction is usually maintained on the joint. Excessive sunlight is, however, not beneficial. In summer it exhausts the patient and even in winter it produces eye-strain and headache. Major Woodruff, U. S. A., insists that excessive sunlight is actually harmful, particularly to blondes ("The Effects of Tropical Light on White Men"). Tuberculosis is dreadfully fatal in certain tropical countries and is more fatal to blondes than brunettes. Open-air treatment is more valuable in winter than in summer, perhaps because cold stimulates respiration and because the winter sunlight is not debilitating.

The tuberculous structures require rest. We have long known how disastrous it is to confine a person to bed in a dark, ill-lighted, and improperly ventilated room. We can, however, confine a person to bed with perfect safety if there is a free flow of fresh air. We must confine certain cases to bed; for instance, cases of tuberculous peritonitis, and some cases of bone tuberculosis and of joint tuberculosis. A patient with tuberculosis who has fever ought to be in bed. We can put such patients to bed without any fear of the disease becoming worse or spreading if the supply of fresh air is plentiful and if the patient is kept warmly covered and wears a skull-cap. Of course, a draft is to be avoided. Patients that are confined to bed do excellently in a tent, in a cottage sanatorium, or on a porch that has been altered for the purpose.

At the very first possible moment the patient should be sent out-of-doors;

and in many cases of tuberculous disease (for instance, vertebral disease) the tuberculous part is supported by means of a brace or a splint.

We thus see the twofold nature of the modern treatment of surgical tuberculosis: rest for the tuberculous part and a life in the open air. Exercise is of importance also, although it should never be taken in excess. If the patient is confined to bed, he should be massaged and rubbed with alcohol, the tuberculous part being usually avoided. Forcible manipulation must never be applied to a focus of tuberculosis because it may lead to dissemination. Gentle massage of a tuberculous part may do good. Wright has shown that it is followed by a reaction like that produced by tuberculin—a reaction due to the absorption of tuberculin from the seat of disease. If a person has fever he must not attempt active exercise, but must be confined to bed.

One should overfeed tuberculous patients if the stomach tolerates it, but not on any single article, or even on any particular one. The diet should contain a sufficiency of fats, proteins, and carbohydrates; and the food should be agreeable to the taste and readily assimilable. Otherwise, disgust will be engendered; and with disgust come indigestion and loss of appetite. The very life of the patient may depend on his remaining able to take a sufficiency of nourishing food.

There is no specific diet for tuberculosis, although many have been suggested. One of the most valuable foods is milk, taken raw or mixed with other articles, such as lime-water or sodium bicarbonate, and frequently with brandy. The use of an exclusive diet of boiled milk is to be deprecated, and in children it sometimes leads to the development of scurvy. Practically anyone can take milk if proper efforts are made.

Soft-boiled eggs are useful; and bread or toast should be eaten with plenty of butter, which is an agreeable form of fat. Vegetables and fruits are desirable.

If the patient can take cod-liver oil without impairing his appetite or digestion, it should be given, provided the weather is not too hot. Cod-liver oil produces diarrhea in very hot weather. Children learn to take it very well. To many adults, however, it is, and remains, absolutely abhorrent. The chief value of cod-liver oil is that it is a fat, and it seems improbable that it contains any elements specifically antagonistic to tubercle. If used, large doses should not be given, as they will not be digested. The common dose for an adult is a teaspoonful two or thee hours after meals. Thirty drops three times a day is usually given a child, and an infant should receive 15 drops three times a day.

We know of no drug or medicine that can with safety be used at the present time with any real hope that it will specially destroy tubercle. Drugs are, of course, given, but they are of secondary importance.

Tonics are used, and in children the syrup of the iodid of iron has considerable reputation. Remedies may be needed to improve digestion or control night-sweats, etc. I do not believe that beechwood creosote or carbonate of guaiacol internally, or iodoform inunctions, or painting the surface with guaiacol confer any real benefit in tuberculosis.

Alcohol is often required. It is not needed in all cases, but is in many. We should avoid it in children, however, unless there is a particular indication for its use. When a tuberculous patient is weak, milk-punch or egg-nog is of service; and in any case of mixed infection alcohol is required in full doses. If fever exists, and the administration of alcohol makes the pulse more rapid and the delirium worse, and causes flushing of the face, the dose is too large and should be diminished. Any patient that smells strongly of alcohol is getting an overdose.

**Tuberculin in Treatment.**—Many able investigators in many lands are striving to work out a safe and satisfactory specific treatment for tuberculosis.

Landerer proved that immunity to tuberculosis can be produced in the lower animals by injection of living bacilli. We dare not practice this on human beings. Injections of tuberculin finally produce immunity to that product. The original plan of using tuberculin therapeutically was to obtain definite reactions again and again. This plan was founded on the belief that the reaction did the good and that cure might be obtained in this way in a few weeks. This utterly reckless plan was most disastrous, produced many deaths, and caused widespread distrust and final abandonment of tuberculin. Tuberculin treatment was not an error, the plan adopted was, as the material was given in large doses to any and all tuberculous patients. Just at present we are witnessing a revival of faith in tuberculin. The object now is to stop short of obvious reaction, believing that reaction is unsafe, as it may at any time get beyond control and do harm. Treatment is begun with very small doses which produce no reaction. Larger and larger doses are very gradually attained until immunity to tuberculin is established.

When an animal becomes immune to tuberculin the body cells resist and finally destroy the tubercle bacilli (Braun, in "Boston Med. and Surg. Jour.," July 23, 1908). From six months to a year is required for the treatment. The essence of treatment is the very small dose and the very gradual increase, reaction being scrupulously avoided.

Many different tuberculins have been recommended. The best known ones are Koch's old tuberculin (O. T.), Koch's new tuberculin (T. R.), Koch's bacillary emulsion (B. E.), the bouillon filtrate of Denys (B. F.), von Ruck's watery extract, and bovine tuberculins. There seems to be no sound clinical reason for insisting on the use of any particular form of tuberculin. Bovine tuberculin does not seem more useful than human tuberculin in tuberculosis of the abdomen, glands, and liver, structures so often the seat of infection with bovine bacilli. Whatever form is chosen is given subcutaneously in the back and near to the skin, so that a local reaction may be quickly recognized. If after any dose there is even a slight elevation of temperature or even a trivial local reaction the dose must not be advanced until that dose can be given without reaction. Loss of weight means that doses are too large. Judiciously small doses are entirely safe even when tuberculosis is complicated. One or two injections are given each week until the maximum dose without reaction is attained. The maximum dose when attained may be given at weekly intervals for months. Another plan is to reach the maximum dose, stop the treatment for months, and then start it again, beginning with the smallest dose.

The initial dose and the maximum dose of certain tuberculins are given in the following table taken from the valuable work of Hamman and Wolman on "Tuberculin." The dose is expressed in cubic centimeters instead of grams:

| Tuberculin. | Initial Dose. | Maximum Dose. |
|---|---|---|
| O. T. | 0.000,000,1 to 0.000,001 c.c. | 1 c.c. |
| T. R. | 0.000,001 to 0.000,1 c.c. | 2 c.c. |
| B. E. | 0.000,001 to 0.000,1 c.c. | 2 c.c. |
| B. F. | 0.000,000,01 to 0.000,000,1 c.c. | 1 c.c. |
| Béraneck's | Of A/32, 0.05 c.c. | Of H 1 c.c. |

Wright gives very small doses of emulsion of powdered bacilli, not with the idea of causing directly body immunity to tuberculin, but to produce immunity by strengthening the phagocytic power of the leukocytes.

When this plan is followed the dose is determined by the opsonic index, and the dose is only raised to a sufficient degree to establish the positive phase

without producing even the most trivial subjective symptoms. One dose causes an increase of immunizing power in the body for about a fortnight and then another dose is given. Many clinicians deny positively the need of giving the dose by the opsonic index. When tuberculin is given by either of the above plans it is entirely safe, and I believe, beyond doubt, is of value in suitable cases. It is not to be used in advanced cases or in febrile conditions. For lupus, tuberculous glands, tuberculous bones, or tuberculous joints one dose a week is given. It is of real service in these conditions, but general treatment must not be discontinued because tuberculin treatment is employed. It may be used after operation to prevent recurrence. When given in this way it is safe, never produces trouble at the site of injection, seems to arrest some cases of tuberculosis, improves the local trouble in many, and benefits the general conditions of most. According to Trudeau tuberculin strengthens the individual's immunity to tuberculosis. According to Wright it causes an increase in deficient opsonins. Tuberculin is of unquestionable value in some cases of lupus.

Ringer has recently strongly advocated the use of tuberculin ("Jour. Am. Med. Assoc.," May 2, 1908). Maragliano treats tuberculosis with the serum of animals which have been injected with dead bacilli and toxins, and he believes that this serum contains quantities of antibodies and antitoxins. The animal is injected many times until its serum becomes highly agglutinative. Maragliano believes that this highly agglutinative serum when introduced into the human body causes the protective mechanism of the body to produce quantities of antibodies and antitoxins. Most clinicians do not favor the use of Maragliano's serum.

**The Local Treatment of Tuberculosis.**—When certain drugs are directly inserted into a tuberculous focus they possess an antagonistic influence. Iodoform is the most powerful of these drugs; guaiacol, balsam of Peru (Landerer), bismuth, and chlorid of zinc (Lannelongue) have a similar action. Iodoform has little or no influence when placed on a free surface exposed to the air, but when in the form of an emulsion it is injected into a tuberculous area, the air being excluded (see page 30), this drug is powerfully antituberculous. Chlorid of zinc seems to act by causing the development of quantities of fibrous tissue, which encapsulates or, perhaps, replaces the tuberculous focus. Some surgeons inject tuberculous nodules with camphorated naphtol. Every region of tuberculosis requires local rest, perhaps by the use of a splint or a brace.

**Special Methods of Surgical Treatment.**—The surgeon may endeavor to extirpate a tuberculous focus, or to drain it thoroughly and to sterilize the area. Extirpation is sometimes, although not very frequently, possible. Complete extirpation is a valuable method, but partial extirpation is dangerous. If a part only of a tuberculous focus is extirpated, many lymph-tracts and blood-vessels are opened; and the incomplete operation may lead to the dissemination of the disease. The methods of surgical treatment suited to different forms of tuberculous disease will be discussed in different sections of this book.

*Bier's Method by Congestive Hyperemia* (see page 112).—Bier believes that passive hyperemia is of the greatest possible benefit. Active hyperemia is obtained by heat, and is especially valuable to induce the absorption of the products of a non-tuberculous chronic inflammation. Passive hyperemia is particularly useful in tuberculosis of joints, tuberculous ulcers, cold abscesses, and tuberculous disease of the tarsus, carpus, and phalanges. If a limb is affected, passive hyperemia is obtained by placing a rubber band around the limb above the part, the band being applied with sufficient firmness to interfere with venous return, but not so tightly as to block arterial entry. This band should be applied daily, and should be kept in place for an hour or so at each application, but pain should not be produced. In the intervals between

the treatments the limb should be at rest. Bier uses special apparatuses for obtaining congestive hyperemia in various parts of the body.

I have seen cure or very great improvement follow this treatment in a number of cases. It is founded on the old idea of Laennec that cyanosis and tubercle are antagonistic. Why this method is beneficial is much debated. Some think that the imprisoned blood takes on increased bactericidal power; some, that the number of leukocytes is greatly increased; some, that quantities of leukocytes migrate; and some, that the amount of bactericidal blood-serum is increased. Bier believes that it depends upon phagocytosis. It would seem possible that the cells in this locality, under the influence of the congestive hyperemia, may form powerful antitoxins.

*Heliotherapy.*—(See page 231.)

*The Finsen Light.*—Finsen pointed out that the chemical rays in sunlight are powerfully germicidal, and that this germicidal power can be notably increased if the rays are concentrated on a part by the use of particular apparatus. He also showed that enormous numbers of chemical rays can be obtained from electric light. The Finsen treatment to-day consists in applying the actinic rays obtained from electric light. They act most powerfully on lupus, but require a very long time to effect a cure.

The *x-rays* are of value in treating certain tuberculous conditions. They are of most use in lupus, their effects in this disease being nearly as powerfully curative as those of the Finsen light, and much more rapid.

The **tuberculous abscess** is called also the *cold*, the *lymphatic*, the *congestive*, the *scrofulous*, the *strumous*, the *wandering*, or the *migrating abscess;* and it is very commonly called the *chronic abscess*. The Germans call it *Senkungsabscess*. Tuberculous abscess is the best designation, as this indicates the cause of the trouble.

The term "cold abscess" is often used because the cutaneous surface over the disease is not warmer to the touch than is the skin of the corresponding part of the opposite side of the body. The term "lymphatic abscess" was employed because it was once thought that such abscesses arose only from lymphatic structures. Scrofulous abscess was the name given it when scrofula was supposed to be a definite disease, the common phase of which was this form of abscess. The term "chronic abscess" is employed because the condition usually develops slowly, and does not present the evidences of acute inflammation; an acute pyogenic abscess developing, as a rule, rapidly, and presenting positive signs of inflammation. I agree with the late Professor Ashhurst that the term "chronic," in this connection, is improper, as it tends to give a wrong idea. It refers merely to time; and we know that a genuine pyogenic abscess that is deep seated may be rather slow in developing, and that a tuberculous abscess that is superficial may develop with considerable rapidity. When used properly, the term "chronic abscess" means that real pus exists, this pus having arisen from the pyogenic infection of the granulation tissue of a lesion of syphilis, tuberculosis, or actinomycosis. In other words, a genuine chronic abscess is secondary pyogenic infection of an infective granuloma. The terms "wandering," "migrating," "gravitating," and "congestive" have been used because the fluid products of a tuberculous inflammation are liable to wander a considerable distance away from the primary focus of disease. For instance, a tuberculous abscess that is discovered in the groin may have arisen from tuberculous caries of the vertebræ. This tendency to wander is not due to gravity, as one of the names of the condition would suggest; but the wandering always takes place in the line of least resistance.

It will be seen from the foregoing that a true tuberculous abscess is not an abscess at all, because it does not contain genuine pus. It is a collection of the degenerated products of tuberculous inflammation; and a tuberculous abscess

may be defined as a circumscribed cavity of new formation, containing the degenerated products of a tuberculous inflammation. These products may have been formed in that region or may have passed to that point from some adjacent or distant focus of tuberculous disease. If a supposed tuberculous abscess is found to contain genuine pus, there must have been mixed infection with pyogenic bacteria; and such mixed infection either causes violent and dangerous inflammation or leads to the formation of a true chronic abscess, in which there is no sign of acute inflammation. The tubercle bacillus is not pyogenic. It can produce inflammation, but not pus, and pus can be formed in a tuberculous focus only by secondary infection with pus bacteria.

**Situations of Tuberculous Abscesses.**—These abscesses are particularly apt to form as the result of tuberculous disease of bones, joints, lymph-glands, and subcutaneous connective tissue, but the brain, any viscus, or any tissue in the body may present the condition.

**Age.**—No age is exempt, but children are most prone to the trouble; and the period of greatest liability is before the age of twenty years.

**Contents.**—The usual terms for the contents are scrofulous, curdy, or caseous pus. As previously stated, it is not true pus, but it resembles pus when viewed by the naked eye. Examination of this fluid by staining methods, by cultures, and by inoculations shows that it contains no pyogenic bacteria. It consists of liquefied and caseated tubercle, masses of coagulated fibrin, and bits of necrotic tissue. The tuberculous material is whitish, yellowish, or yellowish green, thick, and without odor. Floating in this pus are portions of caseous matter, which, as the elder Gross said, resemble bits of soft-boiled rice. Occasionally the tuberculous material, especially if it comes from disease of a lymph-gland or of a bone, is almost watery and nearly colorless, and contains curd-like masses, consisting of tuberculous granulations, coagulated fibrin, and necrotic tissue. It was previously stated that tuberculous pus is free from odor. This is not true of tuberculous pus from the ischiorectal fossa, which is highly putrid; but in an ischiorectal abscess, as a matter of fact, there is usually mixed infection with pyogenic organisms, as well as with the organisms of putrefaction. If tuberculous pus is permitted to stand, the curdy mass settles to the bottom, and a thin serous fluid remains above.

**Formation of Tuberculous Abscess.**—The growth of tubercle bacilli in the tissues causes chronic inflammation. The cells of the tissues, especially the fixed cells, proliferate and form granulation tissue. This granulation tissue consists of multitudes of cell clusters, and each cluster is called a primitive tubercle (see page 214). Each individual tubercle enlarges; myriads of new ones form, and many of the old ones fuse. These new cells, however, do not become vascularized. In the earliest stage of their formation there are blood-channels, but these become closed through endothelial proliferation and through the pressure of cells external to them. The tuberculous area then becomes absolutely avascular. This avascular mass of cells is composed of what are known as epithelioid cells, and the cells obtain nourishment by imbibition. The nourishment is very incomplete. As the nodule enlarges the nourishment grows more and more insufficient. Finally, the adjacent blood-vessels that furnished the fluid for imbibition become occluded, and nourishment is no longer possible. The toxins of the tubercle bacilli, acting upon this area of greatly lowered nutritional activity, produce coagulation necrosis, and caseation follows this. The caseation begins at many points near the middle of the tuberculous nodule. Each area of caseation enlarges. Several of them fuse, and eventually many caseated areas coalesce. The tuberculous lesion may be spreading at the periphery at the same time that it is undergoing caseation at the center. The bacilli in the caseated material soon die for want of nourishment. When an area of caseated tubercle is liquefied by the addition of serum,

what we call caseous or curdy pus is produced, and the lesion is then known as a tuberculous abscess.

The **wall of the abscess** is formed by compressed or solidified tissues. In a very recent case the wall is soft and will readily collapse. In an old case it is dense or actually fibrous and will not collapse. This wall of compressed tissue is not, as used to be thought, a pyogenic membrane which secretes the tuberculous material, but it actually surrounds the tuberculous material and hinders its diffusion. As Roswell Park says, it is not a pyogenic membrane, but it is a prophylactic membrane. The inner surface of the wall of the compressed tissue is lined with tuberculous granulations, which at different points show different stages of the tuberculous lesion. This layer of tuberculous granulations is known as *Volkmann's membrane*. The fluid in the abscess may contain a few living bacteria, but often none can be found; and certainly the bacteria are not multiplying in this fluid, but they exist in numbers and multiply in Volkmann's membrane. When tuberculous matter has been long retained and is thoroughly encapsuled the bacilli soon die for want of nourishment, and because a culture from a supposed tuberculous area fails to show the bacilli of tuberculosis we have not obtained conclusive evidence that the area is not tuberculous. We know this same fact to be true of the fluid of tuberculous empyema.

From the abscess wall there may be one, two, several, or many sinuses tracking out. These sinuses are lined with granulation tissue exactly like the Volkmann's membrane in the main abscess; and they may spread by a sort of crawling progression for long distances, perhaps passing through dense fascia, and at their terminations may form secondary tuberculous abscesses. The wall of an abscess may contain expansions or loculi. If an abscess spreads to some distant place, the tuberculous infection, of course, goes with it, and it is the tuberculous infection that causes the spread. The wandering of a tuberculous abscess is in the line of least resistance, and is not the result of gravity. Injury, breaking, or contusion of this granulation tissue, if unaccompanied with the removal of all the tissue or the killing of all the germs it contains, may diffuse the infective matter and actually cause disseminated tuberculosis. We sometimes see such dissemination after spontaneous opening, non-aseptic operation, or forcible squeezing; and particularly after an imperfect operation that removes only a part of the tuberculous area.

**Terminations of Tuberculous Abscess.**—The abscess may slowly and gradually enlarge, and finally open of itself, either on the skin or on the mucous surface, or into some viscus or joint. It may become encapsuled by fibrous tissue, there being absorption of the fluid and shrinking of the entire focus, the caseous part perhaps remaining or becoming calcified. The tuberculous abscess may actually be replaced by fibrous tissue, and this constitutes a permanent cure. When the tuberculous area is merely encapsuled by fibrous tissue, some living bacilli may remain latent in the wall; and long afterward, as the result of injury or of some other damage, an abscess may re-form at the old site of disease. Sir James Paget called this condition *residual abscess*. As a rule, the abscess, as it shrinks, tends toward cure. The bacilli usually die for want of material to nourish them, but occasionally they remain latent for a long period of time. When they do die, the tuberculous granulation tissue may become healthy tissue, be vascularized through the entrance of blood-vessels, and be converted into scar-tissue. Tuberculous abscess may also be cured by a surgical operation.

**Secondary Infection of a Tuberculous Area by the Bacteria of Suppuration.**—This is liable to occur when the abscess undergoes spontaneous evacuation, and may occur when it has been opened by the surgeon. It occasionally occurs when the abscess has neither undergone spontaneous evacuation nor has

been opened by the surgeon, having been infected apparently as a point of least resistance. When such infection does occur, there is, in all probability, some area of ordinary suppuration elsewhere in the person's body, and the bacteria of suppuration have entered the body fluids. Pyogenic infection is apt to produce violent inflammation and profuse suppuration—a condition that is extremely dangerous, because septicemia is very liable to develop. In some very rare cases suppuration destroys the tuberculous area and cures the tuberculous disease. More commonly, however, it produces illness, and in large abscesses it may cause death. Because of this liability to secondary infection surgeons were long opposed to operating on tuberculous abscess unless it was evidently going to evacuate itself. In some cases secondary infection produces a true chronic abscess (see page 235). Infection by streptococci is much more dangerous than is infection by staphylococci. Acute inflammation with dangerous constitutional symptoms is particularly apt to arise if the walls of the abscess contain very little tuberculous tissue, if they have been bruised or damaged by powerful chemicals, if there is poor drainage (and there is certain to be poor drainage if loculi exist, or if the incision is small and blocked with plugs of fibrin or necrotic tissue), if a partial or imperfect operation has been performed, if a number of virulent bacteria have been introduced, or if the vital resistance is at a low ebb.

**Secondary Infection by the Bacteria of Putrefaction.**—This complication is extremely grave and may produce death. It is commonly associated with pyogenic infection. The wound fluid becomes intensely putrid, violent acute inflammation arises, and the absorption of materials from the wound induces the systemic condition known as sapremia or putrid intoxication.

**Signs and Symptoms of Tuberculous Abscess.**—A purely tuberculous abscess presents no evidence of inflammation except swelling; and, owing to the absence of heat, it has received its name of cold abscess. The cutaneous surface looks and feels normal or is paler than normal until the structures just beneath the skin or the skin itself become involved. When this happens, livid discoloration appears, but the lividity presents a very different appearance from the dusky discoloration of an acute abscess. Neither is the skin edematous or glossy as it is in acute abscess.

There is rarely tenderness in the region of the abscess, and still more rarely spontaneous pain. Pain and tenderness, although frequently absent in the area of a tuberculous abscess, may be complained of at the primary focus of disease. Tenderness is especially likely to be noted at the primary focus; and in cases of joint tuberculosis and of bone tuberculosis it is nearly always present. There may or may not be pain at the primary focus, but referred pain is frequently complained of. For instance, in tuberculous disease of the hip-joint the pain may be referred to the inner side of the knee; and severe bellyache is frequently observed in Pott's disease of the spine. At the point to which pain is referred, however, there is seldom tenderness. For instance, in the bellyache, particularly of Pott's disease of the spine, the belly is not tender, although the spine may be. In sacro-iliac tuberculosis the pain is often referred to the distribution of the sciatic nerve, but the nerve is seldom tender on pressure. In a psoas abscess we find that pain in the spine can be induced by pressing on the spinous process of the diseased vertebra, by concussion to the heels or the head when the spine is held stiff, and especially by flexion of the spine; but the spinal pain is lessened or completely abolished by extension, fixation, and rest. The primary focus of disease, if spinal or articular, produces rigidity in the adjacent muscles, and rigidity secures rest by inhibiting movement, but it also impairs the function of the part. In an intra-abdominal tuberculous abscess there is rigidity of the abdominal muscles.

In a tuberculous abscess fluctuation is usually obtained readily, because

the fluid is not surrounded by a thick mass of granulation tissue and also because a considerable amount of fluid is usually present. A notable characteristic of a tuberculous abscess is the tendency to wander, and it may appear with suddenness at some distant point. Abscesses of the spine wander long distances, but the wandering is not the effect of gravity, and is due to the disposition of the tuberculous matter to travel in the line of least resistance. The temperature of the body may be entirely normal if the infection is purely tuberculous. As a rule, however, there is a slight evening elevation; and the patient is weak and pale, grows tired readily, sleeps poorly, and has a wretched appetite and impaired digestion. The blood examination sometimes shows a notable diminution in the number of red blood-cells, and the hemoglobin is usually lowered to 60 or 70 per cent. There is no leukocytosis. In multiple tuberculous foci, and particularly in tuberculosis in children, there is a marked decrease in the red blood-cells. If secondary infection occurs, there is a rapid and progressive diminution in the number of red cells and usually a trivial increase in polynuclear leukocytes.

A tuberculous abscess underneath the deeper fascia may break through the fascia by making a small opening, and a large secondary abscess may arise in the subcutaneous tissue. The entire abscess is thus shaped like an hourglass, the opening through the fascia being the narrowest point. Such an abscess is called a *shirt-stud abscess*. A tuberculous abscess is liable to form one, several, or many sinuses, and the end of each sinus may expand into a secondary abscess. The surgeon must always make a careful examination to try to determine whether the abscess is the primary disease focus or whether the tuberculous matter has wandered from a distant point. He must also make a thorough examination to see whether anywhere in the body there are other regions of disease. He will often find such a region of disease; for instance, in the lungs. In many cases, however, there is no clinical evidence that other areas of tuberculous disease exist.

A deeply arising tuberculous abscess usually requires weeks or months to reach the overlying skin or mucous membrane and undergo spontaneous evacuation. That spontaneous evacuation is imminent is shown by livid discoloration and thinning of the skin. Finally, at the area in which the skin is thinnest a little tit is elevated. This condition is known as pointing and a rupture occurs at this point, tuberculous pus running out. Spontaneous evacuation is a peril, because it is liable to be followed by secondary pyogenic or putrefactive infection. After spontaneous evacuation has occurred, a true chronic abscess may form; but there may instead be violent acute inflammation, manifested locally by pain, heat, and dusky discoloration. If acute inflammation does arise there develops a fever, which presents evening exacerbations and morning remissions, and is accompanied by an exhausting sweat during the night or early morning. Fatal septicemia or sapremia may follow spontaneous evacuation.

**Results of a Tuberculous Abscess.**—It may undergo spontaneous cure, and the cure may be lasting, but long after an apparent cure a new abscess may form (the residual abscess of Sir James Paget). A tuberculous abscess may remain stationary for a very long time, and then perhaps diminish in size and be cured, or extend in size and rupture. After spontaneous rupture, suppuration may cure the tuberculous area by annihilating the tuberculous tissue; but, as a rule, after spontaneous rupture there is either an acute septic process or a chronic suppuration, constituting a genuine *chronic* abscess.

**Amyloid Disease.**—The pyogenic infection of an area of chronic tuberculous, especially a bony area, if it induces long-lasting suppuration, may lead to the development of *albuminoid, amyloid, waxy* or *lardaceous disease*. Pyogenic infection of a tuberculous area, though by far the commonest, is not the only cause of amyloid disease. It may arise when there has been no pyogenic infec-

tion of the tuberculous area. It may arise after prolonged ordinary suppuration. It may follow bone syphilis, and may be due to cancer, Bright's disease, malaria, chronic dysentery, or prolonged lactation. In this condition a peculiar material is deposited in the middle coat of the smaller arteries and later the inner coat is involved. The albuminoid substance resembles fibrin and there has been much dispute as to its nature. One theory is that this deposit takes place from blood-serum which has been dealkalinized because the flow of pus has removed potash salts from the blood. Krakow seems to have demonstrated that the albuminoid material is a combination of chondroitin-sulphuric acid and histon. This acid, carried by the blood or lymph, "combines with a protein of the fixed tissues" (W. Taylor Cummins, "Proceedings of Patholog. Soc. of Philadelphia," Dec., 1910).

In rare cases this deposit is only a local mass. The corpora amylacea of the prostate and nervous structure are due to albuminoid deposit. Such a deposit may take place directly in a tuberculous focus or a syphilitic lesion.

In the vast majority of instances the disease is general, involving blood-vessels, the membrana propria of mucous membranes, the liver, the kidneys, and especially the spleen. The lymph-glands, tonsils, stomach, intestines, heart, and connective tissues are less often involved. The amyloid material is deposited between the cells and not in them. The tissues of a subject of amyloid disease are very prone to suppurate from even slight infection.

In all or nearly all cases the spleen is involved. The victim of general albuminoid disease is pale, greatly exhausted, emaciated, and very anemic; suffers from diarrhea and usually develops capillary hemorrhages beneath the skin and mucous membranes. The albuminoid material can be detected chemically in the urine if the kidneys are involved. Albuminoid degeneration is incurable and is usually fatal; but if the patient is subjected to proper treatment soon after it begins it may be arrested and never progress.

**Diagnosis of Tuberculous Abscess.**—The fluctuation, the absence of evidences of acute inflammation, the tendency to wander, and, in some cases, the sudden appearance, mark the diagnosis. The surgeon always examines with care to see whether there is some distant tuberculous focus from which the abscess may have wandered, or whether the abscess itself is at the primary seat of disease. The advancing impairment of the general health, the lessened amount of hemoglobin, the normal or almost normal temperature, and the absence of leukocytosis are points in the diagnosis of the condition. In a doubtful case the aseptic use of the tubular exploring needle is important, the fluid that emerges being studied by the microscope after staining, by cultures, and perhaps by inoculating it into guinea-pigs. The fluid that is withdrawn may contain no bacteria that can be demonstrated; but if it is sterile, one should strongly suspect tuberculosis. Various diagnostic tests for tuberculosis will be found on pages 226, 227, 228, 229, and 230.

**Prognosis.**—Advanced albuminoid degeneration makes the prognosis of tuberculous abscess hopeless and any extent of albuminoid degeneration is unfavorable. Secondary pyogenic infection, as already stated, may produce death or a lingering suppuration. The prognosis is worse in very young children than in adults; and in any case it is unfavorable if the exhaustion deepens, if the anemia is marked, if there are tuberculous lesions in distant parts or in important organs or structures, if the patient is unable to take enough food or if he cannot digest what he does take, and if the regions of tuberculosis cannot be extirpated or sterilized. Under other circumstances the prognosis is favorable.

**Tuberculous Abscesses in Various Regions.—Tuberculous abscess of the head of a bone** (see Brodie's Abscess, page 497) arises in the cancellous structure of a long bone, most often in the head of the tibia, and is fre-

quently noted as having been preceded by a trivial traumatism. The focus of tuberculosis seldom induces severe symptoms unless secondary pyogenic infection occurs. A tuberculous nodule forms as a result of tuberculous osteomyelitis. The bone about the nodule is hyperemic, the bony trabeculæ are thickened, and the cancellous spaces "are devoid of fat cells, and they contain a swollen semifibrous material" (Warren's "Surg. Pathol."). The center of the nodule becomes cheesy, the bone trabeculæ are absorbed, and the bone becomes cheesy and broken up, the cheesy mass containing bone fragments. Finally the area becomes filled with tuberculous pus, the cavity which contains it being lined with tuberculous granulations. Distinct sequestra may form and the bone about the diseased focus undergoes sclerosis. In tuberculous abscess of bone pain is continued, but is not usually very severe, is of a boring character, and is worse when the patient is in bed. Attacks of synovitis arise from time to time in the adjacent joint. The bacteria of tuberculosis obtain access to the bone by means of the blood, and find in the bone a point of least resistance. There is no such thing as an acute abscess of bone. A pyogenic inflammation, of such severity that it would cause an acute abscess in soft parts, in bone causes acute necrosis. A less violent pyogenic infection causes a very chronic suppuration.

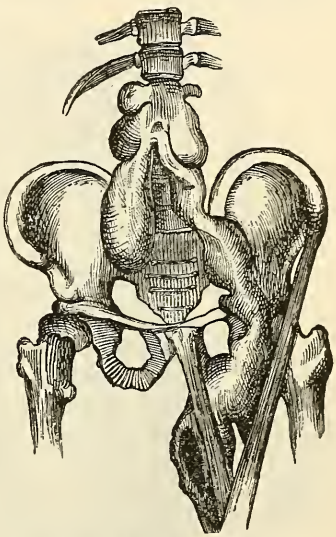


Fig. 102.—Psoas abscess (Albert).

**Retropharyngeal** or **postpharyngeal abscess** is often tuberculous. Such an abscess is usually due to caries of the cervical vertebræ, but can arise in the connective tissue of the parts or as tuberculous adenitis. An abrasion of the mucous membrane may admit the bacilli to the connective tissue or the glands. A swelling projects from the posterior pharyngeal wall, and there is great interference with respiration and deglutition. Caseous matter from caries of the cervical vertebræ may reach the posterior mediastinum by following the esophagus, or may appear in front of or behind the sternomastoid muscle in the neck (Edmund Owen). A tuberculous abscess back of the pharynx is apt to undergo pyogenic infection, in which case the patient develops fever, sweats, pain, and prostration.

**Dorsal Abscess.**—The tuberculous matter in dorsal abscess arises from dorsal caries, flows into the posterior mediastinum, and reaches the surface by passing between the transverse processes. The tuberculous matter from dorsal caries may run forward between the intercostal muscles or between these muscles and the pleura, pointing in an intercostal space, at the side of the sternum, or by the rectus muscle. It may burst into the gullet, windpipe, bronchus, pleural sac, or pericardium. It may descend to the diaphragm and travel under the inner arcuate ligament to form a psoas abscess, or under the outer arcuate ligament to form a lumbar abscess. A psoas abscess may point above Poupart's ligament or in the lumbar region. If it extends below Poupart's ligament it usually points external to the femoral vessels (a characteristic which is said to distinguish it at once from an ordinary femoral hernia), but may burrow in any direction.

**Iliac abscess** arises from lumbar caries, the swelling lying in the iliac fossa and pointing above Poupart's ligament.

**Psoas abscess** is usually due to lumbar caries, but may arise from dorsal

caries. The fluid usually points in Scarpa's triangle external to the femoral vessels, but may descend much lower (Fig. 102). A psoas or iliac abscess, by following the lumbosacral cord and great sciatic nerve, forms a gluteal abscess. These abscesses may open into the bowel, bladder, ureter, or peritoneal cavity. A hernia is almost never mistaken for a psoas abscess, but a psoas abscess is sometimes mistaken for a hernia (Fig. 103). J. Torrance Rugh points out that without a search for spinal kyphosis or muscular rigidity the mistake may be made, but that the presence of a mass in the iliac fossa continuous with the external lump eliminates the possibility of the condition being hernia.

**Lumbar Abscess.**—In a lumbar abscess the fluid produced by dorsal caries descends beneath the outer arcuate ligament, or the fluid from lumbar caries which collected anterior to or in the quadratus lumborum muscle passes between the last rib and iliac crest in the triangle of Petit, the small space bounded by the crest of the ilium, the posterior edge of the external oblique muscle, and the anterior edge of the latissimus dorsi muscle.[1]

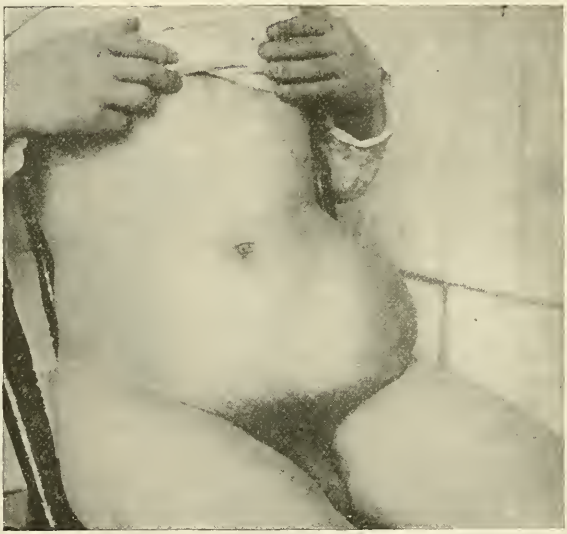


Fig. 103.—Case of cold abscess of the abdominal wall which had been treated as a hernia.

**Tuberculous abscess of the neck** results from tuberculosis of the cervical glands. It is not often that such an abscess attains any considerable size. It tends strongly to spontaneous rupture, and, if this is permitted to occur, a livid, corrugated scar results.

**Tuberculous Abscesses of Joints.**—(See Section XIX.)

**Tuberculous Abscess of Rib.**—It is not uncommon to find a tuberculous abscess of moderate size about a tuberculous rib. The pleura may become involved secondarily.

**Tuberculous mediastinal abscess** may result from the downward passage of a cervical abscess; from tuberculosis of the sternum, ribs, vertebræ or pleura, or from tuberculous mediastinal glands.

**Tuberculous abscess of the breast** is a caseated and liquefied a ea of tuberculosis of the breast. A lump is detected, which slowly enlarges and finally ruptures, sinuses being formed. The axillary glands are apt to be implicated. The patient may belong to a tuberculous stock, as a rule gives a history of

[1] For a lucid description of these abscesses see Owen's "Manual of Anatomy," from which much of the above is condensed.

previous tuberculous troubles of various sorts, and has usually borne children. Tuberculous abscess of the breast causes little or no pain.

**Treatment of Tuberculous Abscess.**—For many years the majority of surgeons would not open a tuberculous abscess unless it was on the point of rupturing. With the advent of antiseptic surgery it was assumed that aseptic incision and drainage would be the proper treatment for these cases; but the results, except in small superficial tuberculous abscesses, have been extremely disappointing. If a large abscess is so treated, pyogenic infection will, in all probability, sooner or later occur, with all its possibilities of disaster. Incision and drainage is, therefore, restricted to small and superficial abscesses.

**Treatment of Small Superficial Tuberculous Abscesses.**—The surgeon must remember that after one has opened an apparently superficial abscess it is his duty to make an examination to see that there is no channel connecting the abscess with a deep or a distant focus. If he finds such a channel, he may be disposed to follow one of the plans of treatment outlined below and on page 244. It is also his duty to see whether there are sinuses tracking off from the abscess; and if these exist, he must slit them up. If there are loculi in the wall of the abscess, he must stretch their mouths. He must be particularly careful to see that he is not dealing with a *shirt-stud abscess*, in which there is a little opening through the deep fascia connecting the abscess above with the abscess below. In a shirt-stud abscess the deep fascia must be freely incised. After the abscess has emptied itself, its walls must be thoroughly scraped with a curet, and the cavity must be drained with a tube or, preferably, with iodoform gauze. If the skin above a superficial abscess is diseased and discolored, and the abscess is on the eve of spontaneous rupture or has ruptured, the discolored skin must be cut away with scissors. If the discolored skin is allowed to remain, a livid and jagged scar will inevitably result. If it is cut away a healthy scar, not very deforming, will result.

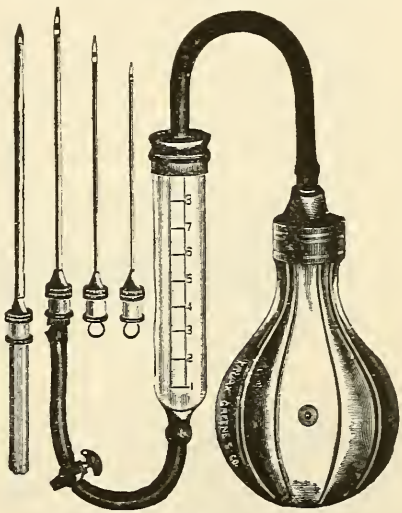


Fig. 104.—Senn's injection syringe.

**Treatment of Tuberculous Abscesses of Considerable Size.**—*Method 1. Aspiration, Irrigation, and the Introduction of Iodoform.*—The operation is carried out with the most scrupulous aseptic care. The trocar is passed through the sound skin; is carried beneath the skin for an inch, as Senn suggests; and is then made to enter into the cavity of the abscess. The stylet is pulled out, and the flow of fluid is aided by very delicate pressure. Occasionally the tube will become blocked by necrosed tissue or plugs of fibrin. It is opened up again by pushing in a wire or forcing through it a stream of sterile fluid. When tuberculous matter ceases to run out of the trocar, a very warm solution of boric acid is thrown in in order to wash the abscess walls. This can be injected with a fountain syringe or with the special apparatus of Senn (Fig. 104). Enough of it is allowed to enter to overdistend the abscess-cavity, as Mr. Callender long ago advised. The fluid is then allowed to pass out; fresh fluid is passed in; and this procedure is repeated, perhaps again and again, until entirely clear fluid flows out. When this takes place, an emulsion of iodoform is thrown in by Senn's syringe. A 10 per cent. emulsion in glycerin is as satisfactory

as the more elaborate formulas. Verneuil used to employ iodoform and ether; but this is painful, is more liable to cause iodoform-poisoning, and sometimes induces gaseous distention and ruptures the wall of the abscess. In order to prevent the danger of iodoform-poisoning the surgeon should not introduce at one time more than 8 dr. of the emulsion, if dealing with an adult; or more than 4 dr., if dealing with a child. After the emulsion has been inserted into the abscess-cavity the wound in the skin is sealed with a bit of gauze and iodoform collodion. Gauze is fluffed up and laid on the skin above the abscess, and the walls of the cavity are then forced toward each other by applying a roller bandage. The part is put at complete rest, and it is usually necessary to put the patient in bed. Sometimes, although very seldom, one injection will produce a cure; but usually, after one or two weeks, it will be observed that the cavity has to some extent filled again. A second operation is then performed; and, if improvement is really taking place, it will be found that the fluid is not nearly so thin as it was at the first operation. It is needless to persist in this method after six or seven attempts have failed to cure. If the abscess has thick and uncollapsed walls it is not fitted for treatment by aspiration and injection.

*Method* 2. *Incision, Cleansing, and Suture.*—If, owing to the considerable size or the rather rigid walls of the abscess, one believes that the aspiration method would be useless; or if the aspiration method has been tried and has failed, one may adopt the following plan. It should not, however, be employed if the walls are *very* thick and rigid. An incision is made at the most

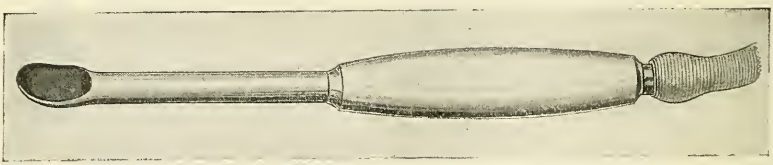


Fig. 105.—Barker's sharp-edged irrigating curet ("Keen's Surgery").

dependent part of the abscess. The walls are scraped carefully with Barker's sharp-edged irrigating curet (Fig. 105), and are rubbed smooth with bits of gauze. The part is freely irrigated with hot boric acid solution, and pressure is applied to arrest bleeding. Iodoform emulsion is introduced; the skin is sutured; dressings, compresses, and bandages are applied, and complete rest is secured. This operation may cure an abscess, or it may be necessary to repeat the procedure two or three or many weeks afterward.

*Method* 3. *Incision and Removal of the Primary Focus of Tuberculosis.*—If the surgeon does not wish to use the iodoform treatment, or if it has failed, and if he finds that the primary seat of disease may be attacked and removed, an operation should be undertaken to get rid of Volkmann's membrane in the last-formed abscess and also to remove the primary tuberculous focus. An incision is made, when possible, that will lay open not only the last-formed abscess, but the primary lesion. Tuberculous tissue is thoroughly removed by Barker's spoon and by rubbing with gauze or, perhaps, by scissors and forceps. Any focus of bone disease is curetted and touched with pure carbolic acid, and loose fragments of bone are removed. The part is irrigated with a hot solution of boric acid; bleeding is arrested by pressure, and the wound is nearly, but not quite, closed, drainage being inserted at the most appropriate spot. Dressings, compresses, and bandages are then applied. In this operation the entire tuberculous area has been removed and the raw surfaces have been forced into contact, and there is scarcely more danger of secondary pyogenic infection than there is in any ordinary wound.

**General Treatment.**—It is never to be lost sight of that in every case of

tuberculous abscess the general treatment of tuberculosis must be rigorously pursued (see page 230). In the treatment of a cold abscess give nutritious food, cod-liver oil, quinin, iron, and the mineral acids. Removal to the mountains or the seaside is often indicated, life in the open air is imperative, and mechanical appliances may be needed for diseases of the bones and joints.

**Tuberculous Abscess of Bone.**—Make an incision to bare the bone. Open the abscess with the trephine, the gouge, or the chisel; curet the interior of the wall of the cavity with a sharp spoon and rub it with bits of gauze; cut away the edges of the bone with rongeur forceps; irrigate the cavity with hot normal salt solution, dry its walls with gauze, and paint the cavity with pure carbolic acid; pack with iodoform gauze and apply antiseptic dressings. It is better not to employ an Esmarch apparatus. Bleeding will not be severe, and when no apparatus is used to prevent bleeding it is possible to see and thus be sure that all the diseased bone has been removed.

**Tuberculous Abscess of Lymphatic Glands.**—In non-exposed portions of the body the capsule of the gland should be incised and dissected or scraped away and the cavity swabbed out with pure carbolic acid or iodin and packed with iodoform gauze, but drainage should not be prolonged. If the abscess is allowed to burst, it will cause an ugly scar; therefore, in exposed portions of the body, as the neck, special effort should be made to prevent a scar by incising early before the skin is involved. When only a little caseated matter exists and the skin is not discolored, prepare the parts antiseptically, incise, rub the interior with gauze, inject iodoform emulsion, and suture the wound. It used to be a custom in such cases to carry a silk thread by means of a needle through the skin, through the gland, and out at its lowest point, the part being then dressed with gauze. In three days the thread was removed and a firm compress was applied. The plan is not satisfactory and incision is to be preferred. When the gland is almost entirely broken down and the skin above it is becoming purple and thin, insert a hypodermatic needle through sound skin into the abscess, draw off the fluid tuberculous matter, and inject iodoform emulsion. This procedure is to be repeated when the fluid again accumulates. By this means we can sometimes effect a cure in a week or so. When an abscess breaks or is on the point of breaking, cut away all purple skin, curet the abscess walls (the abscess having become a tuberculous ulcer), remove the remains of gland and capsule, swab the cavity with pure carbolic acid, and dress with iodoform and antiseptic gauze.

Tuberculous glands, if not cured by general treatment, ought to be extirpated. They should certainly be extirpated before they caseate and form an abscess. If an abscess does form it is treated as directed above, and after healing takes place the diseased glands may be extirpated. If sinuses exist they are curetted and touched with iodin or carbolic acid. After healing, the glands are extirpated. When sinuses exist there is always mixed infection.

**Tuberculous Abscess of a Rib.**—This lesion requires incision of the soft parts and resection of the diseased bone. The tuberculous area is thoroughly curetted, rubbed with pure carbolic acid, and packed with iodoform gauze.

**Tuberculous Mediastinal Abscess.**—In tuberculous abscess of the mediastinum aspiration and injection of iodoform may prove efficient. In some cases it will be necessary to open and drain.

**Tuberculous Abscess of the Mammary Gland.**—Many operators simply incise, curet, pack with iodoform gauze, and dress antiseptically. It is wiser to remove the entire gland and to clear out the axilla, as in an operation for cancer, in order to prevent both recurrence and dissemination.

**Large Tuberculous Abscesses.**—In view of the facts that these abscesses may cause no trouble for years and that an operation may be fatal, some eminent surgeons are opposed to an operation unless the abscess is moving toward

inevitable rupture or is disturbing the function of organs by pressure. Most practitioners believe, however, and I agree with them, that this mass of tuberculous matter is a source of danger through being a depot of infective organisms which may overwhelm the system, and that death will seldom result from an operation performed by one who employs with intelligence strict antisepsis. In no other cases is attention to every detail more important, as a mixed infection may easily take place, and will probably mean death. As W. Watson Cheyne points out, over 70 per cent. of cases of spinal abscess treated by aseptic methods recover completely and without any real illness after such an operation. The recoveries from the old let-alone method will be infinitely less than this, and cases cured by operation usually remain well. The surgeon must always remember that the wall of the abscess and not the fluid in the cavity is the real seat of disease, and this wall must be actually removed or completely sterilized if operation is to be safe. To simply open, drain, and leave the wall to Nature to get rid of as she can is fraught with the gravest peril.

**Psoas Abscess.**—Some of these cases can be treated by aspiration and injection (see page 243), others by incision and subsequent suture (see page 244), others by the radical operation set forth on page 244.

Treves's operation for psoas abscess is described on page 695.

An operation occasionally performed for psoas abscess consists in an incision in the groin, an incision in the back, removal of carious vertebræ, thorough cleansing of the abscess wall, and through-and-through tubular drainage. It has been found, however, that this operation is uncertain and dangerous. It is not advisable to remove carious vertebræ unless the carious parts are loose, and through-and-through tubular drainage is rarely used unless mixed infection already exists. When a large abscess breaks spontaneously it should be widely opened at once, scraped and irrigated, rubbed with gauze, and packed with iodoform gauze. If secondary pyogenic infection of a large tuberculous abscess does occur the patient will develop septic fever and will probably die (*q. v.*).

**Dorsal abscess** and **lumbar abscess** are treated after the same plan as psoas abscess. One incision only is usually necessary unless the fluid has traveled to a distant point.

A **postpharyngeal abscess** must not be opened through the mouth. To open it in this manner puts the patient in danger of suffocation by fluid running into the larynx during or after the operation. Further, mixed infection of the abscess area will be certain to ensue. Septic pneumonia will be apt to arise from inhaled infected particles, and profound gastro-intestinal disturbance will be liable to develop because of the inevitable swallowing of purulent, putrid, and tuberculous masses. Incise the neck and open into the abscess by Hilton's method, going through the sternocleidomastoid muscle or behind it. Rub the wall of the abscess with bits of gauze, remove any loose bone, irrigate with hot normal salt solution, inject iodoform emulsion, insert a tube or pack with iodoform gauze.

**Tuberculosis of the skin** may arise from inoculation with material derived from a bovine or human source. It is frequently found that some other member of the family labors under tuberculous disease or that some family predecessor, direct or collateral, suffered from it. Stelwagon ("Diseases of the Skin") includes all cases under five heads: (1) tuberculosis ulcerosa; (2) tuberculosis disseminata; (3) tuberculosis verrucosa; (4) scrofuloderma; (5) lupus vulgaris.

**Tuberculosis Ulcerosa.**—The disease arises at a mucous outlet and is usually secondary to internal tuberculous disease. Small miliary tubercles form which caseate and are converted into ulcers. An ulcer is shallow,

round or oval in outline, with soft edges, the floor being composed of sluggish or edematous granulations covered with a crust. The discharge is scanty and seropurulent. In some cases there is but one ulcer; in others there are two or several, and the fusion of ulcers produces a serpiginous outline. The ulcers do not tend to heal, but gradually and steadily advance. Such ulcers are met with about the mouth, the genital organs, and the anus.

**Tuberculosis Disseminata.**—This occurs only in children; it is acute in onset and widespread. One type is polymorphic: spots, papules, pustules, and crusted ulcers existing, and lymphatic glands being enlarged. Another type arises after an attack of an exanthematous fever and presents "a rough resemblance to flat lupus tubercles, to sluggish acne papules, and to lichen scrofulosum" (Stelwagon, "Diseases of the Skin").

**Tuberculosis Verrucosa.**—*Anatomical tubercle*, the *verruca necrogenica* of Wilks, is due to local inoculation with tuberculous matter. It may be met with in surgeons, the makers of postmortems, leather workers, and butchers, usually upon the backs of the hand and fingers. It consists of a red mass of granulation tissue having the appearance of a group of inflamed warts. Pustules often form.

**Scrofulodermata or Tuberculous Gummata.**—By "scrofulodermata" we mean chronic inflammations of the skin, the granulation-tissue product of which caseates, mixed infection occurs, and small abscesses, sinuses, or ulcers form. A *tuberculous ulcer* has a floor of a pale color, and has no granulations at all, or is covered with large, pale, edematous granulations. The discharge is thin and scanty. The ulcer is surrounded by a considerable zone of purple, tender, and undermined skin, which is apt to slough. When healing occurs, the skin puckers and usually inverts.

**Lupus** begins usually before the age of twenty-five, but is met with often in individuals in middle life. It is most common upon the face, especially the nose. It is a very chronic and extremely destructive disease. Three forms are recognized: (1) *lupus vulgaris*, in which pink nodules appear that after a time ulcerate and then cicatrize partly or completely. These nodules resemble jelly in appearance; (2) *lupus exedens*, in which ulceration is very great, and (3) *lupus hypertrophicus*, in which large nodules or tubercles arise. Lupus may appear as a pimple, as a group of pimples, or as nodules of a larger size. The ulcer arises from desquamation, and is surrounded by inflammatory products which, by progressively breaking down, add to the size of the raw surface. The ulcer is usually superficial, is irregular in outline, the edges are soft and neither sharp nor undermined, the sore gives origin to a small amount of thin discharge, the parts about are of a yellowish-red color, the edges are solid and puckered and scar-like and there is no pain. The sore is often crusted, the crusts being thin and of a brown or black color; it may be progressing at one point and healing at another; it is slow in advancing, but often proves hideously destructive. The scars left by healing are firm and corrugated, but are apt to break down. Clinically, it is separated from a rodent ulcer by several points. The rodent ulcer is deep, its edges are everted, and the parts about filled with visible vessels. It is not crusted, has not a puckered edge, its edges and base are hard, and it rarely shows any tendency to healing. Many victims of lupus live twenty or thirty years and die without the development of pulmonary tuberculosis. It is estimated that one-third of those who die show signs of pulmonary tuberculosis. Lupus patients exhibit a strong focal reaction to tuberculin and also show von Pirquet's reaction.

**Tuberculosis of Subcutaneous Connective Tissue.**—In this form of tuberculosis tuberculous nodules form and break down (tuberculous abscesses). In the deeper tissues these abscesses are usually associated with bone, joint, or lymphatic gland disease (see Cold Abscess, page 236).

**Tuberculosis of the Mammary Gland.**—(See page 245.)

**Tuberculosis of Blood-vessels.**—It is certain that bacilli in the blood or in tuberculous emboli may establish intravascular tuberculosis.

**Tuberculous Meningitis.**—(See page 801.)

**Tuberculosis of nerves** is excessively rare. Tuberculous neuritis may arise in the course of general tuberculosis. A nerve lying in a tuberculous area may itself become tuberculous. It rarely does so, however. In fact, nerves resist infections though in the midst of them, and for this reason have been called the "aristocrats of the body."

**Pulmonary Tuberculosis.**—In adults the lungs are more commonly affected than any other structure. The lung affection may be primary or may be secondary to some distant tuberculous process. Pulmonary tuberculosis belongs to the province of the physician and requires no description here.

**Tuberculosis of the pleura** is not uncommon. Tuberculous pleurisy may be acute or chronic. In some instances mixed infection takes place and suppuration occurs. The tuberculosis may be primary, but is usually secondary to pulmonary tuberculosis, and may be due to direct extension or to rupture of an area of pulmonary softening. A primary pleurisy not due to traumatism is very apt to be tuberculous. In many cases of tuberculous pleurisy there are tubercles present and in some cases there are none.

**Tuberculosis of the Alimentary Canal.**—A tuberculous ulcer of the lip occasionally arises, and may be mistaken for a cancer or a chancre. A tuberculous ulcer of the tongue is commonly associated with other foci of disease. Such ulcers are separated from cancer by their soft bases and edges and by the rarity of glandular enlargements, and from syphilitic processes by the therapeutic test. Confirmation of the diagnosis is obtained by cultivations and inoculations. Tubercle may affect the pharynx, palate, tonsils, and, very rarely, the stomach.

**Gastric Tuberculosis.**—It is thought that the acid gastric juice must protect the stomach from tubercle, because tubercle bacilli are frequently introduced into the stomach, but the organisms very rarely lodge and multiply in the stomach wall. Furthermore, bacilli when introduced into the stomach are retained but a short time and the stomach walls contain few lymph-follicles (Barchasch, in "Beit. Z. klin. d. Tuberculose," vii, Part III, 1907). It may be assumed that gastric catarrh and motor impairment are predisposing causes. Gastric tuberculosis may be primary, but is usually secondary to pulmonary tuberculosis, infected sputum having been repeatedly swallowed ("Jour. Am. Med. Assoc.," Dec. 28, 1907). Gastric tuberculosis may cause cicatricial stenosis of the pylorus, ulcer (of which I reported an instance), a tumor-like thickening, solitary tubercle, and miliary tuberculosis (Barchasch, Loc. cit.).

**Intestinal tuberculosis** may follow pulmonary tuberculosis, but it may arise primarily in the mucous membrane of the bowel or result from tuberculous peritonitis. Intestinal tuberculosis causes diarrhea and fever, may resemble appendicitis, and may cause abscess and perforation. True tuberculous disease of the appendix occasionally occurs. Tuberculosis of the cecum is by no means as rare as we used to believe (see page 1014). Fistula in ano is frequently tuberculous, and when it is, the lungs are very often involved, the pulmonary lesion being usually primary.

**Tuberculosis of the Liver.**—Tuberculous disease of the liver causes cold abscess or cirrhosis. Typical cirrhosis without tubercles may arise, the bacilli being present in the tissues. Many cases of supposed alcoholic cirrhosis are probably tuberculous. The hepatic cirrhosis which may arise during peritoneal tuberculosis is tuberculous.

**Peritoneal tuberculosis** (see page 1027) may be primary, infection having taken place by way of the blood, may be part of a diffused process, or may follow

intestinal tuberculosis, the serous and mucous coats of the bowel having been at some point in contact or a follicular ulcer having perforated (Abbe). The germ may have entered by the Fallopian tube. Tuberculous peritonitis may be due to ovarian or Fallopian tuberculosis, or to ulceration of a tuberculous appendix. In some cases a caseating tuberculous gland furnishes the causative bacilli. Peritoneal tuberculosis usually causes ascites, tympany, and tumor-like formations composed of adherent bunches of bowel or omentum or distended mesenteric glands (see page 1027).

**Tuberculosis of the Pancreas.**—Tuberculous sclerosis of this organ has been induced experimentally by Carnot.

**Tuberculosis of the spleen** may occur with tubercles or as a sclerosis.

The **heart muscle** is rarely attacked by tuberculosis. In fact, valvular lesions of the left side of the heart actually protect the individual from pulmonary tuberculosis. Non-tuberculous endocarditis may arise in the course of a tuberculous process elsewhere. Tuberculous endocarditis does *very rarely* occur.

The **endocardium** may be inflamed and covered with fibrous exudate as a result of the toxins from some distant point of tuberculosis.

The **pericardium** may be attacked with primary tuberculosis, or the process may be secondary to pleural tuberculosis. There may or may not be tuberculosis.

**Tuberculosis of the brain** induces meningitis and hydrocephalus (see page 801).

**Tuberculosis of the membranes of the spinal cord** is seen alone or in association with tuberculous inflammation of the brain.

**Tuberculous disease of fascia** is common; in fact, fascia is peculiarly prone to infection. Fascia may be attacked primarily, and when it is, the disease is apt to spread rapidly and widely and to produce most disastrous results. The elder Senn regards tuberculosis of the intermuscular septa of the thigh as a very grave condition, which, if extensive, demands amputation of the limb. Secondary tuberculosis of fascia is far more common than the primary form, the original focus of disease being in bone, joint, tendon-sheath, or lymph-gland.

**Tuberculosis of muscle** is rare. Instances of primary tuberculosis have been reported. Secondary tuberculosis is more common, but even this condition is rare, muscle seeming to have a high degree of resistance.

**Tuberculous disease of bone** (see page 494) is very common in youth, and a sprain or a contusion, which is oftener slight than severe, may precede any signs of the disease. The injury establishes a point of least resistance, and in the damaged area the bacilli are depsoited and multiply, or a latent area of tuberculosis is roused into activity by the traumatism. The organisms may be deposited directly from the lymph or blood, or may arrive in an embolus from a distant tuberculous focus (lung or lymph-gland), which embolus is caught in a terminal artery in the end of a long bone and causes a wedge-shaped infarction.

Tuberculous osteomyelitis, as a rule, begins just beneath the articular cartilage or in the epiphysis. There may be one focus, several foci, or many foci in the same bone. The products of the tuberculous inflammation constitute tuberculous nodules which destroy the medullary tissue and hence cut off the nutrition of adjacent bone. Bone trabeculæ are destroyed, and tuberculous granulations take their place, and here and there small dead portions of bone trabeculæ lie as sequestra among the granulations. In some bones, for instance, the vertebræ and the bones of the carpus and tarsus, the tuberculous process spreads widely; in some it tends to remain localized. Tuberculous granulations may be absorbed, may be encapsuled, may be

replaced by fibrous tissue, or may caseate (see page 214). When an osseous tuberculous focus spreads the bone enlarges and becomes spindle shaped, as is seen in a phalanx the seat of tuberculous osteomyelitis, the condition known as *spina ventosa*.

**Tuberculous disease of the joints** (see page 618) is called *white swelling* and also *pulpy degeneration* of the synovial membrane. Joints are especially liable to tuberculosis in youth, although the wrist and shoulder not infrequently suffer in adult life. Joint-tuberculosis is often preceded by an injury. The tuberculous process in rare cases begins in the synovial membrane. Primary synovial tuberculosis is most often met with in the knee-joint. Usually the disease begins in the end of a bone, dry caries resulting, necrosis ensuing, or an abscess forming, which may break into the joint.

*Poncet's rheumatism* or tuberculous articular rheumatism is a condition in which toxic joint inflammation is evidence of latent tuberculous infection perhaps at some distant point, it being often impossible to demonstrate the bacillus in the joint fluid or tubercles at the autopsy (see page 619).

**Tuberculosis of lymphatic glands** is known as *tuberculous adenitis*. It is the most typical lesion of scrofula. Tuberculous adenitis is most frequent between the third and fifteenth years. A person not of the tuberculous type may acquire tuberculosis of the glands, but the disease is unquestionably of much greater frequency in those who are recognized as predisposed to tuberculosis. Tuberculous glands may get well, may even calcify, but usually caseate if let alone. Long after healing they may break down and soften (*residual abscess of Paget*). They very frequently suppurate because of mixed infection. Though at first a local disease, tuberculous glands may prove to be a dangerous focus of infection, furnishing bacteria which are carried by blood or lymph to distant organs or throughout the entire system. Glandular enlargement is in rare instances widely diffused, but it is far more commonly localized. Enlargement of the cervical glands is most common. Tuberculous disease of the mesenteric gland is known as *tabes mesenterica*. Tuberculosis of lymph-glands may be due either to bovine bacilli or to human bacilli.

**Tuberculosis of the cervical lymph-glands** is a very common condition. It is most common in children over two years of age and is often seen in young adults. It is rare in children under two and in persons of middle age. In the majority of cases infection takes place from the tonsils, pharynx, or posterior part of the oral cavity, and in these cases the first glands to enlarge are those just below the parotid salivary gland. In a number of cases enlargement begins in the submaxillary or submental glands, and in these cases infection originates from the teeth, mouth, or face (Dowd, in "Surgery, Gynecology, and Obstetrics," March, 1909). A tuberculous lesion of the scalp may be followed by tuberculosis of the parotid lymph-glands. Cervical adenitis may be unilateral or bilateral and is a very chronic condition. It is predisposed to by enlargement of the tonsils, adenoids, and nasopharyngeal catarrh. In 30 per cent. of cases the bacilli of bovine tuberculosis may be found (Dowd, Ibid.). The enlargements usually arise insidiously, but sometimes (after whooping-cough, measles, or scarlet fever) they come on more rapidly. When first observed the enlargements are small, round, firm, isolated, painless, and somewhat movable. As they enlarge they fuse into an irregular swelling which may be quite tender and is always anchored to surrounding parts. As the glands caseate the mass softens, the skin over it becomes adherent and red, and finally breaks open. Cold abscess may form or mixed infection with pus cocci may take place.

Cervical lymphadenitis may be confused with lymphadenoma. The former, as a rule, first appears in the submaxillary triangle; the latter, in the occipital or sternomastoid glands. The mass in the former is more tender, softer, and

less movable than in the latter. Tuberculous glands weld together, they are apt to remain localized for a considerable time, and they tend to soften. The younger the patient, the greater the probability of softening. In adults there is comparatively slight tendency to softening. Tuberculous adenitis may be accompanied by other tuberculous manifestations. Lymphadenoma from the start affects many glands; it may arise simultaneously in several regions, although in some cases there is a distinct beginning in one region. Lymphadenoma shows very little tendency to suppurate, and does not break down except late in the course of the disease, and is accompanied by great debility and anemia. Tuberculin tests may aid in the diagnosis, but a difficulty is that Hodgkin's disease and tuberculosis may coexist. Malignant gland-tumors infiltrate adjacent glands and other structures, binding skin, muscles, and glands into one hard, firm mass.

Tuberculous cervical adenitis is in most instances a reasonably curable condition. In children under two or three years of age, however, it is a dangerous condition and one apt to be associated with severe pulmonary, osseous, or other complications. Some cases of adenitis can be cured by open-air treatment, food, medicine, tuberculin, and hygienic care. In many, however, operation is indicated in addition to such treatment, and these operations are usually successful if thoroughly performed when the disease is localized and softening has not occurred. When possible operation should be performed when the patient is at the seaside, or, at least, the patient should convalesce there if circumstances permit. Thorough extirpation is the proper operative treatment and any diseased condition of scalp, face, mouth, tonsil, or nasopharynx is to be corrected. My belief is that about 75 per cent. of cases are permanently cured by thorough operation. If a patient is well five years after operation the cure may be regarded as permanent. Cured cases seldom die subsequently of any tuberculous lesion.

It is not uncommon after removal of infected glands and healing of the wound to have several or numerous small, hard nodules form beneath the skin in the area operated upon. Dowd extirpated some of these nodules and found they were not tuberculous, but were fibrous. I have been able to confirm Dowd's statement in several of my own patients.

Medical treatment is not nearly so valuable as surgical treatment in this form of tuberculosis. If medical treatment alone is relied on, many of these cases develop pulmonary tuberculosis. Attridge, quoting Demme and Dowd ("Surgery, Gynecology, and Obstetrics," Dec., 1908), sets the number which develop it when medical treatment is relied on at 21 per cent., and the number developing other distant tuberculous lesions at 8.2 per cent.—a total of 29.2 per cent., and these figures do not include bone infections and late infections of the lymph-nodes. Even in cases supposed to have been cured by medical means it will be found that most of them react to tuberculin, showing that lesions are latent rather than cured. Wohlgemuth shows that complete removal cures 75 per cent. of cases; curetting and drainage cures 63 per cent.; general treatment, 24 per cent. (Attridge, Ibid.). In a series of 100 cases operated upon by Dowd, pulmonary tuberculosis arose in but 1 case and bone tuberculosis in 3 cases. If miliary tuberculosis exists, if the patient is much exhausted, if the infection is not definitely localized, or if an internal organ is the seat of active tuberculosis, operation is not indicated.

Death seldom follows operation. The mortality without operation is probably 10 per cent. Complete extirpation of the involved group of glands is practised when the disease is well localized. If it is not well localized I follow Attridge's plan (Ibid.) and wait until it becomes so, treating the patient in the interval by open-air life, nourishing food, medicine, tuberculin, and the *x*-rays. If softening occurs, the area should be incised and curetted and the exposed sur-

face should be treated by repeated applications of tincture of iodin. When healing occurs, extirpation is to be performed. If sinus formation exists, mixed infection has occurred or will occur and the sinuses must be curetted and treated with iodin until they heal, when the glands may be extirpated. I do not believe in a bilateral extirpation at one séance. The operation even on one side is prolonged and bloody and is all the patient is fit to stand. If both sides of the neck are involved, an interval of several weeks should be insisted on before the other side is attacked.

In chronic cases of cervical lymphadenitis it is invariably necessary to search for intra-oral and nasopharyngeal disease, and if such disease exists it must be treated before the glands are removed. After operation rigid open-air life is insisted on.

**Tuberculosis of tendon-sheaths** (*tuberculous tenosynovitis*) is discussed on page 722.

**Tuberculosis of the Kidney.**—(See page 1290.)

**Tuberculosis of adrenals** may cause sclerosis.

**Tuberculosis** may attack the **Fallopian tubes, ovaries,** or **uterus.**

**Tuberculosis of the urethra, prostate gland, seminal vesicles,** and **bladder** is considered in a section on Regional Surgery.

**Tuberculosis of the Testicle** (see page 1393).—This disease is not rare. It is sometimes primary, but is usually preceded by tuberculosis of the kidney, bladder, or prostate. But one testicle is affected in the beginning, but the other gland is apt to be attacked later. The tuberculous mass softens, becomes adherent to the scrotum, and breaks or bursts, exposing the damaged testicle (*fungus of the testicle*). The cord is apt to be involved in tuberculosis of the testicle.

**Typhobacillosis.**—This condition was described by Landouzy in 1883. It is a toxemia in which the localization of lesions is very much deferred and is preceded by a prolonged typhoid or septic stage (Matas, "Southern Med. Jour.," Oct., 1911). Bacilli of tubercle are widely distributed throughout the body, but for a long time there are no tubercles formed, and all of the symptoms are due to bacillary poison. Matas describes the condition as "a continued fever with remissions and enlargement of the spleen, without signs of visceral localization" (Ibid.). It strongly resembles typhoid, but the pulse is more frequent, the temperature is less regular, there are no spots, and no intestinal or bronchial symptoms pointing to typhoid. Few of these cases die early. Most of them make an incomplete recovery after several weeks, and in periods varying from weeks to months develop evident localized tuberculosis.

**Acute Miliary Tuberculosis.**—In this condition an organ, several organs, or the entire body is infected with tubercle bacilli which have caused the formation of multitudes of tubercles. The symptoms for a time resemble typhoid, but in a short time an organ or organs present evidences of disease. Death occurs in from three weeks to three months.

## XIV. RACHITIS, OR RICKETS

**Rickets** was known by the people and named by them long before any medical man had written of it. It first appeared in the London bills of mortality in 1634. Glisson, in 1650, wrote the first description of it and renamed it rachitis, because of the commonly resulting spinal curvature. Rickets is a chronic disorder of nutrition arising during the early years of life (the first two or three) as a result of insufficient or of improper diet, aided and abetted in many cases by bad hygienic surroundings. A deficiency of fat and phosphate in the food

or the use of a diet which, by inducing gastro-intestinal catarrh, prevents assimilation causes rickets. It is characterized by incomplete osteogenesis and other nutritive failures. The disease is not common in nursing children unless breast-feeding has been unduly prolonged, and children fed upon artificial food are particularly apt to develop it. Holt says such diet is very deficient in fat and often in proteins, and contains an excess of carbohydrates ("Diseases of Infancy and Childhood"). Sir J. Bland-Sutton made some valuable experiments to indicate the injury done animals by denying them natural diet. He fed lion cubs in the London Zoological Gardens on raw horse meat only and the animals developed rickets. The rickety animals rapidly recovered on feeding them with milk and powdered bones mixed with cod-liver oil. The disease is essentially a city malady, "being principally seen in children living in crowded tenements where the effects of improper food are most strikingly shown; yet even here the disease is rare in those who get a plentiful supply of good breast-milk" (Holt, Ibid.). Rickets must not be regarded as a bone disease. It is true the bones are affected, but so are various structures and organs, all of the disorders being due to an underlying nutritive defect. Some maintain that lactic acid or some other toxic material produced in the intestinal canal causes bone inflammation, but most observers do not believe the bone changes are inflammatory. Children are very seldom born with rickets, but develop it later, the period of greatest liability being between the seventh month and the fifteenth month. So-called congenital rickets is usually sporadic cretinism. A child with rickets may become scorbutic (*scurvy rickets*). Some regard rickets as the result of an infection. Glisson, in the seventeenth century, thought it was infectious. Some observers claim to have found bacteria in rickety bone and in the cerebrospinal fluid of rickety children (Mirculi, Sorgente). Others think it results from thymus atrophy. Some blame syphilis, some malaria, some the thyroid gland, some the nervous system. Some believe that disease of the parathyroids alters calcium metabolism—perhaps two much calcium being cast out, perhaps too little being taken in or assimilated.

Whatever may be the cause of rickets, the essential condition in the bones is an insufficient deposit of mineral matter in the new bone cells. The new bone is soft and vascular and bone lamellæ toward the medullary canal are actually absorbed. There is excessive proliferation of cartilage which results in enlargement. The proliferating and imperfectly ossified cells cause enlargements at the ends of long bones and at the sternal ends of the ribs, and various bones bend and are distorted. The parietal bones bulge, the fontanels remain long open; there may be unossified gaps in the occipital bone, membrane only filling them (*craniotabes*). There may be pigeon-breast, bent long bones, curved spine, and distorted pelvis. The bones later may become firmly ossified in deformity. In rickets the spleen and liver are enlarged and the thymus is atrophied.

**Evidences of Rickets.**—Rickets is apt to arise in the spring, but may begin any time of the year. In some few cases it begins with fever. The condition is one of general ill-health; the child is ill-nourished, pallid, flabby; it has a tumid belly and suffers from attacks of diarrhea and sick stomach; it is disinclined for exertion and has a capricious appetite; it is liable to night-sweats: enlarged glands are often noted, the teeth appear behind time, and the fontanels close late. In health the posterior fontanel closes in the second month and the anterior fontanel in the eighteenth month. In rickets the anterior fontanel is often open when the child is three years of age. The sutures are often open at the end of the first year. The head is square in shape, the cranial bones are thick, and areas of thickening known as bosses appear over the parietal bones. The head is large and the forehead bulges. The long bones become much curved, the upper part of the chest sinks in, curvature of the spine appears, and

the pelvis is distorted. The ligaments are relaxed and lengthened and the joints are wobbly. The muscles are feeble and ill-developed. Infantile convulsions are common. Nocturnal restlessness and night terrors are the rule. Laryngismus stridulus and tetany may occur. Swelling appears in the articular heads of long bones, by the side of the epiphyseal cartilages, and in the sternal ends of the ribs, forming in the latter case *rachitic beads*. The lesions of rickets are due to imperfect ossification of the animal matter which is prepared for bone formation, and the soft bones gradually bend. The swellings at the articular heads are due to pressure forcing out the soft bone into rings. Rachitic children rarely grow to full size, and the disease is responsible for many dwarfs. Most cases recover without distinct deformity, but the time lost during the period when active development should have gone on cannot be made up, and some slight deficiency is sure to remain. Bow-legs, knock-knees, and spinal curvatures are usually rachitic in origin. The disease may be associated with scurvy, inherited syphilis, or tuberculosis. In appearance the rickety child is pot-bellied, pale and anemic, and usually fat and flabby, though occasionally thin. The spleen may be enlarged. There is great liability to enlargement of the tonsils, gastro-intestinal catarrh, and bronchial catarrh. The blood is deficient in red corpuscles and hemoglobin, and sometimes there is leukocytosis.

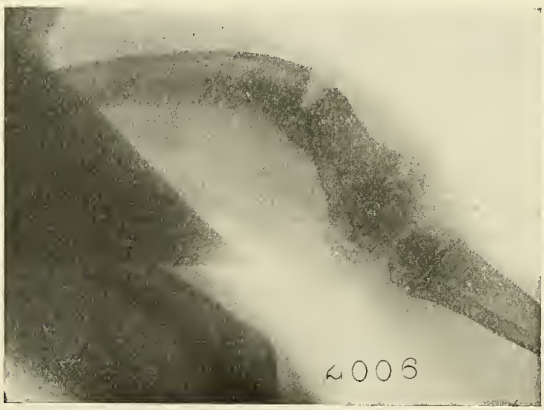


Fig. 106.—Fracture of femur in rickets.

The disease lasts for many months and is usually recovered from. It does not directly produce death, but is a powerful indirect cause of infant mortality because it lessens resistance and predisposes to many diseases. It is almost always afebrile, rarely congenital, and in unusual cases, known as late rickets, develops between the fifth and tenth year. The so-called acute rickets is practically always scurvy (Holt). The victims of rachitis are very liable to fracture the bones from slight force and green-stick fractures are particularly prone to occur (Fig. 106). After fracture of a rickety bone union is usually delayed.

The **treatment** consists in having the child live as much as possible in the open air and sunshine. Salt-water baths are useful. Sea air is very beneficial. Fresh food (milk, cream, and meat-juice) should be ordered. Cod-liver oil, syrup of the iodid of iron, arsenic, and some form of phosphorus are to be administered. It is absolutely necessary to improve the primary assimilation. Slight deformities of the extremities require no special treatment unless they increase. If the deformity is marked or is increasing, use braces; employ massage, manipulation, and faradism. By the time the child is three years of age the bones are usually so firm that the pressure of a brace cannot cure the deformity, though the real test of brace efficiency is the degree of elasticity present in the bones, as determined by the surgeon's hands. After the age of three braces are commonly useless. Pronounced established deformities of the extremities are usually treated surgically. Kyphosis is treated by making the patient lie upon a hard bed without a pillow. The child sits up a few hours each day, the shoulders being held back and support applied to the body. In

bad cases, during the time the child is erect it should wear a brace or plaster-of-Paris jacket. Daily manipulation, the child lying prone, is helpful. Friction and electricity to the spinal muscles do good.

**Scorbutus (Scurvy).**—Many ancient writers described symptoms which must have been due to scurvy. For instance, Pliny, speaking of the army of Germanicus when near the Rhine, describes a condition of illness characterized by sore gums, falling out of teeth, and weakness of the legs. Voyagers in the fifteenth century noted symptoms which we now know were caused by scurvy. In Vasco de Gama's voyage around the Cape of Good Hope more than half of his crew perished of what was certainly scurvy. It seems to have been first specifically described early in the sixteenth century. A most graphic picture of scurvy as it used to occur at sea will be found in "A Voyage Around the World in 1740 by Lord Anson," compiled by the Rev. R. Walter, chaplain of the "Centurion." Scurvy is rare to-day in adults, but was at one time very common among those who took long voyages, or who engaged in campaigns, or were the victims of sieges, and was quite common even in cities. Poupart describes it as he saw it in the St. Louis Hospital of Paris early in the eighteenth century. Of recent years it is very uncommon, and has occurred chiefly among voyagers in the Arctic regions or those who were beleaguered. It can occur in any part of the world, on land or sea. Some years ago I saw several cases in the Philadelphia almshouse. It is important to remember that though scurvy is rare in adults, it is by no means uncommon in ill-nourished infants.

Scurvy is a constitutional malady due to the consumption of improper diet, and especially to the employment of a diet characterized by the absence of vegetables.

The use of salt meat as a staple article seems to favor the production of the disease, but scurvy can occur when there was not a salty diet, and increase of sodium chlorid in the blood is not characteristic of scurvy, but occurs in various forms of anemia. Garrod considered absence of potassium salts to be the real cause. The diminution of potassium salts is supposed to be responsible for diminished alkalinity of the blood, but we know that diminished alkalinity is common in all forms of secondary anemia. Absence of variety in diet, bad water, poorly ventilated quarters, and insufficient exercise favor the development of the disease. Some believe that an organic poison derived from tainted food is responsible (Torup). A bacterial origin has been suggested by Berthenson, Babés, and others. Certain studies made in the Transvaal suggest the bacterial origin of scurvy. Myer Coplans ("Lancet," June 18, 1904) states that it occurred in those getting excellent rations and began as inflammation of the gums, the constitutional symptoms following. If the gum condition is early recognized and cured simply by cleanliness and antiseptics, that is, by pure local treatment, constitutional trouble does not develop. Goadly ("Brit. Med. Jour.," 1910) showed that the mouth of a scurvy patient is acid to litmus, and even though there is no dental caries, in most cases there is early inflammation of the gums. Hewetson examined 400 South African males, varying in age from fifteen to thirty-five. He found 134 with scorbutic gums, although they seemed in good health ("Transvaal Medical Journal," April, 1911).

The effect of great depression of spirits in predisposing to scurvy and in aggravating cases of scurvy has often been commented on. During the siege of Breda in 1625 scurvy was rife. Bad news rapidly increased the number of cases. Good news checked it. Anson made a similar observation.

Scurvy begins with sore gums, weakness, drowsiness, muscular pains, and great susceptibility to cold. The skin is pallid or dirty white, and is occasionally mottled and often peels off. The patient is breathless on the slightest exertion. The pulse is excessively weak and slow. There is no fever unless a com-

plication arises. The gums are often tender and inflamed from the start, but in some cases they are not. After two or three weeks in all cases usually the gums are found to be tender, painful and swollen, and bleeding at frequent intervals; the breath becomes offensive, the teeth loosen and even drop out; subcutaneous hemorrhages take place, giving rise to petechiæ or extensive extravasations; the vision becomes dim; the urine becomes scanty and of low specific gravity; cutaneous vesicles form, rupture, and give rise to bleeding ulcers, and ulcers likewise arise from breaking down of blood extravasation; hemorrhages take place into and between the muscles, and in severe cases beneath the periosteum and into joints, and blood may flow from the nose, lungs, kidneys, stomach, and intestines. Deep hemorrhages are palpable as hard lumps. Bleeding at an epiphyseal line may separate the epiphysis from the shaft. If an inflammation or ulceration arises at any point, fever is observed. In many cases blood-clotting is retarded. Wright maintains that there is diminished alkalinity of blood and that scurvy is really acid intoxication. Other observers dispute this. The examination of corpuscles and hemoglobin gives a picture identical with second-

Fig. 107.—The gums in scurvy.

ary anemia. As a rule the red cells number from 3,000,000 to 4,000,000 per c.mm., but may fall to 1,000,000 or even less. Hemoglobin loss is more marked than corpuscular diminution, hence the color-index is low. There is usually an increase in leukocytes. It was observed by DeHaven, who commanded the Grinnell expedition in search of Sir John Franklin, that scurvy causes old and soundly healed wounds to ulcerate. The same observation was made years before in Lord Anson's voyage. A sailor of the "Centurion" had been wounded fifty years before at the battle of the Boyne. He developed scurvy and the old wound opened. In another case an old and soundly united fracture gave way and felt like a fresh break. Most cases of scurvy get well under proper treatment, but complete recovery is not attained for a long time. Sudden death is liable to occur if any exertion is made. The lightest exercise may be fatal. Even moving a man while he is lying down may cause death. Many cases while quiet and recumbent feel well, eat well, sleep, make no complaint of pain, and yet even slight movement may cause death. Nansen is said to attribute the loss of the gallant Scott and his companions in the Antarctic to

scurvy, the effort necessary to climb a glacier being, in his opinion, the cause of death.

**Prevention and Treatment.**—Captain Cook succeeded in preventing scurvy among his sailors by providing plenty of fresh water; guarding them against fatigue, wet, and extremes of heat and cold; attending to cleanliness and ventilation, and stimulating cheerfulness. This great navigator lost no men from scurvy. After the time of Captain Cook, the British Admiralty, acting on the suggestions of Lind and Blane, provided ships with lime-juice or lemon-juice with the most beneficial results in preventing the disease. As a matter of fact, lime-juice was suggested by John Woodall long before the time of Lind and Blane. Woodall died in 1643. Until comparatively recent years sailors of the United States Navy were accustomed to refer to sailors of the British Navy as "lime-juicers." Scurvy is prevented at the present time by employing a proper diet and by maintaining cleanliness and hygienic conditions.

The following agents are believed to be especially useful as preventives: fresh meat, lemon-juice, cider, vinegar, milk, eggs, onions, cranberries, cabbages, pickles, potatoes, and lime-juice. When the disease develops, give vinegar, lemon-juice, onions, scraped apples, cider, nitrate or citrate of potassium, whisky or brandy, and plenty of nourishing food. Lind used, with great success in many cases, $4\frac{1}{2}$ oz. of lemon-juice or lime-juice and 2 oz. of sugar in a pint of Malaga wine. This was taken during each twenty-four hours. In twenty-four hours improvement would be manifest. Antiseptic mouth-washes are necessary and strychnin is a valuable stimulant to the circulation. Sleep must be secured and ulcers are treated by antiseptic dressings and compression.

**Infantile scurvy** or **Barlow's disease** may exist alone or with rickets (*scurvy rickets*). It occurs most often in the children of the well-to-do, those who have been brought up on artificial foods, in fact, only children who are fed on artificial foods get it. Sterilized milk, condensed milk, or other artificial food may be responsible. It occurs most frequently between the eighth and eighteenth months of life. It is noted that the child has lost its appetite, is losing weight, has lost the use of its legs, lies quiet with the thighs flexed and abducted, cries when touched and when it fears it is going to be touched (Grasty, in "Amer. Jour. Obstet.," 1910). The child is anemic, suffers from gastro-intestinal disorders, spongy and bleeding gums if teeth have erupted, weakness of the legs, general muscular tenderness, night-sweats, and often febrile attacks (Rotch), bleeding from the nose, bleeding beneath the skin (blue spots), bloody urine and stools, bleeding beneath the periosteum, into joints, viscera, or muscles. In some cases hematuria is the first and perhaps the only symptom (J. Lovett Morse, "Jour. Am. Med. Assoc.," Dec. 17, 1904). A subperiosteal hemorrhage is very dense to palpation, is tender, is fusiform in outline, and does not fluctuate. It is sometimes mistaken for sarcoma. In 1 case seen by the author a hemorrhage beneath the periosteum of the femur was mistaken for a sarcoma. The limb attacked is flexed and the child will not move it. In another case hemorrhage into the knee-joint was thought to be inflammatory effusion from traumatism. Separation of an epiphysis may result from hemorrhage between it and the bone. Infantile scurvy is often unrecognized. If promptly treated, recovery is the rule, otherwise death may occur from exhaustion.

*Treatment.*—Keep the child quiet in bed and give liberal amounts of fresh and raw cows' milk and beef-juice from raw meat. Administer orange-juice, grape-juice, scraped apples, and tonics. To children over one year of age give potatoes. Antiseptic mouth-washes are necessary.

## XV. CONTUSIONS AND WOUNDS

**Contusions.**—A contusion or bruise is a subcutaneous laceration due to the application of blunt force, the skin above it being uninjured or damaged without a surface-breach and blood being effused. Punches, kicks, blows from a blackjack, etc., cause contusions. In intra-abdominal contusions the skin of the abdomen is frequently not damaged. In contusions of structures overlying a bone the skin suffers with the deeper structures. If a large vessel is ruptured hemorrhage is profuse and much blood gathers in the tissue. If only small vessels suffer hemorrhage is moderate. An *ecchymosis* is diffuse hemorrhage over a large area, the blood lying in the spaces of the subcutaneous or submucous areolar tissue. A very small ecchymosis is known as a *petechia;* a very large ecchymosis is called a *suffusion* or *extravasation.* A *hematoma* is a blood-tumor or a circumscribed hemorrhage, the blood lying in a distinct cavity in the tissue. In extremely severe contusions tissue vitality may be destroyed or so seriously impaired that gangrene follows. Suppuration rarely occurs, but occasionally does so, and is most apt to in a drunkard or a person of debilitated constitution. When hemorrhage arises in the tissues after a contusing force it soon ceases unless a very considerable vessel is ruptured. The arrest of hemorrhage is brought about by the resistance of the tissues, the contraction and retraction of the vessels, coagulation of blood, and in some cases of severe injury coagulation is favored by syncope. Blood in the tissues, as a rule, soon coagulates, the fluid elements being absorbed and the red corpuscles breaking up and setting free pigment, which pigment may be carried away from the seat of injury or may crystallize and remain there as hematoidin. In some cases inflammation occurs about the extravasated blood, a capsule of fibrous tissue being formed, and the blood being slowly absorbed or the fluid elements remaining unabsorbed (*blood-cyst*), or the blood becoming thicker and thicker, finally calcifying. Blood in serous sacs (joints, pleura, pericardium) coagulates very slowly. As blood is being absorbed it undergoes chemical changes and color changes ensue, the part being at first red and then becoming purple, black, green, lemon, and citron. The stain following a contusion is most marked in the most dependent area. After a bruise of the periosteum a blood-clot forms, much tissue-induration occurs, and a hard edge can be detected by palpation at the margin of the clot.

**Symptoms.**—The symptoms are tenderness, swelling, and numbness, followed by some aching pain or a feeling of soreness. The pain rarely persists beyond the first twenty-four hours. Cutaneous discoloration appears quickly in superficial contusions, but only after days in deep ones. In some regions—the scalp, for instance—it can scarcely be detected; in others, as in the eyelid and vulva, discoloration is early, widespread, and marked. Discoloration and swelling are very marked in regions where loose cellular tissue abounds (eyelids, prepuce, scrotum). The discoloration is at first red, and becomes successively purple, black, green, lemon, and citron. The swelling is primarily due to blood, and is added to by inflammatory exudation. In a more severe contusion a hematoma may form. A recent hematoma fluctuates, but gradually, because of cell-proliferation, the edge becomes hard, but the center continues to fluctuate. The mass gradually grows smaller and finally disappears. A subperiosteal hematoma of the scalp may be mistaken for depressed fracture of the skull. Any form of hematoma of the scalp may be mistaken for an abscess, but differs from it in the absence of inflammatory signs. It occasionally, though rarely, suppurates. In a case in which suppuration occurs an abrasion, which may be very minute, often exists on the skin. In any severe contusion there is considerable and possibly grave, or even fatal, shock.

**Treatment.**—In a severe injury bring about reaction from the shock. Local treatment consists in rest, elevation, and compression to arrest bleeding, antagonize inflammation, and control swelling. Cold is useful early in most cases, but it is not suited to very severe contusions nor to contusions in the debilitated or aged, as in such cases it may cause gangrene. In very severe contusions employ heat and stimulation. When inflammation is subsiding after a contusion, compression and inunctions of ichthyol should be employed. If the amount of blood is very large, massage must not be used because it may cause embolism or fat-embolism. Massage and passive motion are imperatively needed after contusion of a joint. If a distinct cavity exists, aspiration or incision lessens the danger of fat-embolism. A contusion should never be incised unless the amount of blood is large and a distinct cavity exists, or hemorrhage continues, or infection takes place, or a lump remains for some weeks, or gangrene is threatened. For persistent bleeding freely lay open the contused area, turn out clots, ligate vessels, insert drainage-strands or a tube, and close the wound. If gangrene is feared, make incisions and apply heat to the part. If a slough forms, employ antiseptic fomentations. The constitutional treatment for contusion, after the patient has reacted from shock, is the same as that for inflammation. (See Abdomen, etc.)

**Wounds.**—A wound is a breach of surface continuity effected by a sudden mechanical force. Wounds are divided into open and subcutaneous, septic and aseptic, incised, contused, lacerated, punctured, gunshot, stab, and poisoned wounds.

The **local phenomena of wounds** are pain, hemorrhage, loss of function, and gaping or retraction of edges.

*Pain* is due to the injury of nerves, and it varies according to the situation and the nature of the injury. It is influenced by temperament, excitement, and preoccupation. It may not be felt at all at the time of injury. At first it is usually acute, becoming later dull and aching. In an aseptic wound the pain usually remains slight, but in an infected wound it always becomes severe.

The nature and amount of *hemorrhage* vary with the state of the system, the vascularity of the part, and the variety of injury.

*Loss of function* depends on the situation and extent of the injury.

*Gaping* or *retraction of edges* is due to tissue elasticity, and varies according to the tissues injured and the direction, nature, and extent of the wound.

The **constitutional condition** after a severe injury is a state known as *shock*.

**Shock.**—The name "shock" was introduced in 1795 by James Latta to designate the condition ensuing upon severe injury. (See G. C. Kinnaman, in "Annals of Surg.," Dec., 1903.) Shock is a depression of the vital powers arising from an injury or a profound emotion acting on the nerve-centers. No theory of shock is entirely satisfactory. Most observers state that there is exhaustion or inhibition of the vasomotor mechanism. Exhaustion is gradually induced; inhibition is suddenly produced. It is supposed that by overstimulation of sensory nerves violent impressions are conveyed to the nerve-centers, the vasomotor center is exhausted or inhibited, vasoconstrictor power is lost, the peripheral arteries and capillaries are palsied and are depleted or nearly emptied of blood, and the blood is largely transferred to the veins. The blood-pressure is lowered, the cardiac action is impaired, the respiratory action is impeded, and quantities of dark-colored blood gather in the somatic veins, but especially in the veins of the splanchnic area. (See the masterly study of "Surgical Shock," by Crile.) Although this theory finds wide acceptance, some able investigators do not accept it. In shock the abdominal veins are greatly distended and the other veins of the body may also be overfull, the

arteries contain less blood than normal, and an insufficient amount of blood is sent to the heart and to the vital centers in the brain. In other words, in shock there is a deficiency in the circulating blood. The term *collapse* is used by some to designate a severe condition of shock, and is employed by others as a name for a condition produced by functional depression of the vasomotor center the result of mental disturbance, cardiac failure, respiratory failure, or vasomotor insufficiency, rather than of physical injury. Crile regards collapse as inhibition of the vasomotor center, in contrast to shock, which is exhaustion of the center. As a matter of fact, shock and collapse are often both present. That the bombardment of the nerve-centers by a tumult of peripheral impressions causes shock is shown by the fact that if the nerves from a part are thoroughly cocainized so that they will not transmit sensation, operation upon the part produces practically no shock. Crile calls such cocainization the introduction of a *physiological block*. Again, Crile insists that in shock there are demonstrable changes in the brain cells. The changes consist in shrinking of nuclei and dissipation of the granular matter of the protoplasm. Such changes mean that the cells involuntarily discharged nerve force many times, were caused to discharge by impressions brought to them, and by the discharges were exhausted of certain necessary chemical materials. Shock may be slight and transient, it may be severe and prolonged, it is usually sudden in onset, but may come on gradually, and it may even produce almost instant death. I agree with Bloodgood that even a violent injury does not of necessity at once produce it. Every now and then we see a man with a crushed limb who does not exhibit shock, the condition gradually coming on from pain, terror, etc., and being aggravated perhaps by hemorrhage. During an operation if shock arises it is apt to do so gradually, but this is not always the case, for sometimes it comes on with great suddenness, for instance, when traction is made upon the pedicle of the spleen, when the bone of the thigh is sawed through, or when there is a burst of blood from a large vessel. Sudden death from shock is probably due to reflex stimulation of the pneumogastric nuclei and arrest of cardiac action. It is known as *death by inhibition*. Shock is more severe in women than in men, in the nervous and sanguine than in the lymphatic, in those weakened by suffering than in those who are strangers to illness. It is predisposed in disease of the kidneys, diabetes, chronic cardiac disease, and alcoholism. Fear is probably a great factor in shock. Injuries of nerves, of brain, of the intrathoracic viscera, of the intra-abdominal viscera, of the urethra, or of the testicle produce extreme shock. Anything which extracts the body-heat favors the development of shock (exposure to cold air, insufficient covering, chilling the body by solutions or wet towels). In cerebral concussion there is shock plus other conditions. Sudden and profuse hemorrhage greatly aggravates shock. Prolonged anesthetization causes shock. Great shock may occur after the removal of a large tumor or a quantity of fluid from the abdomen. In such a case shock is brought about by the sudden removal of pressure and the consequent rapid distention of intra-abdominal veins. Exposure of tissue and vital parts to air aggravates shock. Crile lays down as the most important causes of shock: fear, pain, and traction.

The influences which cause cells to exhaust themselves by discharge are recognized by the nerve-cells as meaning or suggesting harm to the organism. Such influences cause impulses to escape. Such influences are called by Crile *nocuous* or *noci influences*. Fear itself can cause shock, and fear is a factor even when shock is apparently due to pain or traction. Fear, then, is the ever-existing factor in shock causation. It is often a conscious fear. It may be subconscious fear.

Crile maintains that an individual may be anesthetized by ether or chloroform and feel no pain, and yet noci influences may cause shock. The influences

then act by suggesting painful or harmful things to the nerve-cells, that is, by causing subconscious fear. Crile calls such automatic association *philogenetic association*.

The **symptoms** of ordinary shock (*torpid* or *apathetic shock*) are subnormal temperature; weakness of heart action; irregular, small, weak, rapid, and compressible pulse; cold, pallid, bloodless, and often clammy or profusely perspiring skin; shallow, irregular, and often gasping respiration. A sphygmomanometer will indicate a notable fall in blood-pressure. Consciousness is usually maintained, but there is an absence of mental originating power, the injured person answering when spoken to, but volunteering no statements and lying with partly closed lids and expressionless countenance in any position in which he may be placed. The answers to questions though apparently intelligent are utterly unreliable. There is great motor weakness. The pupils are dilated and react but slowly to light. The sphincters are relaxed. Pain is slightly or not at all appreciated. Nausea is absent and vomiting may, as in concussion, presage reaction. Gastric regurgitation, after a considerable duration of shock, is not unusual, and is a bad omen. It must impress students of physiology that certain of the well-known signs of shock do not seem to indicate that there are paralysis and dilatation of the peripheral vessels, as the popular theory of shock supposes. The signs which do not co-ordinate with the theory are anemia of the surface, lowered temperature of the surface, and small pulse (Seelig and Lyon, in "Surgery, Gynecology, and Obstetrics," Aug., 1910). Shock is not rarely followed by suppression of urine. Whereas the victim of shock is usually stupid and indifferent, he may become delirious. If delirium arises, the condition is very grave. Travers called shock with delirium *erethistic* or *delirious shock*. As a matter of fact, such a state is not genuine shock, but is either a traumatic or a toxic delirium added to or following upon shock. It is usually due to uremia or sepsis. Delirious shock may arise after a person has been bitten by a poisonous snake. Many years ago Travers described a *secondary* or *delayed form* of shock, which comes on several hours after an injury or violent emotional disturbance. This form of shock is seen not unusually in those who have passed through a railroad accident. It may be a sign of hemorrhage, and is sometimes met with after the administration of ether or chloroform. The statements made by a person who has recovered from a severe shock are always unreliable as to events which occurred while shock existed, and are often doubtful as to the details of the accident. Not unusually the memory of the accident is perverted or even destroyed.

**Diagnosis.**—Concealed hemorrhage is difficult to differentiate from shock. The former produces impairment of vision (retinal anemia), irregular tossing, frequent yawning, great thirst, nausea, and sometimes convulsions. In shock the hemoglobin is unaltered; in hemorrhage it is enormously reduced (Hare and Martin). In hemorrhage recurrent attacks of syncope are met with. In pure shock such attacks do not occur. In concealed hemorrhage the abdomen may exhibit physical signs of a rapidly increasing collection of fluid. Shock and hemorrhage are often associated. A usual characteristic of shock is rapid onset, which separates it distinctly from exhaustion. It arises at a much earlier period after an injury than does fat-embolism.

**The Prevention of Shock in Operations.**—Examine the patient with care before operating, giving special attention to the condition of the kidneys. The amount of urine passed and the amount of urea it contains should always de determined when possible. The amount of urea should be estimated from the twenty-four-hour urine. The normal amount of urine in the twenty-four hours is about 50 ounces and the normal amount of urea 2 per cent. Less urea is significant of danger from shock and subsequent kidney complications. If the condition of the patient leads us to fear that there will be dan-

gerous shock, do not purge him severely before operation, and just previous to operation give a rectal injection of hot saline fluid and a hypodermatic injection of $\frac{1}{100}$ gr. of atropin. It is also a good plan in some cases to give a hypodermatic injection of $\frac{1}{8}$ gr. of morphin twenty minutes before operation. It tranquillizes the patient and less ether will be needed to anesthetize him. Examine the patient thoroughly and prepare him carefully beforehand and decide if he should take a general anesthetic at all, and if so, which one. Nitrous oxid diminishes operative shock, Crile says, because cell activity is lessened by deficiency of oxygen.

Many operators believe that ether and chloroform lessen shock. Crile disputes this. He holds that they merely inhibit conscious fear and muscular response to peripheral impressions, but do not save the brain-cells from noci impressions, and hence do not keep cells from exhausting discharges.

It would seem that a drugged brain-cell could not receive influences as acutely and strongly as one untouched by ether or chloroform. I believe that a general anesthetic, given properly and in proper amount, does, to some extent at least, lessen the shock of an operation.

In some cases a local anesthetic should be used, for instance, some cases of typhoid perforation and strangulated hernia. The nerves well above the area of operation should be infiltrated with cocain. This prevents the ascent of peripheral impressions, makes what Crile calls a "physiological block," and so prevents shock. After this infiltration a limb can be amputated below the infiltrated area without pain and without great depression of the vital powers. I have performed a number of amputations most satisfactorily relying for anesthesia purely upon cocainization of the nerves. In these cases there has been very little shock. This is a valuable procedure even when ether is given. I employ it frequently and am satisfied of its great value in preventing shock. The ether prevents conscious fear during the operation, the cocainization of the nerve intercepts harmful impressions in their ascent. Thus is shock prevented. In some few cases in which we fear shock spinal anesthesia is used; in others, scopolamin and morphin. If a general anesthetic is used it must be skilfully given and not a drop is given beyond the amount necessary to maintain thorough anesthesia. Cover every part but the field of operation with hot blankets and put cans of hot water about the patient, or put him on a bed composed of hot-water pipes covered with blankets. Prevent bleeding with the greatest possible care. Operate as rapidly as is consistent with safety and thoroughness. The blood-pressure is of great importance in estimating the degree of shock, and any sudden fall of blood-pressure is ominous. It is a custom with many operators to fix a sphygmomanometer to the arm and have an assistant watch the scale constantly during an operation. If shock develops during an operation hasten on the work, lessen the amount of ether, and apply active treatment. Return the patient to bed as soon as possible and without exposure in cold halls or a windy elevator. Occasionally it becomes necessary to suspend an operation in order to prevent death on the table.

Crile's **anoci association operation** is founded on the prevention of shock by the exclusion as far as possible of all painful, terrifying, and depressing influences from a patient before, during, and after operation. The patient is not kept for days or even for hours waiting in fear. He is to be reassured, made confident, dominated by his surgeon. He is anesthetized by means of nitrous oxid and nitrogen, ether being added only if necessary. The tissues are infiltrated with a local anesthetic. During the operation all noci impressions are carefully excluded. This method, in the hands of Crile, Bloodgood, and others, has given highly satisfactory results.

**Treatment.**—In treating ordinary apathetic shock raise the feet and lower the head, unless this position causes cyanosis. At least place the head flat and

the body recumbent. Wrap the patient in hot blankets and surround him with hot bottles, hot bricks, hot-water bags, or cans of hot water. Always wrap a can, a bottle, or a bag in flannel or some other material to avoid burning the patient. Ordinary stimulants seem of little or no value and drugs given by the stomach are not absorbed. Salt solution may be thrown into a vein (*intravenous infusion*), may be given by the rectum (*proctoclysis*), or subcutaneously (*hypodermoclysis*). Intravenous infusion does good, but, unfortunately, the benefit is very temporary except in cases associated with hemorrhage. In hemorrhage it should always be given, and it should be given mixed with adrenalin chlorid (1 teaspoonful of the 1 : 1000 solution of adrenalin chlorid is added to 1 liter of salt solution). The operation of intravenous infusion is described on page 465. The custom of giving salt solution in a vein has become so common that resident physicians are apt to resort to it as a routine. It is to be remembered that if given rapidly or in too great quantity it may gather in the chambers of a dilated right heart and arrest a heart so weakened that it has almost reached its limit of function. I am satisfied that the rapid administration of salt solution intravenously is responsible for some deaths. Crile maintains that the only way "to increase and sustain the blood-pressure when the vasomotor center is exhausted" is to "create a peripheral resistance either by a drug acting on the blood-vessels themselves or by mechanical pressure."[1] In order to accomplish this he uses adrenalin chlorid. Because of the rapidity with which this drug is oxidized he gives it intravenously, slowly and continuously from a buret, using a solution of a strength of from 1 : 50,000 to 1 : 100,000 in salt solution. The rate of flow should be "controlled by a screw-cock attached to the rubber tube." Crile also places the patient in a rubber suit and distends the suit by means of an air pump and thus obtains equable pressure upon the cutaneous surface and an increase of peripheral vascular resistance. The difficulty with giving the solution in a vein is, the drug first comes in contact with "the vessels having the least power of influencing blood-pressure," and before a notable rise can be affected by arterial action "it is necessary that the solution should pass through the right heart, the lungs on its way to the aorta, then finally affecting the coronary arteries" (Crile, in "Am. Jour. Med. Sciences," Jan., 1909). The best way to use adrenalin solution in severe shock is to introduce it as Crile now advises, that is, into the arterial system and toward the heart. Occasionally, by this means, resuscitation from apparent death may be accomplished. Crile calls this method *centripetal arterial transfusion*. It is applied as follows ("Am. Jour. Med. Sciences," April, 1909):

"In human resuscitation the technic is as follows: The patient in the prone posture is subjected at once to rapid rhythmic pressure upon the chest, with one hand on each side of the sternum. This pressure produces artificial respiration and a moderate artificial circulation. A cannula is inserted toward the heart into an artery. Normal saline, Ringer's, or Locke's solution, or, in their absence, sterile water, or, in extremity, even tap water, is infused by means of a funnel and rubber tubing. But as soon as the flow has begun the rubber tubing near the cannula is pierced with the needle of a hypodermatic syringe loaded with 1 : 1000 adrenalin chlorid and 15 to 30 min. are at once injected. Repeat the injection in a minute if needed. Synchronously with the injection of the adrenalin the rhythmic pressure upon the thorax is brought to a maximum. The resulting artificial circulation distributes the adrenalin that spreads its stimulating contact with the arteries, bringing a wave of powerful contractions and producing a rising arterial, hence coronary, pressure. When the coronary pressure rises to, say, 40 mm. or more, the heart is likely to spring into action. The first result of such action is to spread still further the blood-pressure-raising adrenalin, causing a further and vigorous rise in blood-pres-

[1] George Crile, in "Boston Med. and Surg. Jour.," March 5, 1903.

sure, possibly even doubling the normal. The excessively high pressure is most favorable to the resuscitation of tissue, especially of the central nervous system (Stewart). Just as soon as the heart-beat is established the cannula should be withdrawn, first, because it is no longer needed, and second, because the rising blood-pressure will drive a torrent of blood into the tube and funnel. Unless there has been hemorrhage, the only object in the use of saline infusion is to serve as a means of introducing the adrenalin into the arterial circulation toward the heart. Bandaging the extremities and abdomen tightly over masses of cotton is very useful."

In prolonged shock direct transfusion of blood should be employed (see page 463). The use of hot and stimulating rectal enemata is important. The

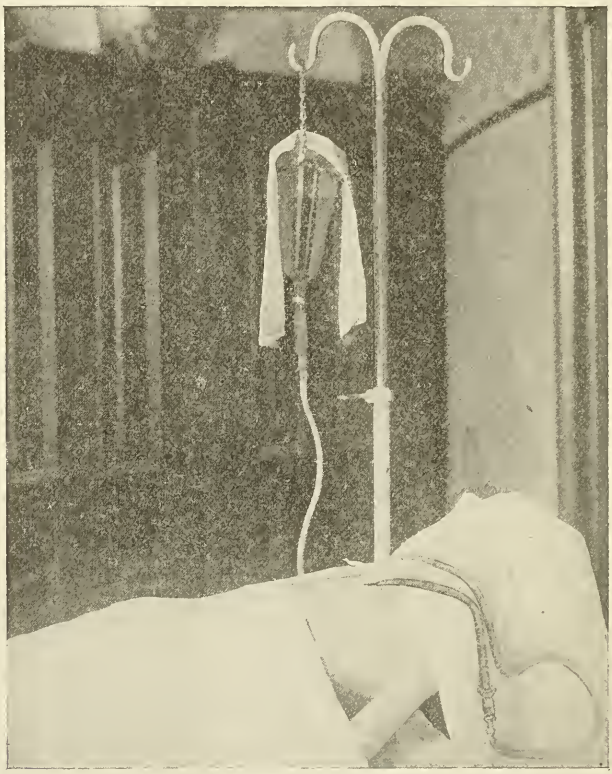


Fig. 108.—Subcutaneous saline infusion (Senn).

rectum may absorb fluids when the stomach refuses to do so. Enemata of hot normal salt solution are beneficial (*proctoclysis*). The tube is carried as high as possible and the injection is introduced so as to distend the colon. *Hypodermoclysis* is given as follows: Insert an aspirator tube into the cellular tissue of the loin, scapular region, or under the mamma, cleansing the part first. The tube is attached to a fountain syringe, which is filled with warm normal salt solution, and is hung at a height of 2 or 3 feet above the bed (Fig. 108). In an hour's time a pint or more of fluid will enter the tissue and be absorbed. It is the custom to give hypodermatic injections of ether, brandy, strychnin, digitalis or atropin, or inhalations of amyl nitrite. Crile has demonstrated experimentally that strychnin is perfectly futile in pure shock and may actually aggravate the condition. In collapse it is of some value. We believe this

statement is true clinically. Strychnin goads a heart to increased action when that organ has not sufficient blood passing into it to enable it to firmly and strongly contract. The use of strychnin in shock has been compared by Hare to beating a dying horse to make it pull. I believe that atropin is of great benefit in shock, especially if the skin is very moist. This drug, according to my colleague, Prof. Hobart A Hare, is a sedative to the vagus; but what makes it particularly valuable is that it acts upon the vasomotor system, combats the dilatation of the blood-vessels, maintains vascular tone, opposes stagnation of blood in any vessels, and increases the amount of moving blood. If the skin is very moist, atropin is particularly indicated. Senn recommends the hypodermatic injection of sterile camphorated oil, a syringeful every fifteen minutes, until reaction begins. Inhalation of oxygen is often of much service, and artificial respiration may be necessary. Opiates are contra-indicated in shock. Mustard plasters should be placed over the heart, spine, and shins. A turpentine enema is useful. An enema of hot coffee and whisky is valuable. In severe cases of shock, bandage the extremities. Bandaging for the relief of shock is called *autotransfusion*. This procedure increases peripheral resistance and enables the body to utilize to the best advantage the small amount of circulating blood, and sends most of it to the brain, where it will maintain the activity of the vital centers and keep up circulation and respiration. For this purpose ordinary muslin bandages may be used, or gauze bandages, or the bandages of Esmarch. Crile's rubber suit accomplishes the object more satisfactorily than does bandaging the extremities. Abdominal massage helps drive out the imprisoned blood, and after massage sets free the abdominal blood apply a compress and binder. In serious cases artificial respiration and stimulation of the diaphragm with a galvanic current may be used. If shock comes on during an operation, the operation must be hurried or even abandoned, and proper treatment must be instituted at once. The anesthetist should give very little ether when shock becomes at all evident. Should we operate during shock? We should only do so when death without instant operation is inevitable. We must operate, if it is necessary to do so, to arrest hemorrhage, to relieve strangulated hernia, intestinal obstruction, obstruction of the air-passages, compound fractures of the skull, extravasated urine, or intraperitoneal extravasations from ruptured viscera. If hemorrhage can be temporarily controlled by pressure or a clamp, so much the better, and the permanent arrest can be effected after the reaction from shock. It is not wise, in the author's opinion, to amputate during shock. A tourniquet or Esmarch bandage should be applied, and attempts be made to bring about reaction, and when reaction is obtained the amputation should be performed. It is only just to say that some eminent surgeons oppose this rule. Roswell Park says that "shock is often alleviated by the prompt removal of mutilated limbs which, when still adherent to the trunk, seem to perpetuate the condition." The same teacher believes in operating at once upon severe compound fractures.[1] After every operation keep careful watch upon the amount of urine passed, see to it that the patient takes sufficient fluid, and if the urine becomes scanty put a hot-water bag to each loin, give diuretics by the mouth, secure cutaneous activity, give saline purgatives, and administer hot saline enemata. If the condition is not soon benefited, the custom is to infuse hot saline fluid into a vein. I am doubtful if intravenous infusion of saline fluid is beneficial in suppression, and I even fear it may do harm (see the studies of Widal, Marie and Crouzon, Merklen, and others). Rössle ("Centralbl. f. Chir.," 1907, xxxiv) says that in certain cardiac and renal conditions salt solution damages the capillaries and does actual harm. In urinary suppression following acci-

[1] Park's "Surgery by American Authors."

dent or surgical operation (*postoperative suppression* or *anuria*) the condition is so dreadfully grave that it is justifiable to expose each kidney and split the capsule in order to relieve tension and in the hope of thus abating congestion. In fact, I believe this should always be done. In a case in which there had been total suppression for three days I did this operation. During the next thirty-six hours the patient passed 12 oz. of urine, but died of complications. *Postoperative suppression of urine* is almost invariably fatal. Delayed shock is treated in the same manner as apathetic shock if hemorrhage can be excluded. If hemorrhage is the cause, the bleeding must be arrested, and blood be transfused, or saline fluid be infused into a vein. If delirious shock is due to sepsis, the treatment is that of sepsis. If it is a nervous delirium, give morphin and other sedatives. If due to uremia, the treatment is obvious.

**Fat-embolism.**—(See page 191.)

**Fever.**—(See Fevers, page 128.)

**Treatment of Wounds.**—All wounds, other than those made by the surgeon, are regarded as infected. The rules for treating such wounds are: (1) arrest hemorrhage; (2) bring about reaction; (3) remove foreign bodies; (4) asepticize; (5) drain, coaptate the edges, and dress; and (6) secure rest to the part and combat overaction of the tissues. Constitutionally, allay pain, secure sleep, maintain the nutrition, and treat inflammatory conditions.

*Arrest of Hemorrhage.*—To arrest hemorrhage the bleeding point must be controlled by an Esmarch band or digital pressure until ready to be grasped with forceps; it is then caught up and tied with catgut or aseptic silk. Slight hemorrhage ceases spontaneously on exposure of the bleeding point to air, and moderate hemorrhage ceases permanently after the temporary application of a clamp. An injured vessel when not of the smallest size must be ligated, even if it has ceased to bleed. Capillary oozing is checked by hot water and compression. If a large artery is divided in a limb, apply a tourniquet before ligating. (See Wounds of Vessels.)

*Bringing About of Reaction.*—(See Shock.)

*Removal of Foreign Bodies.*—Remove all foreign bodies visible to the eye (splinters, bits of glass, portions of clothing, gun-wadding, grains of dirt, etc.) by forceps and a stream of corrosive sublimate solution, sterile water, or normal salt solution. In a lacerated or contused wound portions of tissue injured beyond repair should be regarded as foreign bodies and be removed with scissors.

*Cleaning the Wound.*[1]—To clean the wound shave the surrounding area, if it is hairy; scrub the surface about the wound with ethereal soap, green soap, or castile soap; wash with water, scrub with alcohol, and then with corrosive sublimate solution (1 : 1000). An accidental wound is infected, and must be well washed out with an antiseptic solution. In every wound in which we have reason to suspect tetanus infection a preventive dose of antitoxin should be given. We have particular occasion to apprehend tetanus if the wound is contaminated with feces, street dirt, stable dust, or stable refuse, or if it was infected with a toy pistol such as boys use to celebrate the Fourth of July. A clean wound made by the surgeon need not be irrigated; in fact, irrigation with an antiseptic fluid leads to necrosis of tissue, causes a profuse flow of serum, and necessitates drainage. If clots have gathered in a wound they must be removed, as their presence will prevent accurate coaptation of the edges. In an infected wound they are washed out by a stream of corrosive sublimate solution. In a clean wound they are washed out by hot salt solution. If dirt is ground into a wound, as is often seen in crushes, pour sweet oil into the wound, rub it into the tissues, and scrub the wound with ethereal soap. The oil entangles the dirt, and the soap and water remove both oil and dirt. After the rough cleansing irrigate with corrosive sublimate solution. In

[1] The use of iodin as a germicide is discussed on page 68.

some cases, especially in bone injuries, it is necessary to scrape the wound with a curet. If a fissure of the skull is infected, enlarge the fissure with a chisel in order to clean it. In a badly infected wound one of the most valuable agents for use in producing disinfection is pure carbolic acid. After cleaning the wound, it is necessary in certain regions to examine in order to determine if tendons or considerable nerves have been cut. If such structures have been divided, they must be sutured with fine silk, chromic gut, or kangaroo-tendon.

*Drainage, Closure, and Dressing.*—Superficial wounds require no special drainage, as some wound-fluid will find exit between the stitches and the rest will be absorbed. A large or deep wound requires free drainage for at least twenty-four hours by means of a tube, strands of horsehair, silk or catgut, or bits of iodoform gauze. An infected wound must be drained invariably. Good drainage may, to a considerable extent, compensate for imperfect antisepsis. If capillary drains are employed, apply a moist dressing. Otherwise the strands dry and fail to act as drains. Approximate the edges with interrupted sutures of silk or silkworm-gut if the wound is deep and considerable tension is inevitable. Catgut is used for superficial wounds and for those where tension is slight. If there is decided tension, silver wire may be used. In very deep wounds buried sutures must be used. These sutures may consist of absorbable material (kangaroo-tendon or catgut) or unabsorbable material (silver wire) or very fine silk. Of late I have been following the Johns Hopkins custom and have closed all clean wounds with sutures of very fine iron dyed silk passed by small and very sharp sewing needles. By the use of these fine sutures a minimum amount of tissue necrosis occurs, the risk of infection is greatly lessened, and the resulting scar is the smallest that can be obtained. As is well known, tight sutures cause tissue necrosis and hence predispose to infection. It is impossible to tie the fine black silk very tight, because if we do so it breaks. It requires considerable practice to learn to tie the sutures without breaking. This fine silk can be buried without fear, as it never causes a sinus, and it is used for layer sutures with perfect confidence. I learned this plan from Dr. Harvey Cushing, and am much pleased with it. If the wound is infected, dress with warm, moist antiseptic gauze. If it is not infected, dress with dry sterile gauze. The custom once was to cover even dry gauze with a rubber-dam to diffuse the fluids, but we now prefer to omit the rubber-dam and use plentiful dressings. A dry dressing absorbs wound-fluids quickly and is less likely to become infected. Change the dressings in twenty-four hours or sooner if they become soaked with discharge. Dressings are changed for cause, but not according to scheduled time. They must, of course, be changed when they become soaked with wound-fluid, and soaking may occur in a few hours, but may not occur for days. As long as the temperature remains normal and the wound free from pain, if the dressing is not wet with discharge, it can be left in place unless removal is necessary to take out a drainage-tube. If pus forms, open the wound at once. Many surgeons sprinkle wounds before approximation and wound surfaces after approximation with a drying powder. These powders are of great use in infected wounds, but are not necessary in clean wounds. Among the substances employed are salicylic acid, boric acid, calomel, acetanilid, aristol, iodoform, subiodid of bismuth, and glutol. In large wounds which cannot be approximated it is occasionally advisable to skin-graft by Thiersch's method. A small wound which cannot be sutured is dusted with an antiseptic powder and dressed. A granulating wound is dressed as is a healing ulcer. In recent infected wounds rest associated with Bier's treatment comprise the means of local treatment. Incision is usually required. In later infections or severe infections the wound must be opened widely and drained.

A sloughing wound is opened, is dusted with iodoform or acetanilid, and is dressed with hot antiseptic fomentations.

*Rest.*—Severe wounds require the confinement of the patient to bed. Bandages, splints, etc., are used to secure rest. In a closed wound rest need not be protracted, in fact, our former custom was to insist upon it for too long a period. The slight irritation of moderate motion stimulates repair. We no longer feel it necessary to keep laparotomy cases in bed for three full weeks, but we get them up in from seven to ten days. By doing this we secure just as firm a cicatrix, greatly lessen the annoyance from constipation and flatulence, and diminish notably the number of cases of postoperative pneumonia and phlebitis. I do not, however, advocate getting such patients up in twenty-

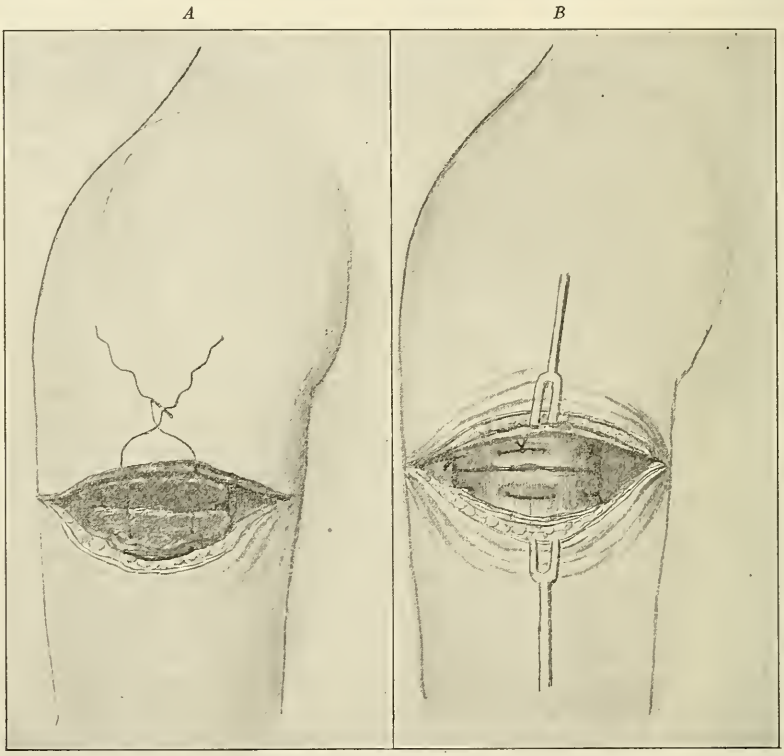



Fig. 109.—Muscle suture: *A*, Transverse wound of biceps muscle, showing marked retraction of muscle-ends and mattress suture in place; *B*, muscle suture completed (Senn).

four to thirty-six hours, as is advised by some surgeons. A patient with an infected wound or an open drainage wound should be confined to bed. The methods of combating inflammation have previously been set forth.

*Constitutional Treatment.*—Bring about reaction from depression, but prevent undue reaction. Feed the patient well, stimulate him if necessary, attend to the bowels and bladder, secure sleep, and allay pain. Watch for complications, namely, inflammation, suppuration, gangrene, tetanus, erysipelas, suppression of urine, and pneumonia. Observe the temperature closely; it may be a danger-signal of urgent importance.

**Incised Wounds.**—An incised wound is a clean *cut* inflicted by an edged instrument. Only a thin film of tissue is so devitalized that it must die. These wounds have the best possible chance of union by first intention.

The pain may be very severe; but if the instrument is sharp and used quickly it may be trivial. The pain is less severe than that caused by some other varieties of wounds. The acute pain does not last long, and is followed by smarting. The hemorrhage is profuse, varying, of course, with the region cut. Bleeding from the scalp is violent, because there are numerous vessels which lie in fibrous tissue and cannot retract nor contract. The edges of incised wounds retract because of tissue elasticity, and the wound "gapes." If the skin or fasciæ are divided at a right angle to the muscle beneath, there is wide gaping. If the cut is parallel to the muscle-fibers, the gaping is slight.

When the skin is violently pulled upon, it tends to split in a certain line. Langer and Kocher speak of this as the line of cleavage, and point out the direction of these lines in various situations. A cut across the line of cleavage is followed by wide gaping. A cut in the direction of the line of cleavage produces slight gaping, and is followed by a trivial scar.

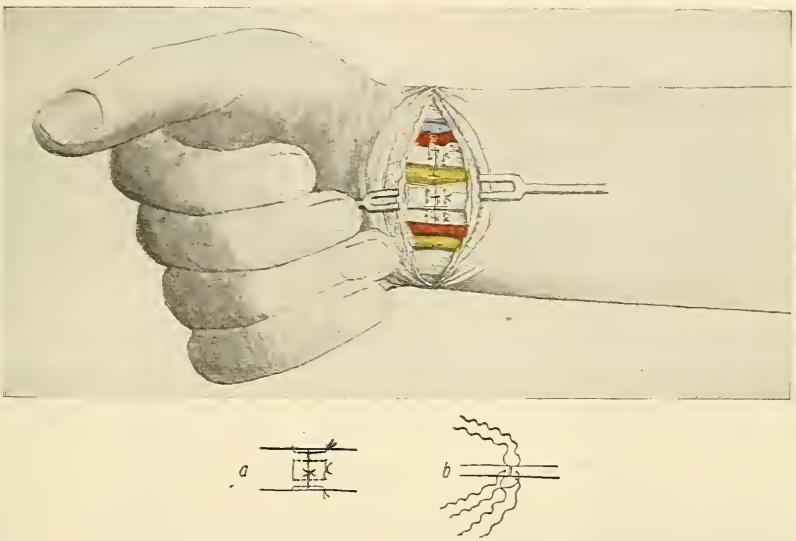



Fig. 110.—Suturing of tendons and nerves in incised wounds: *a*, Primary tendon suture; *b*, primary nerve suture (Senn).

When a muscle is cut across the wound edges widely separate. When a tendon is completely cut across extensive separation occurs.

An incised wound can be thoroughly inspected, all divided structures can be identified, foreign bodies can be easily removed, and disinfection can be satisfactorily carried out.

*Treatment.*—According to general principles. Arrest hemorrhage, asepticize, etc.

Examine the wound carefully to see if a nerve, a tendon, or a muscle is divided, and if such injury is discovered, suture at once (Figs. 109 and 110). If the wound is extensive or deep, it may be necessary to use buried sutures in order to keep the sides of the wound in contact. If the surface of a wound is approximated, but the depths are not, the dead space or cavity becomes filled with fluid, and infection almost certainly occurs. If buried sutures have not been used, such a cavity must be obliterated by the judicious application of pressure upon the surface. This is secured by the adaptation of a mass of loose or fluffed-up gauze, and the firm application of a bandage or binder. An

incised wound is usually closed with interrupted sutures (Figs. 111 and 112). In adjusting the sutures, see that the edges of the wound are not inverted, but are neatly approximated with raw edge to raw edge. Tie the stitches firmly but not tightly. If a stitch is tied too tightly it will make a ridge, as shown

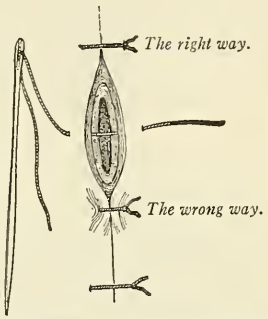



Fig. 111.—The interrupted suture (after Bryant).

Fig. 112.—Tying an interrupted suture. The knot is placed to the *side* of the wound as shown in Fig. 109.

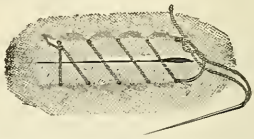


Fig. 113.—Continuous suture.

in Fig. 111, and undue tightness is sure to cause necrosis, and is often productive of a stitch-abscess. As previously stated, I usually close wounds with sutures of very fine black silk. This will break if we try to tie it tightly, and as it never causes a sinus when retained in the tissues it can be used for buried

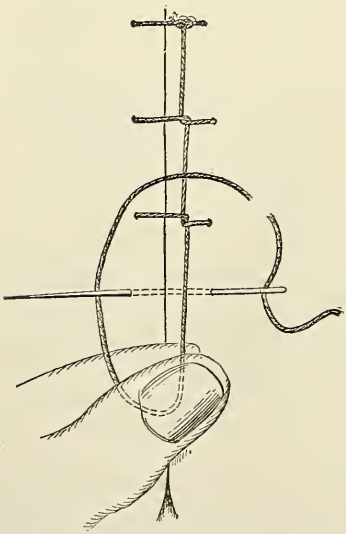


Fig. 114.—Ford's suture: A square knot, a single knot, a double or friction knot, and the first method of passing the needle to tie a single knot immediately.

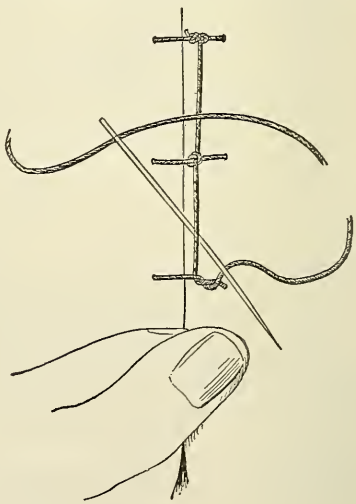


Fig. 115.—Ford's suture: Showing two square knots, a single knot, and the method of completing a square knot.

sutures as well as for the skin. A silk suture and catgut suture should be tied with the reef knot; a suture of silkworm-gut may be tied with either a surgeon's knot or a reef knot. If a wound is on the face, particular care must be employed in closing it, in order to limit the amount of disfigurement. Fine silk sutures are passed with a small sharp needle or a subcuticular stitch is used.

In a clean wound stitches may, as a rule, be removed in from six to eight days. In a large wound one-half the stitches are removed at one sitting, and in a day or two the rest are removed. Stitches are promptly removed if they begin to cut out or if infection occurs.

The old continued suture is rarely used for skin-wounds at the present time. This suture is employed to suture the dura after division, to suture the two layers of pleura together before an abscess of the lung is opened, to suture the peritoneum after laparotomy, and to suture the mucous membrane after certain operations upon the stomach. The continuous suture is shown in Fig. 113. A continuous suture knotted after each emergence was devised by Ford. It is very useful in suturing the parietal peritoneum (Figs. 114 and 115.)

Halsted's subcuticular stitch (Fig. 116) makes a most perfect closure of the skin-wound, and is followed by the smallest possible scar. In closing a deep wound the muscles and fasciæ are sutured in layers by buried sutures before the subcuticular stitch is inserted. It is only used in wounds which are almost certainly clean (as those made by the surgeon), and in wounds which do not require drainage. The suture material should be of silver wire caught upon a curved Hagedorn needle or silkworm-gut carried by a long, straight, round needle. The suture is passed through the corium on each side of the wound, as shown in Fig. 116. The curved needle must be held in the bite of a needle-holder. When the suture has been passed the ends are pulled upon, and the skin-wound closes neatly.

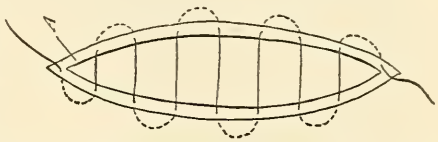


Fig. 116.—Halsted's subcuticular suture: *A* is the true skin.

Halsted's suture does not penetrate the cuticle; hence, in passing it the white staphylococcus is not carried through stitch-holes and into the wound, an accident which might be followed by infection of a stitch hole or even of the wound. When it is desired to withdraw this suture, take one end in the

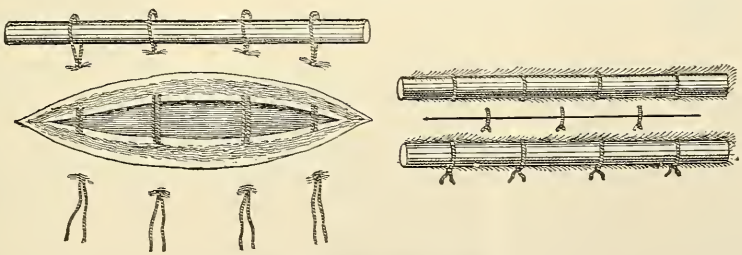


Fig. 117.—The quilled suture.

bite of the forceps, cut it off short with scissors, catch the free end with forceps, and pull steadily upon it.

In very deep wounds or wounds in which there is much tension after approximation the quilled suture (Fig. 117) or the button suture (Fig. 118) may be used. The twisted suture or hare-lip suture is shown in Fig. 119.

Problems of drainage, dressing, etc., are discussed on pages 76, 77, and 78.

If infection occurs, the wound becomes swollen, tender, painful and discolored, and the temperature of the patient soon becomes elevated. In such a condition cut the stitches, disinfect, and drain.

**Wounds of Mucous Membranes.**—If the surgeon intends to inflict a wound upon a mucous surface, he should see to it that the patient's general condition is good. Thorough asepsis is impossible, and a good result depends

largely upon the vital resistance of the tissues. Before operating, irrigate the part frequently with boric acid, peroxid of hydrogen, or normal salt solution. When ready to sew up the wound be sure that all irritant fluids are removed (saliva in the mouth, etc.). Cleanse the wound with hot normal salt solution. The stitches must include submucous tissue as well as the mucous membrane, and consist of silver wire, catgut, silk, chromic catgut, or silkworm-gut. After sewing up a wound in the mouth wash the oral cavity at frequent intervals with salt solution, and follow each washing with an insufflation of iodoform.

In accidental wounds irrigate with salt solution, dust with iodoform, and close as directed above. Corrosive sublimate is so irritant that it does harm when applied to a mucous membrane.

**Contused and Lacerated Wounds.**—A contused wound results from a blow or a squeeze which bruises and crushes the tissues and splits or ruptures the skin. It is a common injury when force is applied to tissues over a bone. The blow of a blackjack upon the scalp may cause either a contusion or a contused wound. A contused wound is irregular in outline, has jagged edges, and is surrounded by a broad zone of contusion. The worst form of contused wound is a crush of an extremity produced by being run over. The skin is often widely separated from the tissues beneath.

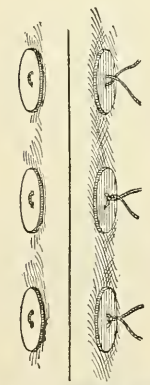


Fig. 118.—Button suture.

A lacerated wound results from tearing apart of the tissues. It, too, is irregular and jagged, and is accompanied by more or less contusion. A *brush-burn* is a contused-lacerated wound due to friction. Both lacerated and contused wounds contain masses of partly detached and damaged tissue, the vitality of which is endangered. Nerve-trunks, muscles, and great vessels may be torn across. Hence, such wounds are apt to slough, frequently suppurate, and are occasionally followed by cellulitis or even by gangrene. There is more danger of tetanus than in incised wounds. A wound especially apt to be followed by tetanus is made by the toy pistol. In contused and lacerated wounds the edges are discolored and cold to the touch, and there is little primary hemorrhage unless a cerebral sinus is opened or a great vessel is torn. There is considerable danger of secondary hemorrhage if large vessels have been bruised. In wounds of this nature the pain is often slight, but it may be violent. Shock is very severe.

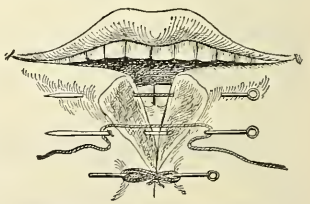


Fig. 119.—The twisted suture.

*Avulsion of a limb* is a dreadful form of lacerated wound. The thumb or a finger may be torn off or the arm may be wrenched from the body with or without the scapula. In such cases the wound is large, jagged, and irregular, long strings of muscle or tendon hang from the gap, the wound edges are cold, but the bleeding is trivial. The shock is, of course, profound.

*Avulsion of the scalp* may be produced when the hair is caught in machinery. The American Indian inflicts this injury when he scalps a conquered foe. In some cases of avulsion of the scalp the periosteum is removed with the flap; in most it is not. The flap usually consists of skin and aponeurosis. In this form of laceration there is severe bleeding.

*Treatment.*—The surgeon brings about reaction and endeavors to asepticize the wound and skin about it (see page 266), arrests hemorrhage, and ligates any visible damaged vessel whether it bleeds or not. Hopelessly damaged tissue should be cut away, doubtful tissue being retained. In some cases amputation

is necessary. If we apprehend tetanus, give an injection of antitetanic serum as a preventive. Secure thorough drainage, in some situations making counter-openings if necessary. Tube-drainage may be necessary or iodoform gauze in strands may be used. Contused wounds and lacerated wounds, except when on the face, are seldom closed by sutures. They are rarely closed, because the damage is so great and the blood-supply so interfered with that primary union will not occur. In the face the blood-supply is so good that primary union may be obtained in part or entirely, and it is worth while to try to obtain it. Cold must not be applied to a region of lowered vitality because it might cause gangrene. Heat is useful. Hence, it is advisable, even from the start, to dress with hot antiseptic fomentations, and this mode of dressing becomes imperative if sloughing begins. An excellent fomentation is made by soaking the gauze in a hot solution of acetate of aluminum, using 1 fluidram of a 7½ per cent. solution to an ounce of water (Waterhouse, "British Med. Jour.," July 9, 1910). Of course, the part must be kept at rest. After healing has advanced the occasional application of an 8 per cent. ointment of scarlet red greatly stimulates cicatrization.

If suppuration occurs, the surgeon sees to it that the pus has free exit, and if necessary secures free exit by making incisions. Bier's treatment and rest are useful for infections.

After avulsion of a limb the patient is reacted from shock, large vessels are sought for and tied, damaged tissue is cut away, the wound is drained by gauze and is *partly* approximated by sutures. After avulsion of the scalp bleeding vessels are carefully ligated. A portion of the scalp may be torn away, but a pedicle may connect it with the balance of this structure. In such a case cleanse the parts thoroughly and suture the flap in place (W. T. Bivings, "Phila. Med. Jour.," June 7, 1902). If the portion of scalp is entirely separated, adopt Gussenbauer's suggestion when possible and graft pieces of the avulsed scalp. In any case the ulcer resulting from avulsion must be repeatedly grafted. Abbe obtained healing in a case after four years by the use of 12,000 grafts.

**Punctured wounds** are made with pointed instruments, as needles, splinters, etc. The depth of a punctured wound greatly exceeds its surface area. After the withdrawal of the instrument inflicting the injury the wound partly closes at points, blood and wound-fluid cannot find exit, and if, as is probably the case, bacteria were deposited in the tissues, infection by pus organisms is very likely to occur, and if it does occur suppuration spreads widely. There is also danger of infection by tetanus bacilli. Such a wound may involve an important blood-vessel, and in such a case profound hemorrhage may occur; otherwise hemorrhage is slight. A great cavity of the body may be penetrated or an important organ may be wounded. Large-sized foreign bodies may be driven into the tissues or a portion of the instrument may break off and lodge. Pain is rarely severe unless a considerable nerve has been damaged. If both a large vein and artery are punctured, varicose-aneurysm or aneurysmal-varix may arise.

*Treatment.*—When possible, inspect the instrument which did the damage to see if a piece has been broken off. If there is severe hemorrhage, enlarge the wound and tie the bleeding vessels. In a puncture not made by the surgeon the wound must be regarded as infected. If a wound is made by a dirty instrument through skin known to be unclean, it is proper that the skin about the puncture be sterilized, that the wound be enlarged, that foreign bodies be removed, that the wound be irrigated with an antiseptic solution or be painted with pure carbolic acid, and be drained with a tube, a strip of gauze, or a piece of rubber tissue. Such treatment, though painful, and appearing unnecessarily severe or even cruel to the sufferer from a trivial puncture, is neces-

sary, and may save the patient from serious illness or from death. Every deep puncture inflicted by an instrument not surgically clean, and every puncture inflicted by a nail, a splinter, a meat-hook, a rusty pin, a tooth of a cat or dog, etc., must be regarded as grossly infected and must be treated by incision, sterilization, drainage, hot antiseptic fomentations, and rest. If the puncture is superficial and is made with a smooth-pointed instrument like a needle, when the instrument was not grossly infected the parts may be dressed with hot antiseptic fomentations, but they should be inspected daily for evidence of infection, and at the first sign of trouble an incision must be made. If a foreign body is retained in the tissue it must be removed.

Pure carbolic acid is a most efficient agent to sterilize a punctured wound.

If an important cavity of the body has been invaded by a puncture exploratory incision is necessary (see Brain, Thorax, Abdomen). In punctures with contaminated instruments the antitetanic serum finds a valuable place as a preventive of tetanus.

**Stab-wounds** were formerly considered with punctured wounds, but Senn wisely placed them in a class by themselves. Stab-wounds are inflicted by penetrating the tissues with a pointed or narrow instrument—for instance, a dagger, a knife, the blades of scissors, a bayonet, or a sword. Such wounds are narrow and very deep. A stab-wound may cause rapid death by penetration of a large blood-vessel. Some great cavity of the body may be penetrated and internal hemorrhage will then occur. The body may be transfixed by a sword or bayonet. Bone is rarely injured unless the skull is penetrated or the chest entered. In stab-wounds there is usually great hemorrhage and shock.

*Treatment.*—Whenever possible, look at the instrument which did the damage and see if a piece is broken off. If no great cavity is entered, treat by general rules: arrest bleeding, react from shock, etc. The treatment of penetrating wounds of the abdomen, thorax, and cranium is discussed in the special sections.

**Arrow-wounds** might be considered under the head of punctured wounds or stab-wounds. When hostilities with the red men were frequent and before mercenary traders had fitted out the savages with rifles, arrow-wounds were common among the men of the frontier. They are now very rare. Military surgeons still encounter them, especially in some parts of Africa and in the Philippine Islands. An arrow-wound may be a trivial puncture of the skin, a deep wound of the soft parts with or without bone injury, a penetration of a joint, or of one of the body cavities. The skull cavity may be entered by an arrow. In some of these cases there is a puncture of the bone without the formation of fissures, but usually when bone is punctured there is fissure formation, splintering, or depression. A large blood-vessel may be divided by an arrow and violent bleeding result or fatal concealed hemorrhage may take place from a wounded viscus. Some tribes poison arrows. It is said that the Piutes were the only tribe of North American Indians which did this.

Some tribes in South America use curare, others use snake-poison, others used decomposed meat. In Northern Nigeria some form of strophanthus plays a part in nearly all the poisons used (Allan C. Parsons, in "Brit. Med. Jour.," Jan. 23, 1909). The same author points out that the poison used is generally complex and contains also various animal and vegetable ingredients, particularly decomposed organic matter, plant juice containing strychnin, and soil contaminated with tetanus organisms.

*Treatment.*—An arrow is always septic and should be extracted. Sometimes when it has been buried deeply in a part it should be pushed across and extracted through a counteropening after the protruding shaft has been cut off. An arrow-head cannot be pulled out by the shaft. The barbs on the head catch and prevent extraction and the neck of the shaft is apt to break. The

tissues should be freely divided down to the head of the arrow and on each side of it, when it can usually be withdrawn by forceps. If imbedded in bone, the head must be gently rocked from side to side to loosen it, every care being taken to avoid breaking a stone or bending an iron arrow head. If an arrow has penetrated the abdomen, a laparotomy should be performed. If it has entered a joint, the joint should be freely opened. If it has entered the chest, one or more ribs will require resection. If it has entered the skull, trephining is indicated. Any bleeding vessels are to be caught and tied. The track of the arrow should be carefully disinfected and drainage should be secured. It is particularly important to remove a poisoned arrow at once. After removing a poisoned arrow, if the nature of the poison is known, proper treatment should be applied to antidote the poison. The French Colonial surgeons fill wounds inflicted by poisoned arrows with tannic acid. The same custom is followed by English surgeons in West Africa (Allan C. Parsons, in "Brit. Med. Jour.," Jan. 23, 1909).

**Gunshot-wounds** are contused or contused-lacerated wounds inflicted by materials projected by explosives. A bit of rock or a crowbar hurled by dynamite inflicts a gunshot-wound, as does a shell-fragment, a pistol-ball, small birdshot, a rifle bullet, pieces of a hand grenade, a flying cap, a piece of wadding, grains of powder, a buckshot, a fragment of metal broken off a shell, grapeshot and canister, or a cannon-ball. Injuries by shell-fragments, portions of a bursted boiler, pieces of masonry or wood, are either lacerated or punctured wounds, and need no special consideration here. In this article we treat of injuries caused by bullets and shot, that is, by missiles propelled from firearms.

Firearms are instruments by means of which missiles are projected to a distance by the expanding gases of burning gunpowder. There are many different sorts of firearms. Artillery includes various sizes of guns upon supports, from the great 12-inch guns of a battleship, which fire shells weighing 850 pounds, to machine-guns, which fire ordinary rifle bullets. Field artillery uses little but shrapnel-shells. Such a shell is a case of steel, cylindroconoidal in shape, containing a number of bullets and a charge of explosive, the shell exploding by means of a time-fuse. Canister is an iron casing containing bullets unassociated with an explosive discharge within the casing. The canister breaks when fired, and the balls separate over a large area. It is used only at close range—that is, 300 or 400 yards.

Among small arms may be mentioned muskets, revolvers, shotguns, and rifles. Wounds from the old-time musket ball are now never met with except in warfare against barbarous tribes. The musket has a smooth bore and fires round bullets of soft lead. This round, soft bullet, being large, moving with comparative slowness, and flattening easily, is very liable to glance, to deform, and to lodge. When a musket is fired at close range and the bullet strikes the tissue at a right angle, it produces a punched-out entrance wound. If the velocity is low or the impact is not at a right angle to the tissues, the entrance wound may "be formed of triangular flaps," the corners of which are inverted.[1] The entrance wound is surrounded by a bruised area. The track of the bullet is larger than the bullet, and is so badly contused and lacerated that some tissue is devitalized; and the shaft of a bone, if struck, is likely to be splintered. If the ball emerges, the wound of exit is larger than the bullet, and forms triangular and everted flaps. Healing by first intention seldom occurs in such wounds. The old smooth-bore musket, firing a round bullet, has been displaced by the rifle propelling a conical projectile.

In the firearms of civilians, as a rule, the bullets are made of lead, hardened and shaped by compression or hardened by an admixture with tin. The conical or cylindroconoidal rifle bullet has much greater velocity and pene-

[1] "Wounds in War," by Surgeon Col. W. F. Stevenson.

trating power than the round bullet. Hence, it is more liable to penetrate and less likely to deflect and to lodge. The tissues in the track of this bullet are less devitalized than in the track of the round bullet. The cutaneous surface is not so much contused. The wound of entrance is about the size of the bullet, and is punched out or inverted; and the wound of exit is larger than that of entrance and is often everted. The bones are more seriously comminuted than by the round bullet, and osseous fragments may be driven widely into the tissues. In fact, "an explosive effect" may occur at close range. Delorme lays it down as a rule that comminution of bone makes the wound of exit larger; and he asserts that a wound of exit larger in diameter than the thumb means comminution of bone.

*Gunshot-wounds Seen in Civil Life.*—Wounds are occasionally inflicted by the sporting rifle or the shotgun, and frequently by blank cartridges; but the vast majority of such wounds seen by the civilian surgeon are inflicted by the revolver.

Fig. 120.—Lodged shot.

*Wounds from the Sporting Rifle.*—In the sporting rifle a large charge of powder is employed. Some sporting rifle bullets have no hard jackets. Others have an incomplete hard jacket. In a bullet with a partial hard jacket the "nose" of the bullet is exposed and soft. The bullets are usually larger than those used in the military rifle. Such bullets deform in the tissues, and inflict dreadful, tearing wounds. If a bullet of a sporting rifle strikes a limb, amputation may be required. If it strikes the head or trunk, it will almost certainly produce a fatal wound.

*Wounds from the Shotgun.*—The degree of injury is in direct relation to the adjacency of the wounded individual to the gun, when the discharge takes place, to the size and number of the shot, and to the charge of powder. Single shot may bruise the surface and fail to enter the tissues or may enter the tissues.

When many shot enter together they strike as a solid body. Single shot are usually deflected from vessels and nerves, and seldom lodge in bone, but, rather, flatten on the bone surface. Even a single shot lodged in the eyeball is apt to produce violent inflammation which may destroy the eye. Numerous shot entering together at close range produce extensive contusions of the surface and fearful lacerations of the tissues, and often inflict irreparable damage. Bone may be fractured and bits of clothing or other foreign bodies may be carried into the wound with the shot. At close range toes or fingers may be blown off, an eye may be blown out, or portions of tendon or muscle may be shot away. At close range dreadful subcutaneous lacerations are caused by the gases. Primary hemorrhage is seldom severe because the wound is lacerated; but secondary hemorrhage is to be feared, and serious infection usually follows such injuries. Buckshot at close range inflict grave or dreadful wounds. The United States Army is supplied with a cartridge for use in riots. This cartridge contains two shot, each about the size of a buckshot.

*The Treatment of Shotgun Wounds.*—If the shot be scattered and lodged it is seldom necessary to remove them. As a rule, such cases require only cleansing of the skin and aseptic dressings. If shot lodge in a joint, they impair function; if in the face, they produce deformity. In both of these cases removal is necessary. When a shot lodges in the eye it usually, but not always, causes blindness. If the eye is gravely damaged it must be enucleated. In serious lacerations produced by shot at close range the hopelessly damaged tissue must be cut away, hemorrhage must be arrested, foreign bodies must be removed (though no protracted search is either necessary or desirable to remove grains of shot), the wound must be disinfected as well as possible, and free drainage must be employed. It is wise to give a prophylactic dose of antitetanic serum.

*Blank-cartridge Injuries.*—These injuries can occur only at close range. They consist of burns and lacerations, frequently a wad or a bit of clothing lodge in the tissues, and tetanus is a not unusual sequence. The explosive used in the toy pistol is a fulminate, and bits of the envelope of the explosive may be driven quite deep into the tissues. There is considerable danger of tetanus after injuries inflicted by the toy pistol. What in the United States is called "Fourth of July tetanus" is tetanus following such an injury, the small boy being prone to employ a toy pistol to contribute noise to the celebration of the nation's birthday (see page 203).

Blank-cartridge wounds and toy-pistol wounds are treated by cleansing the skin, enlarging the wound, removing foreign bodies, disinfecting, and draining. A prophylactic dose of antitetanus serum should always be given.

*Wounds Inflicted by the Revolver Bullet.*—The revolver varies in caliber from .22 to .45. Whereas it is true that certain military revolvers of the automatic type fire a hard-jacketed rifle bullet, the revolvers of civil life propel cylindroconoidal unjacketed bullets at a velocity of about 700 feet a second. A revolver bullet of the civilian's weapon never produces an explosive effect. It is liable to deform in the tissues, is often deflected from bone or tendon, and is very apt to lodge. The shape of the bullet, the velocity with which it is propelled and with which it rotates, and its hardness make it unlikely that at any near range the bullet will merely contuse, and not enter, the skin. Unless striking at an angle to the perpendicular, it will almost always enter. In some cases, however, a pistol bullet, like a spent rifle bullet, may fail to enter the tissues. It then grazes the surface and inflicts a brush burn or simply contuses the part. Sometimes it perforates, more often it lodges. Whereas it may be deflected, it comparatively seldom is; and it often deforms, though it does not do so to anything like the degree that the soft, round bullet does. If a bullet enters the tissues, a cavity, or an organ, and lodges there, it causes a

*penetrating wound.* If it enters and emerges it causes a *perforating wound.* The bullet may not enter alone, but may carry with it bits of clothing or other foreign bodies, though this complication is much rarer in injury with the conical bullet than with the round ball. On one occasion I removed a piece of coat from the interior of the lung, to which it had been carried by a pistol bullet. In another instance I removed a piece of shirt from the interior of the abdomen, to which it had been carried by a similar bullet. A revolver bullet may break bone, though it is not nearly so liable to do so as a rifle bullet.

In studying a gunshot-wound one must consider the *wound of entrance*, the *tissue track*, and, if the bullet has emerged, the *wound of exit.* It is usually stated that if a revolver bullet fired from a distance of 10 feet or more from the person struck hits the skin at a right angle, it makes a wound of entrance that is smaller than the bullet because the skin is elastic. It is a certain fact that one cannot assert from a mere inspection of the wound of entrance what size bullet a man was struck with. A .22 often leaves a most trivial opening. Careful separation of the margins enables us to measure a wound of entrance, and if this is done it will be found that a wound of entrance is never smaller than the bullet. (See Wm. S. Wadsworth, in "International Clinics," Vol. IV, Twentieth Series.) The shape of the wound is somewhat, but not regularly, circular, because a certain amount of tissue is destroyed. The margins are also somewhat depressed. It has a punched-out look, and the edges, as Draper tells us, are frayed in appearance ("Text-book of Legal Medicine"). The edges of the wound look thickened and are contused, this discoloration being noted for a distance of 1 inch or even 2 inches from the margin of the wound. The skin surface is distinctly blackened; but unless the weapon were fired at very close range this is not due to burning, but rather to staining with a mixture of burnt gunpowder and the grease of the outer surface of the bullet. The appearance of the wound of entrance will be very different if the bullet strikes the surface at an acute instead of a right angle. Then the wound will not be round, but oval or, perhaps, linear. When a bullet is fired very near to the surface of the body the hair of the skin will be burned, there will be some staining with gunpowder around the wound, and powder-grains will be found lodged in the skin. The burning is due to hot gases and with the gases come powder-grains. Whether the weapon inflicting the wound was close or distant there is bruising of the skin, but when the powder is found in the tissues or on the surface and when the surface is scorched the weapon must have been close at the time of discharge. Hot gases singe hairs, clothing, or the skin itself. The nearer the skin the weapon was held the more severe the burning. Wordsworth says, "all gas phenomena require very close range, usually within 18 inches." What is called the smudge is due to "the débris and smoke from the powder" and takes place at a range of less than 18 inches (Wadsworth, in "International Clinics," Vol. IV, Thirtieth Series). The absence of embedded powder, however, does not *prove* that the shot was not close, because the weapon employed might have been one using smokeless powder. When smokeless powder was used the burn is the same as from black powder, the smudge is apt to be of an orange color, tattooing is rarer, and when it does occur shows fewer grains. If the smokeless powder contains graphite it produces a smudge. If the weapon were fired at close range the skin may have been burnt by burning gases or the clothing may have been burnt and the skin scorched by the burning clothing. Staining of the skin with powder can be washed off, but when the skin has been burnt it is dry like parchment. When unexploded powder-grains are lodged in the skin the resulting condition is spoken of as *tattooing*, and this always means a very close shot. Powder-grains may cause severe wounds. It has been held by some that powder-grains are *never* found in the skin unless the bullet has been fired from a distance of less than 3 feet, but this is too arbitrary

a statement to make in a court of law. In any medicolegal case experiments should be made with a weapon and ammunition similar to those used in inflicting the wound in order to determine the real facts of the case. Draper ("Text-book of Legal Medicine") makes the following important statement relating to burns of the skin: "If the weapon is held in the hand in the ordinary way, hammer and sight on the barrel directed upward, the wound in the skin will show, immediately above its orifice, a brand or scorching caused by a slight recoil in the act of firing. The location of this brand will change as the position of the hammer is changed. If the weapon is held in a vise and fired this relation of the brand to the wound is obliterated. This observation, first made and published by Dr. D. B. N. Fish in 1883, supplies an accurate index of the position in which the pistol is held in firing." Wadsworth is of the opinion that the "flip" is usually but not always toward the hammer side. It is modified "by the grip on the handle at the instant of discharge." If the muzzle of a pistol is pressed lightly against the skin gases enter with the bullet and "burst the skin outward, giving a large ragged wound, which is not a wound of entrance, though always called so, but a wound of exit of the gas" (Wadsworth, in "International Clinics," Vol. IV, Thirtieth Series). If the muzzle is held firmly against the surface the gases cause a horrible wound under the skin. This condition is not the same thing as the explosive effect of a bullet. In passing through the tissues the revolver bullet makes a contused-lacerated wound, and we may find along this wound powder-grains (if the bullet has been fired at close range) and portions of clothing, pieces of the bullet itself, or perhaps of bone. A bullet may pass directly through both walls of the skull, traversing the brain in its passage. It may pass through a wall of the skull and lodge within the cavity of the cranium. In some cases it makes an opening of entrance that is smallest on the external surface of the bone and largest on the inner table. In other cases it makes extensive comminuted fractures. When a bullet tracks its way through a muscle it makes a jagged, contused, lacerated wound. It does the same in the brain. In both cases the track of the bullet is larger than the bullet, and the tissue for a considerable distance wide of the track is contused or actually destroyed. In passing through an aponeurosis or a serous membrane the bullet may make a round orifice or a slit-like tear. Of course, the nature of the wound in the tissues will be greatly affected if the bullet is deformed by having struck bone, or if it carries bits of bone along with it. The deflection of a bullet from an aponeurosis, fascia, or bone so alters its course that the missile becomes very difficult to locate and remove. In some cases a bullet has entered near the front of the body and passed around the wall of the chest until it has almost reached its point of entrance, or else has lodged or emerged at some point of this course—in either case constituting what is known as a *contour wound*. Contour wounds are not infrequently seen upon the head. For instance, a bullet may strike the frontal region, pass around under the scalp, and lodge in or emerge from the occipital region.

When the bullet does not lodge, but emerges from the body, the wound of exit must be studied. If an undeformed bullet passes straight through the body it makes a wound of exit that is somewhat larger than the bullet. It has a torn-out appearance, but without distinct destruction of tissue, and exhibits an irregular outline and eversion of the edges. The margins of such a wound are bruised, but are never scorched and never show powder-grains. If a bullet has been deformed by hitting bone, or if it has driven bone before it, a very large lacerated wound of exit may be formed. It is important to remember that the presence of a number of wounds on the surface of the body does not in itself prove that a number of different bullets have been fired, for in certain circumstances one bullet may make several wounds. A few years ago I saw

a case in which a bullet had penetrated the right hand and the right thigh, and had lodged in the left thigh. There were three wounds of entrance and two wounds of exit. Many very extraordinary cases of this sort have been reported.

**Symptoms of a Gunshot-wound.**—Hemorrhage is often considerable, but ceases spontaneously unless a large vessel has been divided. If hemorrhage is profuse the constitutional symptoms of hemorrhage exist (see page 436). These symptoms are of great importance in abdominal wounds. A pistol ball seldom causes severe primary hemorrhage, because it will not often penetrate a large artery. It is apt to push aside a vessel and secondary hemorrhage is not unusual. Even if a large vessel is wounded and a succession of violent hemorrhages occur, a man may live for several days. Secondary hemorrhage may follow a gunshot-wound because of contusion of vessels or of infection.

Pain is often not noticed at first. The injured individual, if greatly preoccupied or excited, may not know that he has been struck by a bullet. There may be only a feeling of numbness, but usually there is a dull or stinging pain. If a large nerve has been injured there may be violent pain. Even trivial gunshot-wounds frequently produce profound shock, and yet it may happen that severe wounds may be accompanied by but slight shock. In most gunshot-wounds of the brain, abdomen, and spinal cord the shock is very great.

**General Considerations as to Treatment.**—The dangers are shock, hemorrhage, and infection. Bullets are not aseptic when they enter a part, but a bullet usually carries few bacteria, and if infection is not inserted in the track of the ball the wound will in most instances heal kindly. A stationary bullet, when there is no infection, is usually let alone. "The fate of a wounded man is in the hands of the surgeon who first attends him" (Nussbaum). The danger of a wound depends upon the size and velocity of the bullet, the part struck, "and the degree of asepsis observed during the first examination and dressing" (De Nancrede). The rules of treatment are: bring about reaction, arrest hemorrhage, preserve asepsis, and, in some cases, remove the ball. Always notice if a wound of exit exists. It is a good plan, when endeavoring to determine the extent of injury, to put the parts in the position they were in when the injury was inflicted. We should try to ascertain the size and nature of the weapon, and the range at which it was fired. Examine the clothing to see if any fragments are missing and could have been carried in. Such fragments render sepsis almost inevitable. The surgeon must not feel it his duty to probe in all cases. In many cases it is better not to probe at all. Never probe when there is a wound of entrance and a wound of exit. Explore for and remove the ball when there is infection or when sure that it has carried with it foreign bodies; or when its presence at the point of lodgment interferes with repair; or when it is in or near a vital region (as the brain). We must locate the ball when it is necessary to know its position in order to determine the question of amputation or resection. If the wound is large enough the finger is the best probe. The *x*-rays render the use of the probe seldom necessary.

*Fluhrer's aluminum probe* is a valuable instrument (Fig. 121). It is employed especially in brain-wounds, and is allowed to sink into the track of the ball by the influence of gravity after the part has been placed in a proper position. If a lead bullet is deeply embedded it is possible to distinguish the hard projectile from a bone by inserting the asepticized stem of a clay pipe, a bit of pine wood, or *Nélaton's porcelain-headed probe* (Fig. 122). On any one of these appliances lead will make a black mark. No such test can be applied to a military bullet, for this has a hard metal jacket, and will not make a black mark on a white substance.

Though Nélaton's probe will not show the difference between a hard-jacketed projectile and bone, it is a valuable instrument to follow the track of any bullet wound. The porcelain head ought to be larger than it is usually made; in fact, it should be nearly the size of the bullet (Senn) (Fig. 123).

In passing a probe use no more force than in passing a catheter.

The *induction balance* of Graham Bell has been employed to determine the situation of a bullet. The bullet may be located by *Girdner's telephonic probe*. In order to construct this instrument take a telephone receiver, fasten one of the wires to a metal plate and the other one to a metallic probe. Moisten a portion of the patient's body and place the metal plate in contact with it. The surgeon places the receiver to his ear and inserts the probe into the wound.

Fig. 121.—Fluhrer's aluminum gravitation probe (natural size, except the length, which is 12 inches).

Fig. 122.—Nélaton's bullet-probe.

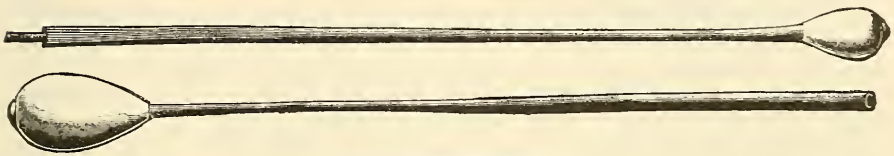


Fig. 123.—Senn's bullet-probe.

If the probe strikes metal, a click is heard with distinctness. A bullet may be located by *Lilienthal's probe*. This apparatus consists of a mouth-piece, two insulated copper wires, and a probe. The mouth-piece is composed of two plates, one of copper and one of zinc, which are applied to the sides of the tongue. An insulated wire runs from each plate and into the metal probe. The tip of the probe is composed of two or four pieces of metal, is separated from the shank by a washer of rubber, and is attached to the wires. The operator closes the teeth upon the mouth-piece and inserts the probe into the wound. If the probe touches the bullet a distinct and continuous metallic taste is appreciable.

The best means of discovering a bullet is to use the *fluoroscope* or take a *skiagraph*. In order to locate it accurately view it through a series of squares,

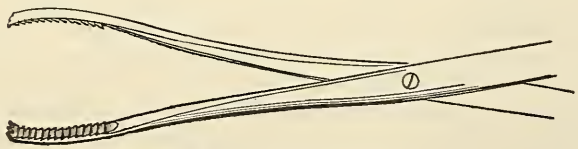


Fig. 124.—Bullet-forceps.

insert guide-pins, or, better than either of these plans, employ Sweet's apparatus. Bullets are readily seen by the fluoroscope in the superficial soft parts, and are discovered in deeper structures (bone, abdomen, lung, brain, etc.) by taking skiagraphs.

In extracting the ball use very strong forceps (Fig. 124). The old American bullet-forceps is useless for the extraction of the hard-jacketed ball, as the points will not penetrate and the instrument will not hold.

If hemorrhage is severe in a gunshot-wound, enlarge the wound, find the

bleeding vessel, and tie it. Before handling a gunshot-wound asepticize the parts about it and irrigate the wound with hot sterile salt solution. In some situations a wound should be drained with a short tube or a bit of iodoform gauze; in other regions this is unnecessary. The dressing should be antiseptic. Primary union rarely takes place after a wound inflicted by a pistol-ball or an ordinary rifle-ball, because of the inevitable necrosis of damaged tissue in the track of the ball, but in some cases it can be obtained. Primary union is frequent after injury by the small hard-jacketed modern army projectile. Healing begins in the depths of the wound and extends toward the wound of entrance, or, if there be also a wound of exit, toward both. Radical operations may be demanded: laparatomy, trephining, rib-resection, joint-resection, or amputation.

*Excision* may be required when there is great comminution. *Amputation* is sometimes demanded because of severe injury to the soft parts (as by a shell-fragment), great splintering of a bone, grievous injury of a joint, damage to the chief vessels or nerves, or the destruction of a considerable part of a limb. Perform a primary amputation if possible, and make the flaps through tissue that will not slough. In civil practice, with careful antisepsis, more questionable tissue can be admitted into a flap than in military practice, where transportation will become necessary and antisepsis may be imperfect or wanting. It has been shown in recent years that even when a large joint has been perforated by a small hard-jacketed projectile, amputation or resection is rarely required if the wound was treated aseptically from the beginning, but this is scarcely true of the revolver bullet.

**Wounds by Grenades.**—In the Russo-Japanese War grenades were largely used at close range. At close range they are much more destructive than rifle bullets because they explode and hurl fragments all about. The grenade may be cast by the hand, but it can be projected to a much greater distance "when fixed to and fired from the muzzle of a rifle by the discharge of a small blank cartridge" (Lt.-Col. Borden, in "Keen's Surgery," vol. vi). Grenade fragments produce nasty lacerated and usually infected wounds.

**Wounds by Cannon-balls.**—A solid shot was apt to kill a man instantly, tear off a limb or lacerate it extensively. Strange cases have been reported in which balls weighing 5 and 6 pounds were embedded in the tissues. In some cases of injury by spent balls the bone is destroyed and the muscles disorganized while the skin is intact. At the present time the projectiles usually fired in war by cannon are shells. A shell is a metal case containing an explosive charge and perhaps also bullets.

**Wounds by Shells.**—Sometimes a shell fails to explode and may then produce fearful mutilation or tear off a limb. Bursting shells may cause "injury by their concussion-blast, their flame may produce extensive burns, and their fumes may be overpowering. Actual wounds are inflicted by the fragments and by splinters, to which has been communicated the nature of missiles" ("Naval Surgery," by Surgeon-General Stokes, in "American Practice of Surgery"). The fragments vary in size, one man may be struck by many of them, wounds may be deep or superficial, may be horrible pulpifications, grave lacerations, or slight tears. Fragments are usually embedded, but may not be deep. Fractures are common. All shell wounds suppurate.

An ordinary shell contains only an exploding charge and injury is inflicted by the fragments of the exploded shell. Some shells explode by a time-fuse, others by percussion when the shell strikes a hard substance. The shrapnel shell is filled with bullets.

**Wounds in War Inflicted by Rifle-bullets.**—During the last few years frequent and notable improvements have been made in the military rifle. The range and rapidity of firing have been vastly increased, the velocity of the

projectile and its penetrating power have been enormously added to, and the trajectory has been decidedly lowered. Hence, the zone dangerous to an enemy has been lengthened. In order to accomplish these things changes have been made in the gun, the explosive, and the projectile. It is a far cry from the old Brown Bess, of song and story, to the modern Lee-Enfield of the British Army, or the Springfield of the United States Army. All modern military rifles are of small caliber, that is, less than .35 inch. The Springfield rifles of the days of the war between the States had a caliber of .45 inch. The old Springfield projected a bullet at an initial or muzzle velocity of 1300 feet a second; whereas the modern rifle sends a projectile on its way with an initial velocity of 2700 feet a second, the bullet rotating on its long axis more than 2500 times during the first second of translation. At a range of 1000 yards it will penetrate nearly 13 inches of pine wood. At a range of 100 yards it "will penetrate a steel plate .3843 inch thick" (Borden, in "Keen's Surgery," vol. vi). Up to 5000 yards a modern rifle can inflict a fatal wound, and it can be used point-blank at a range of from 500 to over 700 yards. With the present gun of the United States Army the point-blank zone of danger is about 718 yards. A bullet from a modern military rifle, even after having struck some solid, hard body, may grievously injure a man by ricochet. With a magazine rifle, at 2500 yards, from 5 to 10 per cent. of the balls will ricochet from turf. At 3000 yards they will bury in turf, but may ricochet from very hard ground. The United States Army now uses a magazine Springfield that weighs less than 9 pounds. The barrel is 24 inches in length and the diameter of the bore is .30 inch. The rifling makes one complete turn in every 10 inches. With this weapon, by magazine fire, 25 aimed shots may be fired in a minute; and when used as a single-loader, 23 aimed shots (Surgeon-General O'Reilly, U. S. A., in "Keen's Surgery," vol. iv).

*Old-fashioned Black Gunpowder as Compared with Smokeless Powder.*—There are many different varieties of smokeless powder, but each is essentially a nitro-powder. Among these smokeless powders are melenite, used by the French; lyddite, employed by the British; and shimose, adopted by the Japanese. The United States forces use cellulose nitrate in perforated cylindrical, amber-colored grains. Nitro-powder is very nearly smokeless because all the products of its combustion are gases. Of the products of the combustion of black gunpowder, 57 per cent. by weight settle out from the atmosphere in solid form on cooling.

There are great advantages in the use of smokeless powder. It is much more powerful than black gunpowder; hence, a smaller charge can be employed. The modern Springfield requires a charge of 47 gr.; and at the time of the discharge the pressure in the chamber is about 49,000 pounds to the square inch. Smokeless powder gives the bullet a greater velocity, causes less recoil, and fouls the barrel infinitely less than black powder; and the absence of smoke maintains a clearer atmosphere for observation, and also furnishes no sign of location which might prove of advantage to the enemy.

*Projectiles.*—The bullet of a modern rifle is conical, has a lead core, and is hardened by being covered with a mantle or jacket of copper, steel, or nickel, or of alloys of copper and nickel, or of copper, nickel, and zinc. The hard jacket is absolutely essential, because the speed of the projectile is so great that no soft bullet would take the rifling. Fragments would be torn off from the bullet in the gun, and the grooves of the gun would soon be filled with metal, the gun becoming useless. The projectile of a modern Springfield rifle is elongated and pointed. The air-resistance is least in a bullet of this shape. The core is composed of lead hardened with tin, and its jacket is of nickel and copper.

The military surgeon deals with wounds inflicted by these small, dense,

## MAGAZINE RIFLES OF SMALL CALIBER

| Country | United States | England | Germany | France | Russia | Italy | Austria, Bulgaria, Greece | Spain | Portugal | Belgium | Roumania | Turkey | Holland | Japan |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Pattern | Springfield | Short Lee-Enfield | 1888 | Lebel | 3 Line Nagant | Männlicher carcano | Männlicher | Mauser | Kropatscheck | Mauser | Männlicher | Mauser | Männlicher | Year 30 |
| Date (Gun is model of 1903, ammunition is model of 1906.) | 1903 | 1903 | 1888 | 1886 | 1891 | 1891 | 1888 1890 | 1892 | 1886 | 1889 | 1892 | 1890 | 1892 | 1900 |
| No. of bullets | 5 | 10 | 5 | 8 | 5 | 6 | 5 | 5 | 9 | 5 | 5 | 5 | 5 | 5 |
| Magazine system | Clip | Charger | Clip | Tube in fore end | Charger | Clip | Clip | Charger | Tube in fore end | Charger | Clip | Charger | Clip | Charger |
| Length of barrel | 24 in. | 25.19 | 29.134 | 31.496 | 29.922 | 30.75 | 30.12 | 29 | 31.633 | 30.67 | 28.74 | 29.134 | 31.1 | |
| Caliber | .30 in. | .303 | .311 | .315 | .300 | .2569 | .315 | .2756 | .315 | .301 | .2569 | .3012 | .2569 | .256 |
| Sights: Point-blank | 200 yds. | 200 | 273 | 273 | 310 | 437 | 246 | 437 | 328 | 547 | 437 | 273 | 437 | |
| Sights: Extreme | .... | 2800 yds. | 2242 | 2187 | 2096 | 2187 | 2460 | 2187 | 2406 | 2187 | 2296 | 2187 | 2296 | 2187 |
| Weight without bayonet | 8 lb. 14 oz. | 8, 2½ | 8, 6 | 9, 3½ | 8, 13 | 8, 6 | 9, 11 | 8, 9½ | 10, 3 | 8, 9½ | 8, 7½ | 8, 9½ | 9, ¾ | |
| Bullet: Material of core | Lead, tin | Lead | Lead | Lead | Lead | Lead | Lead | Lead | Lead | Lead | Lead | Lead | Lead | |
| Bullet: Envelope | Cupro-nickel | Cupro-nickel | Steel, cupro-nickel | Cupro-nickel | Cupro-nickel | Cupro-nickel | Steel, lubricated | Steel, nickel | Steel | Cupro-nickel | Steel, cupro-nickel | Cupro-nickel | Steel, nickel | Copper |
| Bullet: Weight | 150 gr. | 215 | 227 | 216 | 214 | 163.5 | 244 | 172.8 | 248 | 219 | 162 | 213 | 162 | 162.9 |
| Weight of charge | 47–50 gr. | 31.5 | 42.44 | 41.66 | 33 | 31.6 | 42.44 | 37.8 | 70 black | 47 | 36.26 | 40.2 | 36.26 | |
| Muzzle velocity (ft. secs.) | 2700 | 2060 | 2034 | 2073 | 2034 | 2305 | 2034 | 2288 | 1750 | 2034 | 2305 | 2129 | 2305 | 2395 |
| Pressure in chamber, tons per square inch | 22 | 15.75 | 21 | 17.75 | 10.15 | 17.1 | 19.7 | 22.3 | 15.75 | 19.7 | .... | 19.7 | | |

(Adapted, with modifications, from Spencer's " Gunshot Wounds" and Stevenson's "Wounds in War.")

hard, conical projectiles, impelled with great velocity, and carried long distances. The old bullet was liable to lodge, was often deflected in the tissues, was flattened out on meeting with resistant structures, such as bone or cartilage, and, after flattening, became larger, tearing and lacerating the soft parts and comminuting the bone. The modern projectile is likely to penetrate, is rarely deflected, and is so hard that its shape is often but little altered on meeting with resistant structures. Hence, it was thought that the new bullet would prove more humane than the old projectile and inflict wounds that would be more easily treated, because the bullet would be unapt to lodge and extensive damage would seldom be inflicted. This view has proved, to a great extent, correct.

With the modern rifle of small caliber and the hard projectile propelled by smokeless powder the range has been notably increased, the trajectory of the bullet's flight has been greatly lowered, and the danger-zone to an enemy has been correspondingly lengthened.

Fig. 125.—1, Krag-Jorgensen; 2, new Springfield.

*Mechanics of Projectiles.*—If a moving bullet were acted upon by no force but propulsion, it would continue to move in the direction that it was pursuing when it left the muzzle of the gun and its course would be a straight line, but it is acted upon by other forces. Even in a vacuum its course would not be a straight, but a curved line, because gravitation would draw it toward the earth. Under ordinary circumstances the air also resists its forward progress.

A moving bullet is urged onward by the force of the exploding powder. This onward movement is called the *motion of translation.* The rate of forward movement is the *velocity,* and this is expressed in feet per second. Air-resistance causes the velocity to lessen rapidly, and the farther away from the gun the projectile is, the greater is its loss of velocity. For instance, on leaving the muzzle of the Lee-Enfield rifle a bullet has a velocity of 2060 feet a second (*muzzle velocity* or *initial velocity*); at 700 yards it has a velocity of 1039 feet a second; at 2000 yards, 571 feet a second; at 3000 yards, 369 feet a second ("Gunshot-wounds," by Major C. G. Spencer). The muzzle velocity of the

bullet of the United States Army Springfield is 2700 feet a second. The velocity of a bullet at any particular portion of its flight is called *remaining velocity.*

A bullet fired from a rifle rotates on its long axis. This rotation is called *spin* or the *movement* of *rotation,* and is in the direction of the groove of the rifling. It is this motion that keeps the point of the bullet toward the front and prevents rotation on its short axis, which would be responsible for increased air-resistance, diminished striking force, and lessened range. If a cylindro-conoidal bullet were fired from a smooth bore, it would rotate on its short axis at even as short a range as 9 yards, and would strike a target in its length (Stevenson's "Wounds in War").

The diminution in the transverse diameter of bullets has necessitated an increase in length in order to maintain their weight and sectional density. (*Sectional density* is the weight divided by the area of the cross-section.) The increase in length makes an increased rapidity of rotation indispensable. The higher the pitch of the rifling, the more rapid the rate of rotation imparted to the bullet. The Minié rifle had a complete turn in 78 inches. The United States Army Springfield has a complete turn in 10 inches. The velocity of spin as the bullet leaves the barrel of a Springfield is about 2500 times a second. The velocity of rotation changes as the velocity of translation changes; and when translation ceases, because the energy of propulsion has expended itself, rotation also ceases. "But when the motion of translation is suddenly and completely arrested by contact with an obstacle, then, if the bullet is not broken up, the motion of rotation continues until its energy is expended" ("Wounds in War," by Surgeon-General W. F. Stevenson, C. B., A. M. S.). A rifle-bullet in its flight deviates a little laterally, and in the direction of the groove of the rifling. In the United States Army rifle the groove of the rifling is toward the right when the gun is held with the butt toward the shoulder; hence, the deviation of the bullet is toward the right. This lateral deviation is called *drift.*

*Influence of Gravity and Air-resistance.*—We have previously stated that, even if moving in a vacuum, the line of flight of a bullet (the *trajectory*) would be a curved and not a straight line, because of the influence of gravity, which pulls the bullet toward the earth. The bullet would fall 16 feet the first second, 48 feet the second second, 80 feet the third second, and so on. A bullet moving forward in a vacuum would advance through equal distances in equal periods of time, and, as gravity would draw it toward the earth with increasing rapidity, the trajectory would be a parabola (Fig. 126, line A–E–F–G).

Air-resistance strongly retards the advance of a bullet and causes it to rapidly lose its velocity, and a bullet fired in air does not advance through equal distances in equal periods of time. Because of air-resistance a bullet falls to the earth sooner than it would under the influence of gravity alone. Hence, the trajectory of a bullet in air is not a true parabola, but the line of descent is much nearer to the vertical than would be the case in a vacuum (Fig. 126, line A–*e*–*f*–*g*).

Air-resistance depends upon the velocity of the bullet, the cross-section area of the bullet, the shape of the head of the bullet, the atmospheric density, and the steadiness of flight ("Gunshot-wounds," by Major C. G. Spencer). Air-resistance is least in the bullet that tapers rapidly. A bullet begins to lose its steadiness of flight about 1000 yards from the muzzle of the rifle.

*The Danger Zone.*—Owing to the fact that the trajectory is a curved line elevation must be given to rifles except at point-blank range, and the degree of elevation must be increased according to the range. Point-blank range for a Springfield is up to 718 yards. By this term is meant that when a gun is aimed horizontally the entire course of the bullet up to 718 yards is dangerous for infantry. For longer ranges the rifle must be elevated and the bullet "shot into the air." Sighted at 2000 yards the Snider sent a bullet 866 feet above the line

of sight; the Martini-Henry sighted at the same range, 357 feet; the Lee-Enfield at the same range, 194 feet. It becomes evident that when a rifle is elevated the bullet rises far above a man's head, and continues to rise to what is known as the *culminating point*, when it begins to descend. It does not become dangerous

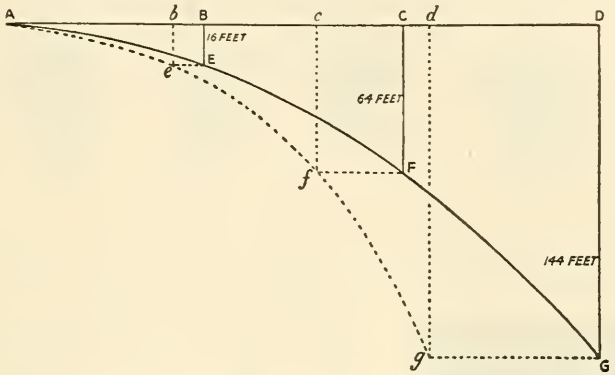



Fig. 126.—Trajectories in vacuo and in air (Stevenson).

to men until it gets near to the earth. The point at which it becomes dangerous to cavalry is called "*the point of first catch for cavalry*" (Fig. 127). The point at which it becomes dangerous for infantry is called "*the point of first catch for infantry*" (Fig. 127). The *dangerous zone* (Fig. 127) is from the point of first

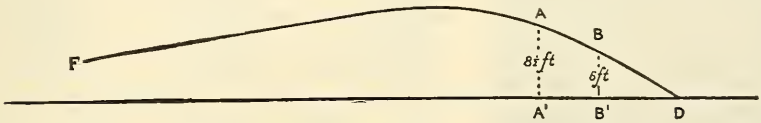



Fig. 127.—Trajectory, showing dangerous zones: A–B–D, Trajectory of bullet; D, point of termination of the bullet's flight; A–B, point of first catch for cavalry; B, point of first catch for infantry; A'–D, dangerous zone for cavalry; B'–D, dangerous zone for infantry (Spencer).

catch to the termination of the bullet's flight, because anywhere in this zone men may be struck, but between the point of first catch and the man firing the gun soldiers are perfectly safe. The point of first catch for cavalry is about $8\frac{1}{2}$ feet and for infantry about 6 feet above the ground.

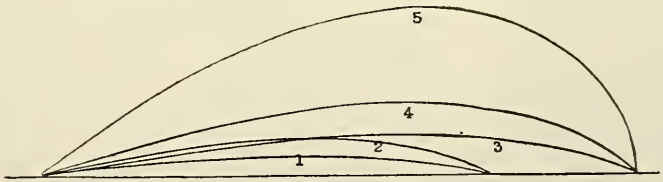



Fig. 128.—Trajectories of bullets from certain rifles: 1, Trajectory of Lee-Enfield at 1500 yards action-point, 81 feet; 2, trajectory of Martini-Henry at 1500 yards, action-point, 178 feet; 3, trajectory of Lee-Enfield at 2000 yards, action-point, 194 feet; 4, trajectory of Martini-Henry at 2000 yards, action-point, 357 feet: 5, trajectory of Snider at 2000 yards, action-point, 866 feet; horizontal scale, $\frac{1}{24000}$ (1 inch = 2000 feet); vertical scale $\frac{1}{12000}$ (1 inch = 1000 feet). Vertical measurements are represented on twice as large a scale as horizontal measurements (Spencer).

The more nearly vertical the line of the bullet's descent, the shorter is the danger zone; the less vertical the line of descent, the longer is the danger zone. The higher the culminating point of the trajectory, the more vertical is the line of descent, hence the shorter is the danger zone; the lower the

culminating point, the flatter is the trajectory, and the less vertical the line of descent, hence the longer the danger zone. The chief object of improvements in rifles is to lower the trajectory (that is, make it less curved) and thus lengthen the danger zone, render marksmanship more accurate, and insure velocity.

*Power of the Bullet to Wound.*—According to Spencer ("Gunshot-wounds"), this depends upon its energy and the ease with which its energy is converted into work on striking. Energy is largely a matter of range. At short range energy is enormous, but it rapidly diminishes as the range is increased. At 3000 yards energy is only about one-sixteenth of what it is at 300 yards. The ease with which a bullet converts energy into work depends upon (see Spencer (Ibid.): (1) The area of the cross-section of the bullet. The larger the bullet, the worse the wound. (2) The deformation of the bullet. Such deformation enlarges the area. A bullet that expands on striking is said to "*mushroom.*" The modern bullet seldom deforms much unless its jacket has been more or less torn off; and it inflicts, as a rule, a much less grave injury than does the soft bullet. It has been found that this very humanity of the bullet is at times regarded as an objection. The bullet lacks "stopping power" unless it strikes a vital part or a large bone, and a wounded man may continue to fight and charge. Civilized men will usually stop when hit, but savages very often will not. Hence, in warfare with barbarous people, it was until recently the custom to modify the bullet. A portion of the soft bullet at the apex of the projectile was left exposed, and such a bullet was said to be uncovered or to have a "*soft nose.*" It was called a *Dumdum* bullet, because such missiles were first made at Dumdum, the ordnance factory near Calcutta. When a Dumdum bullet strikes, it spreads and expands, or "mushrooms," and inflicts an extensive and dreadful wound, which stops the most ferocious savage or the most fanatical tribesman. These expanding or deformable bullets are often wrongly called "explosive bullets." They have been forbidden by The Hague Convention, although Stevenson and some other surgeons maintain that they are more humane than were the bullets of the Snider or the Martini-Henry rifle. The present weapon of the United States Army will produce wounds "which will resemble the wounds made by the 'deformable' bullets whose use is forbidden in civilized warfare" (Borden, in "Keen's Surgery," vol. vi).

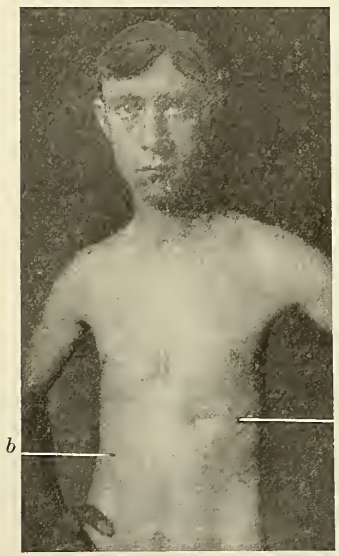



Fig. 129.—Mauser bullet-wound of chest: *a*, Wound of entrance; *b*, point where bullet was extracted (Major Charles F. Kieffer, U. S. A.).

*The Resistance Encountered.*—If energy is great (as at close range) and a very resistant tissue is struck (bone), dreadful injury may be inflicted, but if a tissue with little resistance is struck but little damage may be done. At a long range the energy of the bullet is so lessened that the danger to resistant tissue will be much less; whereas injury of soft parts may be much the same as at closer range.

*The Nature of the Wounds Inflicted by the Small Projectile.*—The effect of lessening the size of the bullet is to decrease its wounding power, because, other things being equal, the larger the bullet, the greater its wounding power. In many instances the modern bullet will make a clear track, without lacera-

tion or comminution. It was thought, as has been stated, that this projectile would prove humane, that it would kill comparatively few, and that the wounded would receive injuries which would incapacitate them, but from which most of them would subsequently recover. Recent wars have indicated that at a range of over 1500 yards the bullet, as a rule, penetrates cleanly,

Fig. 130.—Deformation of leaden bullets (natural size) (Seydel).

making a wound that heals by first intention. Sir Frederick Treves expressed his experience by saying, "the Mauser bullet is a very merciful one."

Very many studies have been made of the action of the modern bullet, and numerous experiments have been carried out—firing through boxes filled with wet sand, firing into thick oak, firing at cadavers at fixed distances with reduced charges, and firing at corpses and at live horses with service charges.

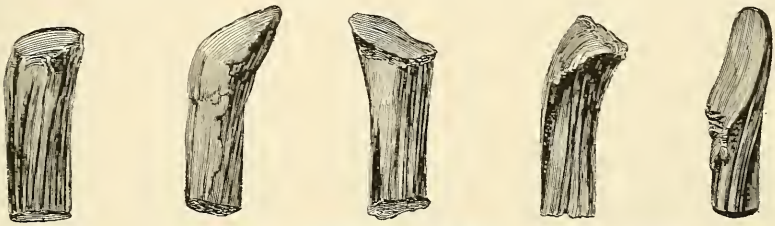


Fig. 131.—Deformation of small-caliber jacketed bullets (after Bruns).

De Nancrede, some years ago, wisely cautioned us to remember that experiments upon the cadaver, employing reduced charges and standing at fixed distances, are uncertain in their provings. "The difference," he said, "between the velocity of rotation and the angle of incidence with reduced charges at fixed distances and service charges at actual distances is marked. The tension of living muscle and fascia, as compared with dead tissues, and the physical

Fig. 132.—1, Empty Krag jacket removed from thigh after penetrating 6 inches at 340 yards; 2, 3, 4, 5, lodged Krag bullets removed from wounds after deflection from frozen ground. Ranges from 50 to 300 yards.

change of semiliquid fat of adipose tissue and medulla to a more solid condition by the loss of animal heat, influence the results" (Ibid., "Gunshot-wounds," Roswell Park's "Surgery by American Authors").

All the theoretical conclusions derived from experiment and the observations made on the occasional victims of suicide or homicide have been put to the test of war in recent years; and we now draw our deductions from a study of the

wounds in the Chitral Expedition, the Greco-Turkish War, the Spanish-American War, the South African War, the taking of Pekin, and the Russo-Japanese War. (Reports from the Balkan War are not as yet accurate or comprehensive.) Preconceived opinions have, to a great extent, at least been confirmed. It has been found that the wounds are usually non-infected and are apt to heal by first intention unless inflicted by ricochet, which deforms the bullet. At a range of 1500 yards or more the wounds are commonly clear tracts, without much splintering of bone or laceration of tissues. The wound of entrance is extremely small and could be overlooked by a careless observer. It is usually circular, it may be triangular. The bullet is far less liable to lodge in the body than was the older bullet. If it perforates, the wound of exit is usually small, and may be either round or a slit. The wound of exit is large if the injury was inflicted at close range, or if bone was splintered and fragments were driven along with the bullet.

Theoretically, the projectile does not flatten, but it has been found that in many instances it does flatten a little (Fig. 131). Its coat is apt to be torn off when it strikes hard bone at a distance of less than 1800 yards. Treves has pointed out that if a bullet smashes a bone and lodges, the shell, as a rule, peels off from the core. Then the bullet may be distorted or broken into fragments. When a hard-jacketed bullet is "traveling over 2000 feet per second" it "will hold together" and penetrate any human structure "without breaking up, but as the velocity drops, there comes a point when the resistance is too great for the momentum of the bullet to overcome quickly, and then the bullet piles up on itself, just as it does when it strikes a very hard object, and the lead crowds to the front part of the bullet till the nose of the jacket bursts. When this happens the wounds are very serious and have given rise to rumors of the use of 'dumdum' or soft-nose bullets or of explosive bullets" (Wadsworth, in "International Clinics," Vol. IV, Twentieth Series). At long range the bullet may lodge, or it may also do so if it hits a man after bounding from a stone, hard ground, or a piece of metal. It may lodge in compact bone. In Cuba 10 per cent. of the wounded suffered from lodged bullets. In the Russo-Japanese War less than 10 per cent. of the wounded suffered from lodged bullets.

It is seldom that bits of clothing are carried in with the bullet, but sometimes they are, and some fabrics are more liable to be carried in than others. Threads are not unusually carried in on the roughened areas cut into the bullet by the gooves of the rifling. If the bullet ricochets from stony ground, bits of stone may enter the tissues.

Blood-vessels are likely to be cut and not pushed aside, as was often the case with the old-time bullets. Cases of arterial contusion and subsequent secondary hemorrhage are, however, occasionally met with. If a large vessel is struck and cut, primary hemorrhage is profuse and may prove rapidly fatal. The modern bullet is seldom deflected in the tissues, but, as a rule, it passes straight ahead. The skin is usually split by it. Fascia and muscle are likely to be much damaged, but in a transverse wound of muscle the fibers may be separated rather than destroyed.

Although under most circumstances this bullet is humane, it has been found that in some instances it pulpifies structure for a considerable distance around the track of the ball, producing what is known as an *explosive effect.* The cause of this condition has been much debated, though it never means that the bullet has exploded. Some think that it is always due to comminution of bone and the blowing of bone-fragments ahead of and around the ball. Most believe that the sudden impact against the tissues engenders waves of force, which cause explosive and distant damage. Certain it is that an explosive effect causes horrible and often irreparable injury.

Explosive effects are most frequently seen at close range, when the velocity

of translation and the frequency of rotation are most marked. Such injuries were seen in the marines killed at Guantanamo, in persons killed during the Milan riots, and in many instances in South Africa, China, and Manchuria.

A pistol-bullet has no explosive action at all; and the old-time large, soft bullet possessed it only at a very close range. The modern projectile is apt to produce explosive effects up to 500 yards, but it does not invariably do so. Up to 1300 yards it is liable to produce them in the skull and brain; and at this distance a single bullet may entirely destroy the cranium. Explosive effects may at times occur at longer distances upon the liver, spleen, kidneys and lungs, and upon hollow viscera containing fluid. At a distance of 500 yards or less a bone will usually be shattered into many fragments; whereas, at a range of 1500 or 2000 yards the bone will, as a rule, be cleanly perforated, usually without comminution.

It is often extraordinary how little trouble follows a wound by a modern projectile and how quickly healing occurs. This is due to the facts that the tissue is cleanly perforated, that foreign bodies are seldom carried in, and that the wound rarely becomes infected. This freedom from infection is not due to the bullet being sterile. It is not sterile, and when the gun is fired the bullet does not heat sufficiently to certainly destroy bacteria. The bullet is carried in a dirty belt or pouch, is handled by dirty hands, and is not clean when put into the rifle, but its sides may be scraped cleaner by the rifling and the burning powder may disinfect it in part. The point, however, contains bacteria on its surface. They are few in number; are readily scattered in the tissues; and, in most instances, are overcome by tissue resistance. The clean track of the bullet which is usual in wounds inflicted at ordinary fighting ranges impairs tissue resistance much less than a badly contused and lacerated wound. In some observed cases there have been almost no symptoms after perforation of the lung. In others, none after perforation of the abdomen, a joint, or the skull. The bullet when it has become stationary seldom does harm, unless lodged in the brain. It is obvious that in most conflicts the modern rifle has proved to be humane and that its humanity is largely a matter of range. At a range of 1500 yards or over modern rifles (except the Springfield) are humane weapons.

What used to be called a *wind contusion* is a severe and often a dreadful injury. The skin being unbroken, bones may be broken, viscera ruptured, tissues torn asunder. The older surgeons believed that such an injury was produced by the wind pressure, a projectile passing close to, but not touching the surface. We know now that they result from a projectile's glancing along the surface, the elasticity of the skin saving it from immediate destruction, although in many instances it sloughs later.

A bullet in striking never gets hot enough to burn a part. It is needless to say that bullets are never deliberately poisoned.

**Symptoms.**—*Pain* is seldom severe in wounds of the soft parts, but violent, immediate pain is felt when a bone or a nerve is injured; the pain is usually stinging or burning, but is seldom of long duration, except in bone injuries, spinal cord injuries, and nerve injuries. Sometimes a man does not know he has been struck. It is common to have anesthesia or numbness and loss of muscle-function about the wound for several hours or days.

*Shock* is very variable. In some cases it is scarcely noticeable, in others it is overwhelming. It is most marked in wounds of bone, the spine, the abdomen, and the brain. It is greatly aggravated by hemorrhage. Hemorrhage is great if a large vessel is struck. If the vessel is in a limb it is seldom that much blood escapes externally, but a large hemorrhage occurs in the tissues. Such cases reach the hospital for treatment, and are spoken of as "traumatic aneurysm." They were quite common in South Africa. Secondary hemor-

rhage is uncommon. When it does occur it is usually a result of infection, but it may arise, as in a soft-bullet injury, from contusion of a vessel. De Nancrede reports secondary hemorrhage, in the absence of infection, from contusion of the brachial artery. If a great vessel is divided in the chest or abdomen, the patient rapidly bleeds to death on the field, and seldom reaches the hospital at all. A military bullet may cause arteriovenous aneurysm. Makins, De Nancrede, and others have reported cases. De Nancrede saw one arteriovenous aneurysm of the subclavian, two of the femoral, and one of the external iliac from Mauser bullet wounds.

Primary infection is rare. The bullet wound tends to remain uninfected unless bits of clothing or other foreign bodies have been carried in, unless the bullet was deformed, unless the wound was at close range, or unless unnecessary and uncleanly probing was practised. If suppuration occurs, it is apt to remain localized. Pyemia and true septicemia are rare. In the Russo-Japanese War suppuration seems to have been common. In the Japanese hospitals at least 60 per cent. of wounds of the soft parts by undeformed bullets suppurated. At most in only one case out of ten does the bullet lodge. (Report on Russo-Japanese War, by Maj. Chas. Lynch, Medical Department, General Staff, U. S. A.) It is stated that among the Russians suppuration occurred in 30 per cent. of the cases. More wounds suppurated in winter than in summer. The Russians used a larger bullet than the Japanese, and the wound inflicted by the Russian bullet was far more liable to suppurate than was that produced by the Japanese projectile. Practically all wounds of bone made by Russian bullets suppurated. (Lynch, Ibid.) The above remarks upon the military projectile do not apply in all particulars to the new bullet adopted by the United States (model of 1906). It is of the same caliber as its predecessor, but it is .17 inch shorter, 70 gr. lighter, and its point is decidedly sharper. The muzzle velocity is 400 yards per second greater. Point-blank range has been extended from 600 to 718.6 yards. Because the new bullet is short and because the center of gravity is toward the base the bullet is easily deflected and is prone to enter the tissues sideways, inflicting a frightful wound, instead of the usual small puncture of the ogival headed bullet of 1903. La Garde has pointed out that even the skin resistance may cause the bullet to turn. Hence it is evident that the bullet of the Springfield now in use is distinctly not a humane weapon; it will inflict horrible injury and will be very apt to kill the victim outright (Lt.-Col. Borden, U. S. A., in "Keen's Surgery," vol. vi). It has not yet been tested in war.

**Treatment.**—The military surgeon is a specialist, and he must know many things besides the treatment of the sick and injured. He must be a master of hygiene; he must possess executive capacity; he must be able to discipline others and to subject himself to discipline; he must be forceful, self-reliant, and resourceful; he must be acquainted with the laws and regulations of the military establishment; and he must have a special knowledge of gunshot-wounds as received in battle. Even the best qualified civil surgeon is unfit to pass into military service without special instruction. It is for this reason that the United States insists that every man appointed to the Medical Corps of the Army or Navy shall receive special instruction in the Army Medical School or the Naval Medical School before he goes to a regiment or a ship. The wounds received in war are peculiar, and treatment appropriate for a wound inflicted by a revolver bullet is often inappropriate or impossible for a wound inflicted by the projectile of a military rifle.

In civil life the patient has the best of surroundings. Every care can be given him. Numerous skilled assistants are at hand if needed. The problems of the case are entirely surgical, and the case can be dealt with purely according to its surgical necessities. In war there are problems of transportation

which are not presented in civil life, for strategic necessity may compel hurried movement. Accommodations, also, may be bad. Shelter may be imperfect, climate and meteorologic conditions may be most trying. Food may be scanty and inappropriate. Medicines may be scarce. There are sure to be too few assistants. After some engagements in South Africa the British surgeons had to care for numbers of men under difficulties that were appalling, among which were fearful clouds of dust and swarms of flies. In other words, the military surgeon, after a battle, is seldom able to treat his cases purely in accordance with surgical necessities, but his conduct must be influenced by other, often imperative, needs. If there are numerous wounded, he does not have time to do immediate laparotomies. He will lose some cases because he has not done laparotomy, but he would lose many other cases from delay in treating dangerous but remedial conditions were he to make many simpler cases wait until his laparotomies had been performed. He is forced to make the abdominal wounds wait, and after long delay there is seldom any use in opening the abdomen at all.

Again, the wound inflicted by the bullet of a military rifle is very different in nature and in danger from the wound inflicted by a revolver bullet. In the former, if a large vessel is struck, it is usually perforated or divided, and profuse bleeding occurs, either into a cavity or in the tissues. If the bleeding occurs in a cavity the patient usually dies on the field, and does not reach the first dressing-station at all. If it occurs in the tissues a "traumatic aneurysm" forms. In revolver bullet wounds primary hemorrhage is seldom severe. Wounds with revolver bullets are very apt to suppurate. Wounds with the undeformed hard-jacketed projectile that has not ricocheted very commonly escape primary infection.

Wounds that in civil life might require only a resection, may in military practice require amputation. The promise of aseptic healing leads the military surgeon to trust many wounds without operation which in civil life would be operated upon at once, and both surgeons would be right in the different courses pursued by them.

In civil life the rule is absolute to open the abdomen for every case of gunshot-wound entering that cavity. The experience of all military men is that more cases get well under a policy of non-interference than with laparotomy. In military surgery laparotomy can be performed only when there is *"time to do it"*; and, even then, is performed only when there is hemorrhage or else certain evidence or a very strong probability that an organ or viscus has been struck or perforated. On account of the difficulties in the treatment of the wounded in military life, as compared with civil life, military surgery is a pure specialty; and the details of the treatment of wounds in war must be sought for in treatises by military surgeons. The watchwords of the military surgeon are to preserve asepsis and to avoid meddlesome interference.

In handling patients in the field the clothing is cut away (if the wound is under the clothing). If possible, the wound and skin about it are washed with alcohol and painted with iodin (tincture, diluted one-half with alcohol) and then the dressings are applied. If this cannot be done the dressings are applied at once. The dressing should be absorbent and, if possible, antiseptic rather than aseptic gauze. Absorbent cotton should be placed over the gauze, and a bandage of linen should be applied to hold the dressing in place.

In warfare at the present day an attempt is made to limit the death-rate from gunshot-wounds by protecting them from infection at an early period after the accident. Esmarch offered a suggestion which has been adopted in the armies of all civilized countries. Every officer and private soldier carries a package which contains antiseptic dressings, and at the first opportunity after the infliction of a wound, if possible on the field, these dressings are applied by

the soldier, by a comrade (for even the privates are instructed in the application), or by an ambulance man. If not applied on the field, they are applied at the first dressing-station by a surgeon or a hospital steward. The dressing is removed only when there are indications calling for surgical interference. Many wounds heal under this primary dressing. In the United States Army the first-aid package is carried in a metal case to prevent contamination and damage by moisture. The case is hermetically sealed, but can be easily opened. It is carried hooked to the cartridge belt. It contains two bandages, two compresses of absorbent corrosive sublimate gauze, and two No. 3 safety-pins, all wrapped in waxed paper. One compress is stiched to the center of each bandage, and the bandage is so folded that the compress can be opened without touching its inner surface. Each private of the hospital corps and the orderly of each medical officer carries the hermetically sealed tubes—each tube contains 1 gm. of iodin and 1½ gr. of iodid of potassium. By adding 50 c.c. of water or alcohol a proper antiseptic solution is obtained. In injury of limbs amputation is seldom necessary. It is done when the great vessels are injured, when the soft parts are grievously lacerated, when an articular surface is badly comminuted, and perhaps when there is menacing secondary infection. Excision is occasionally performed when there is comminution. As a rule, a wound of an articulation is recovered from by antiseptics and splinting.

Most lodged balls are let alone. If the wound is infected, or is known to contain foreign material other than the bullet, the bullet and all other foreign material must be removed. A bullet in the brain should be removed, though we often wait perhaps for days to let the tissues regain resisting power (De Nancrede). Serious hemorrhage always calls for operation. Tie the vessel in the wound if possible; if not, tie the main trunk above, and if this fails, amputate (De Nancrede). In a chest wound delay for symptoms. In an abdominal wound do not operate except for hemorrhage or for evident visceral injury.

In the foregoing article I have obtained facts from numerous sources. The following books and articles I found particularly serviceable: "Wounds in War," by Surgeon-General W. F. Stevenson, of the British Army; "Gunshot-wounds," by Major C. G. Spencer, of the British Army; "Military Surgery," by Surgeon-General Robert M. O'Reilly, U. S. A., in Vol. IV of "Keen's Surgery"; "Naval Surgery," by Surgeon-General P. M. Rixey, U. S. N., in Vol. IV of "Keen's Surgery"; Treves, in the "British Medical Journal," 1900; Senn, in "The Hispano-American War"; Makin, "Surgical Experiences in South Africa"; "Chirurgie de Guerre," Paris, 1897, by Constans; "Surgical Notes from the Military Hospitals in South Africa," by Dent, in the "British Medical Journal," 1900; "Gunshot-wounds," by Maj. Wm. C. Borden, U. S. A., in the "American Practice of Surgery," by Bryant and Buck, Vol. II; "Delorme's Traite de Chirurgie de Guerre"; "Recent Reports of the Surgeon-General of the U. S. A. and of the Surgeon-General of the U. S. Navy"; the Chitral Campaign," by H. C. Thomson; "Les Projectiles des Armes de Guerre," by Nimier and Laval; "Volumes of the Proceedings of the Associations of Military Surgeons, U. S. A."; Follenfant, in "Archives de Medicine et de Pharmacie Militaire," July, 1906; "Reports of Military Observers in Manchuria during the Russo-Japanese War," by Maj. Charles Lynch, Medical Department, General Staff, U. S. A., 1907; Neate, "The Military Surgeon," 1911; "United States Magazine Rifle," Government Printing Office, Washington, 1909; Borden, in "Keen's Surgery," Vol. VI; Surgeon-General Stokes, U. S. N., in "American Practice of Surgery," 1911; Wadsworth, in "International Clinics," Vol. IV, Twentieth Series. (For gunshot-wounds of special structures, see Bones, Joints, Abdomen, Brain, etc.)

**Poisoned wounds** are those into which some injurious substance, chemical or bacterial, was introduced. This poison may be microbic and capable

of self-multiplication, or it may be chemical, and hence incapable of multiplication. There are three classes of poisons:[1] (1) mixed infection, as septic wounds, dissection-wounds, and malignant edema; (2) chemical poison, such as snake-bites and insect-stings; and (3) infection with such diseases as rabies, glanders, etc.

**Septic** or **infected wounds** are those which putrefy, suppurate, or slough. Septic wounds should be opened freely to secure drainage, and hopelessly damaged tissue should be curetted or cut away. The wound should be washed with peroxid of hydrogen and then with corrosive sublimate, dusted with iodoform or orthoform, either drained by a tube or packed with iodoform gauze, and dressed by hot antiseptic fomentations. The part must be kept at rest and internal treatment should be stimulating and supporting. If lymphangitis arises, the skin over the inflamed vessels and glands is to be painted with iodin and smeared with ichthyol, and quinin, iron, and whisky are given internally. The temperature is watched for evidence of general infection or intoxication. The patient must be stimulated freely, nourishing food is given at frequent intervals, pain is allayed by anodynes if necessary, and sleep is secured. In infected wounds of the extremities Bier's treatment is very useful.

**Dissection-wounds** are simple examples of infected wounds, and they present nothing peculiar except virulence. They affect butchers, cooks, surgeons who cut themselves while operating on infected areas, those who make postmortems, and those who dissect. A dissection-wound inflicted while working on a body injected with chlorid of zinc possesses but few elements of danger unless the health of the student is much broken down. If a wound is simply poisoned by putrefactive organisms, there is rarely serious trouble. Postmortems are peculiarly dangerous when the subject has died of some septic process. When a wound is inflicted while dissecting, wash it under a strong stream of water, squeeze, and suck it to make the blood run, lay it open if it be a puncture, paint it with pure carbolic acid, and dress it with iodoform and hot antiseptic fomentations. Trouble, of course, may follow, but often it is only local, and a small abscess forms. It should be treated by hot antiseptic fomentations and early incision. Occasionally lymphangitis arises, adjacent glands inflame, and constitutional symptoms arise. It is rarely that true septicemia or pyemia arises unless the wound was inflicted while making a postmortem upon a person dead of septicemia or while operating on a septic focus. If glands enlarge and soften, it may be necessary to remove them surgically.

**Malignant edema** or **gangrenous emphysema** arises most commonly after a puncture. It is due to a specific bacillus which produces great edema. The emphysema which soon arises is due to mixed infection with putrefactive organisms. Pus does not form, but gangrene occurs. The disease is identical with one form of traumatic spreading gangrene (see page 170).

**Symptoms.**—These are identical with those of traumatic spreading gangrene with emphysema.

There is a rapidly spreading edema, followed by gaseous distention of the tissues and by gangrenous cellulitis. The zone of edema is at the margin of the emphysema, and the process spreads rapidly. The emphysematous zone crackles when pressed upon. The area of edema is covered with blebs which contain thin, putrid, reddish matter, and the skin becomes mottled. If a wound exists, the discharge will be bloody and foul. If incisions are made, a thin, brown, offensive liquid flows out. High fever rapidly develops, the patient becomes delirious, and often coma arises. In most cases death ensues in from twenty-four to forty-eight hours.

[1] "American Text-Book of Surgery."

**Treatment.**—If malignant edema affects a limb after a severe injury amputate at once, high up. If it affects some other part or begins in a limb after a trivial injury, make free incisions, employ hot, continuous antiseptic irrigations or the hot antiseptic bath, and stimulate freely (see page 171).

**Stings and Bites of Insects and Reptiles: Stings of Bees and Wasps.**—A bee's sting consists of two long lances within a sheath with which a poison-bag is connected. The wound is made first by the sheath, the poison then passes in, and the two barbed or twisted lances, moving up and down, deepen the cut. The barbs on the lances make it difficult to rapidly withdraw the sting, which may be broken off and remain in the flesh. Edematous, discolored swelling quickly arises. The pain is severe and burning. Besides bees, hornets, yellow jackets, and other wasps produce painful stings like bee-stings. The sting of a wasp is rarely broken off in the tissues because the beards on the darts are shorter; hence the sting is not so firmly fixed in the flesh, and also because these insects are more rapid and nimble in their actions. Stings of bees and wasps seldom cause any trouble except pain and swelling. In rare cases syncope occurs. Widespread urticaria may develop. Erysipelas or phlebitis may arise. In some unusual cases a bee-sting is fatal; persons have been stung to death by a great number of these insects.

**Symptoms.**—If general symptoms ensue, they appear rapidly, and consist of great prostration, vomiting, purging, and delirium or unconsciousness. These symptoms may disappear in a short time, or they may end in death from heart-failure. Stings of the mouth may cause edema of the glottis.

**Treatment.**—To treat a bee-sting, extract the sting with splinter forceps if it has been broken off and is visible in the wound. If it is not visible, squeeze the part lightly in order to expel it, or at least expel the poison. Pressure may be most satisfactorily made by means of the barrel of a key. The poison is counteracted by touching with ammonia or washing the part in ammonia-water, touching with pure carbolic acid, painting with tincture of iodin, or soaking in a strong solution of common salt or carbonate of sodium. The part may be dressed with lead-water and laudanum, a solution of washing-soda, or a solution of common salt. If constitutional symptoms appear, stimulate.

**Other Insect-bites and Stings.**—If a tick bites a person it clings to the victim. If an attempt is made to pull it off the barb remains in the tissues and an abscess follows. If a little ammonia is dropped on a tick the insect will at once withdraw its barb. A tick bite never causes constitutional symptoms. The mandibles of a *poisonous spider* are terminated by a movable hook which has an opening for the emission of poison. The bite of large spiders is productive of inflammation, swelling, weakness, and even death. The bite of the poisonous spider of New Zealand produces a large white swelling and great prostration; death may ensue, or the victim may remain in a depressed, enfeebled state for weeks or even for months. The *tarantula* is a much-dreaded spider. The scorpion has in its tail a sting. The sting of the *scorpion* produces great prostration, delirium, vomiting, diaphoresis, vertigo, headache, local swelling and burning pain, followed often by fever and suppuration, and occasionally even by gangrene, but it is rarely fatal. *Centipedes* must be of large size to be formidable to man, and the symptoms arising from their stings are usually only local.

**Treatment.**—To treat the bite of a poisonous spider or sting of a scorpion tie a fillet above the bitten point; make a crucial incision, favor bleeding, and paint the wound with pure carbolic acid or some caustic or antiseptic (if in the wilds, burn with fire or gunpowder); dress antiseptically if possible, and stimulate as constitutional symptoms appear. Slowly loosen the ligature after symptoms disappear. Chloroform stupes and ipecac poultices are recommended; also puncture by a needle and rubbing in a mixture of 3 parts of

alcohol and 1 part of camphor (Bauerjie). Antiscorpion venene is recommended by Todd for scorpion stings.

**Myiasis.**—When the larvæ of dipterous insects are deposited in the tissues the condition is called myiasis. Certain varieties of flies sting with the ovipositor and lay their eggs in the skin or in the mucous membrane of the nose. The bot-fly may do this, and the larvæ or maggots of bot-flies are called bots. Inflammation arises in the area containing the ova and suppuration may occur. Myiasis is most common in tropical and subtropical countries, but the bluebottle fly may be responsible for the condition in temperate climates.

**Treatment.**—Incision and application of pure carbolic acid to kill the larvæ. If in the nasal passages inhalation of chloroform may prove fatal to larvæ.

**Maggots in Wounds.**—In tropical countries especially flies may lay eggs in wounds and maggots form in immense numbers. Larrey saw many such cases. I have seen maggots several times in foul leg ulcers. Antiseptic dressings prevent such an occurrence and antiseptics will destroy larvæ. Iodin is very successful.

**Chigger.**—The chigger or sand flea is common in the tropics and subtropics. The female when impregnated may enter an abrasion or may pass through "the soft skin, especially in the sole between the toes" or "around the nails" (Madden, in a "System of Surgery," edited by C. C. Choyce).

Inflammation occurs and the body of the flea is recognizable as a small black spot. Suppuration may occur and the flea may be cast out with the pus. Sinuses may arise and persist. There is some danger of erysipelas, tetanus, and gangrene (Ibid.).

**Treatment.**—Apply a hot solution of bicarbonate of soda for some hours, enlarge the opening with a knife, remove the insect without breaking it up, and dress the part antiseptically (Ibid.).

**Fish Stings.**—The spines of certain fish inflict a poisoned wound, as they are covered with an irritant material obtained from the skin. The spines of the catfish are known to do this. Certain fish actually inject poison along a spine, the poison coming from a receptacle or bag. These wounds are very painful, are septic, and are apt to be followed by lymphangitis, erysipelas, erysipeloid, gangrene, or general sepsis.

**Treatment.**—Incise, favor bleeding, swab with pure carbolic acid or iodin, and apply hot antiseptic fomentations.

**Snake-bites.**—The poisonous snakes of America comprise the copperheads (red vipers or upland moccasins), water moccasins (rice snakes or cottonmouths), harlequin snakes (coral snakes), and rattlesnakes. The cobra of India is the most deadly of reptiles. In some countries great numbers of people and the lower animals are killed by poisonous serpents. In India during 1898, 21,921 persons and at least 80,000 cattle were killed by snakes ("Brit. Med. Jour.," Nov. 25, 1899). The coral snake is found in the southeastern United States, and is common in South Carolina, Georgia, and Florida. It is often discovered in sweet potato fields.

The water moccasin, which is semi-aquatic, infests "the lagoons and sluggish waterways of the southeastern portion of the United States" ("Reptiles of the World," by Raymond L. Ditmars).

The copperhead, which is a variety of mocassin, is found east of the Mississippi River from Florida to Massachusetts, and west of the Mississippi in Texas. In the South it lives on plantations, in the North in or near forests (Ibid.). In practically every part of the United States there dwells some variety of rattlesnake. Fifteen species are catalogued as dwelling within our borders (Ibid.). Some inhabit prairie, some desert, some rocky land, some timber regions, some dwell adjacent to water. The diamond-back rattlesnake is the most poisonous serpent of the United States. A small rattler is not nearly so dangerous to life as a big rattler. Next to a big rattlesnake in poisonous

power comes the water moccasin. Mr. Ditmars characterizes the coral snake as "highly formidable" and the copperhead as "highly venomous." Statistics seem to contradict the belief that the copperhead is very dangerous. Prentiss Willson ("Jour. Am. Med. Assoc.," Aug. 27, 1910) has established 99 instances of persons bitten by copperheads. There were only 5 deaths. It used to be taught that there is no essential difference in the action of venoms of different varieties of snakes, and that the venom of an Indian cobra is practically identical with the venom of an American rattler, any apparent difference in action depending upon difference in toxic power and the different dose of poison introduced. We now know that there are essential differences in venoms (Leonard Rogers, in "Lancet," Feb. 6, 1904). The natural toxic power of the poison varies in different species and also in different members of the same species. Poison injected into a vein may prove almost instantly fatal. The poison is not absorbed by the sound mucous membranes. Poison is harmless when given by the mouth and swallowed, but if directly introduced into the intestines of an animal it is certainly fatal. The pancreatic ferment destroys the toxic power of the venom (R. H. Elliot, in "Brit. Med. Jour.," May 12, 1900). The venom is discharged through the channeled fangs of the reptile, having been forced out by contractions of the muscles of the poison-bag. The coral snake, like the cobra, has short and rigid fangs. Rattlesnakes, cotton-mouths, and copperheads have long and movable fangs. A coral snake bites like a cobra. It grasps the object, sinks in its fangs, and then advances its fangs by chewing, thus inflicting several wounds. In viperine snakes the teeth lie along the back of the mouth and are only erected when the reptile strikes. The maxillary bones of the rattlesnake are very short and move with great freedom at the prefrontal articulation. The fangs are canaliculated, are attached to the maxillary bones, and move with the bones. The poison gland drives the poison into the canals of the fangs. The canal emerges from the front of each fang near the tip. The fangs, when depressed, are carried in a fold of mucous membrane. When the animal is ready to strike the fangs are erected into a vertical position and carried to the front of the mouth. Cope describes the movement used by the rattlesnake in biting. The body springs forward, but never more than two-thirds of the reptile's length, the jaws seize the tissues and then the fangs penetrate and move to and fro as the poison emerges from them. Snake-poison is a thin, greenish-yellow, turbid, sterile fluid, of acid reaction and of a distinctive odor. The two chief poisonous principles are called venom-peptone and venom-globulin (Gustave Langmann, "Medical Record," Sept. 15, 1900).

**Symptoms.**—Rogers ("Lancet," Feb. 6, 1904) divides poisonous snakes into two classes: the *colubrines* (of which the cobra is an example) and the *viperines*, which are not so poisonous (this class includes rattlesnakes and puff adders). Colubrine venom, according to this observer, causes paralysis of the respiratory center and of the motor end organs of the phrenic nerves, destruction of red blood-corpuscles, lessened coagulability of blood, and death by respiratory paralysis. Viperine venom causes paralysis of the vasomotor center and great destruction of red corpuscles. Some viperine venoms may cause thrombosis, and death from any one of them is due to vasomotor paralysis. The venom of some snakes, Rogers says, contains a mixture of the above-mentioned venoms (among such snakes are the Australian colubrines and the American pit-adders). The mortality from snake-bites varies. The mortality in India from cobra bites is about 25 per cent. (Sir Joseph Fayrer). The mortality in America from rattlesnake bites is about the same. According to Willson the mortality from copperhead bites is only 5 per cent. ("Jour. Am. Med. Assoc.," Aug. 27, 1910). The local symptoms are: pain, soon becoming intense; mottled swelling of the bitten part, which swelling may be enormous, and which is due to edema and extravasation of blood, and assumes a pur-

puric discoloration. The *bite of a cobra* produces inflammation and marked spreading edema. It may be recovered from without symptoms or with trivial symptoms; it may induce profound systemic involvement. The general symptoms begin in a comparatively few minutes. The coagulating power of the blood is lost, and there is great destruction of red corpuscles. The patient is terror-stricken and soon becomes unable to stand because of weakness of the legs. Glossopharyngeal paralysis arises, and talking and swallowing become impossible. There is a profuse flow of saliva, perhaps nausea and vomiting. The patient may be dull mentally, but is not unconscious. The paralysis becomes widespread, and finally the diaphragm and respiratory center become involved, and death occurs from respiratory paralysis. Artificial respiration may prolong life for hours (Sir Joseph Fayrer). Bad cases usually die in three or four hours, but life may last for many hours. A *rattlesnake* bite produces severe pain and mottled swelling from blood extravasation. In some cases there is enormous swelling from edema and blood; the discoloration in such a case is purpuric. The blood of the victim quickly undergoes hemolysis and loses the power of coagulation. It was previously stated that in laboratory experiments it has been shown that viperine poison may produce thrombosis, but it does not do so in man, as it contains a very small amount of the coagulating element (Rogers). Extravasations of blood occur in serous and mucous membranes and in the skin, petechial spots frequently arising upon the cutaneous surface. There may be free bleeding from mucous surfaces and great extravasation beneath the conjunctivæ. These blood extravasations are due, according to Flexner, to destruction of vascular endothelium. General symptoms begin in from a few minutes to several hours. The symptoms are those of profound shock, possibly with delirium, the vasomotor center being exhausted and finally paralyzed. There are usually muscular twitching and convulsions, and finally paralyses are noted in most cases (pharyngeal palsy, paraplegia, and ascending paralysis). There may be complete consciousness, or there may be lethargy, stupor, or coma. Death may occur in about five hours, but, as a rule, it is postponed for a number of hours. If death is deferred for a day or more, profound sepsis comes upon the scene, with glandular enlargement, suppuration, and sometimes gangrene.

**Treatment.**—Cases of snake-bite must, as a rule, be treated without proper appliances. The elder Gross was accustomed to relate in his lectures how he had seen an army officer blow off his finger by a pistol the moment after it was bitten by a rattlesnake, and thus escape poisoning. If the bite is upon a limb, and it usually is, twist several fillets at different levels above the bite to prevent the dissemination of the poison from the limb throughout the body. If possible the fillets should be elastic, but in an emergency any available material must be used. As soon as fillets have been applied above the bitten area make crucial incisions to the depth of each bite. A rattlesnake bite, a copperhead bite, and a water mocassin bite show but two punctures. A coral snake bite shows several. After incising suck or cup it if possible, and cauterize it with pure nitric acid or by a cautery. There is no danger in sucking the wound, provided there are no abrasions upon the lips, cheeks, or tongue. Before sucking, fill the mouth with a dilute solution of permanganate of potash, to oxidize and thus destroy the poison. An expedient among hunters is to cauterize by pouring a very little gunpowder on the excised area and applying a spark, or by laying a hot ember on the wound. Some surgeons inject in many places about the wound a 10 per cent. watery solution of chlorid of calcium to chemically neutralize the poison. It is taught by others that if a man is bitten by a large and deadly snake, the surgeon, if one is at hand, should at once amputate well above the bite.[1] Wynter Blyth pointed out that permanganate of potassium mixed

[1] Charters James Symonds, in "Heath's Dictionary of Practical Surgery."

with an equal weight of cobra venom renders the venom inert. A number of surgeons have treated snake-bites by injecting in and about the wound a 1 per cent. solution of permanganate of potassium, but this plan is inefficient. Rogers ("Lancet," Feb. 6, 1904) says we should tie fillets around the limb above the bitten part, take a knife and enlarge the wounds, and rub in crystals of permanganate. The fillets are not to be removed suddenly, and they had best be kept on for some time. Remove the highest constricting band first; if no symptoms come on after a time, remove the next, and so on; if symptoms appear, reapply the fillets. Whatever local treatment is employed, stimulants are given. Some give strychnin hypodermatically; others, ether; others, digitalis. Halford, of Australia, advocated the intravenous injection of ammonia (10 min. of strong ammonia in 20 min. of water). Adrenalin as given in shock is indicated if there is a marked fall in blood-pressure, and autotransfusion and external heat are also indicated. If the respiration is failing, artificial respiration and oxygen inhalation are required. Few beliefs are more tenaciously held than that large amounts of whisky or brandy should be given to the victim of snake venom. And yet this belief is false. In a person badly poisoned by snake venom the medullary centers are depressed and threatened with paralysis. Large doses of alcohol increase this tendency and may hasten death. It is well known that if a drunken man is bitten by a large poisonous snake he is practically certain to to die, because the depression produced by alcohol is enormously accentuated by the venom. Moderate doses of whisky or brandy are useful (1 to 1½ ounces every half-hour). The wounds made by the incisions of the surgeon are kept open for a number of days by the insertion of bits of rubber tissue, and warm and moist antiseptic dressings are used. Attempts are being made to obtain a curative serum. Animals can be rendered immune by giving them at first small doses of the poison and gradually increasing the amount administered. It is asserted that the serum of immune animals will cure a person bitten by a venomous snake. Cures have been reported after the use of Calmette's antivenene serum. Antivenene is obtained by immunizing a horse by injecting attenuated venom. The mixture to be attenuated consists of 80 parts of cobra venom and 20 parts of viperine venom. It takes a number of months to obtain strong antivenene. The dose is from 10 to 20 c.c. hypodermatically, repeated if necessary in three or four hours. It seems certain, however, that no single serum can antidote the venom of all varieties of serpents (A. T. F. Macdonald, of Australia), and it has been shown that, though Calmette's antivenene is antagonistic to colubrine venom, it is inert against viperine venom. Again, as Rogers says, it deteriorates quickly in hot climates and is seldom on hand when wanted. The horse can be immunized against rattlesnake venom, and antivenene obtained from the horse may be used against rattlesnake poison.

The **poisonous lizard** (*Gila monster*), of the southwestern United States, can certainly kill small animals, but it is doubted by many that its bite is ever fatal to man. When we recall, however, that small animals which die of the poison present symptoms identical with those produced by serpent venom, we should regard the Gila monster with careful respect.

The lizard bites and hangs on. Each tooth of the lower jaw has a conducting channel for poison and there is a poison gland for each tooth.

The treatment of a bite of the Gila monster is practically the same as for a snake-bite.

**Anthrax (malignant pustule, charbon, wool-sorters' disease, milzbrand,** or **splenic fever)** is a term used by some as synonymous with ordinary carbuncle, but it is not here so employed. It is a specific contagious disease resulting from infection with the bacillus of anthrax. Cattle anthrax has long been known. Virgil refers to it (Ponder, in "Lancet," Nov. 4, 1911).

Pollender showed more than fifty years ago that a rod-shaped organism is present in the blood of animals dying from splenic fever. Duvaine insisted that the organisms caused the disease. Koch proved it in 1876. Animal anthrax is particularly common in the East and in Russia, and is frequently met with in Germany, Italy, and South America. In some regions so many cases arise year after year that the district obtains an evil notoriety. It is stated that in Novgorod, Russia, in four years "56,000 horses, cattle and sheep, and 528 men perished from anthrax" (Frank S. Billings, in "Twentieth Century Practice"). It is a rare disease in the United States. In Philadelphia cases occasionally occur in workers in the woolen mills. The author has seen 5 cases of human anthrax, 4 of which occurred in Philadelphia and 1 in New Jersey. Herbivora are most liable, next omnivora, but carnivora seldom suffer. Anthrax, as met with in man, is a disease contracted in some manner from an animal with splenic fever. It may be contracted by inoculation while working around diseased animals, while handling or tanning their hides, or sorting their hair or wool; brush-makers, spinners, workers in horn and combers, rag sorters, veterinary surgeons, clippers, stockmen, farmers, and butchers may become inoculated. Infection may take place through the hair-follicles of unbroken skin. Menschig reported 2 such cases ("N. Y. Med. Jour.," Nov. 18, 1905). Anthrax may be conveyed by eating infected meat or by drinking infected milk. Flies may carry the poison. Inhalation of poisoned dust may infect the lungs. Catgut ligatures may be contaminated and carry the poison. Blood-stained wools and hides are a particular peril. It is stated that in England 1 out of 7000 leather makers dies of anthrax each year (Ponder, in "Lancet," Nov. 4, 1911). Ponder points out that 40 per cent. of English cases (arising among those working in the hide, skin, and leather industries) are due to Chinese or East Indian goods; that no anthrax has ever been traced to wet salted hides; that infection may arise at wharf, dockyard, or tannery; that the pulmonary form occurs particularly among wool workers; that among those who work with hides, skin, and leather cutaneous anthrax is the common form, and intestinal anthrax is extremely rare.

Fig. 133.—Anthrax. Case in author's wards in Philadelphia Hospital, recovered. Treated by ipecac both externally and internally.

In all probability many slight cases are recovered from without being recognized. This would explain the fact that it is particularly among recent employees that anthrax seems to occur (Mitchell, "Brit. Med. Jour.," April 4, 1911). Many older employees may be immune because they have had the disease. Many attempts have been made to render animals immune (Pasteur, Woolbridge, Hankin). Pasteur's method has been used in France with decided success, but with less success in other countries. Pasteur devised a method for vaccinating animals against anthrax by injecting attenuated cultures beneath the skin. First, a much-attenuated culture is used and later a stronger one. When an animal has been actively immunized, its blood-serum contains pro-

tective materials of a specific nature and may be used therapeutically. The immunity is regarded by some authorities as "a phagocytic immunity" (Jordan's "General Bacteriology"). Certain organisms are antagonistic to anthrax (the streptococcus of erysipelas, the pneumococcus, the Micrococcus prodigiosus, and the Bacillus pyocyaneus).

**Forms of Anthrax.**—There are two forms of the disease—external and internal. Internal anthrax may be intestinal from eating diseased meat, laryngeal or pulmonary from inhalation of poisoned dust. Intestinal anthrax arises only when the bacilli in the meat contain spores. Koch and others have pointed out that the non-sporulating bacteria are destroyed by the gastric juice. Internal anthrax is quick in progress, and death sometimes takes place within twenty-four hours of the onset of symptoms. It cannot be diagnosticated with certainty unless bacilli are found in the blood.

**External anthrax** may be *anthrax carbuncle* or *anthrax edema*. *Anthrax carbuncle* or *malignant pustule* appears on an exposed portion of the body, especially the face, neck, hand, or finger, in 90 per cent. of cases of external anthrax. I have seen one upon the temple and one on the neck. It appears in from twenty-four hours to six days after inoculation, and presents an itching, burning papule with a purple center and a red base; in a few hours the papule becomes a vesicle which contains bloody serum, and the tissues about the papule become swollen, reddened, and indurated. The vesicle bursts and dries, the base of it swells and enlarges, other vesicles appear in circles around it, and there is developed an "anthrax carbuncle," which shows a black or purple elevation with a central depression surrounded by one or more rings of vesicles. The surrounding tissues become purple, and great edema may spread widely, the vesicles grow very large, new vesicles form, and gangrene may occur. Pain is trivial or absent. Lymphatic enlargements occur, but pus does not form. The constitutional symptoms may rapidly follow the local lesion, but may be deferred for a week or more. The patient feels depressed, has obscure aches and pains, and is feverish, but usually keeps about for a short period. In some cases with constitutional symptoms there is no elevated temperature, and such cases are frequently fatal. After a time he is apt to develop rigors, high irregular fevers, sweats, acute fugitive pains, diarrhea, delirium, typhoid exhaustion, dyspnea, cough, and cyanosis. The carbuncle of anthrax is distinguished from ordinary carbuncle by the central depression, the adherent eschar, the absence of pain, tenderness, and suppuration of the first, as contrasted with the elevated center, the multiple foci of suppuration and sloughing, and the more severe pain usual in the second. If anthrax has a visible lesion and the constitutional symptoms are slight or absent, the chance of cure is good. In cases which get well a line of demarcation forms about the pustule and the gangrenous area is rather rapidly cast off, a granulating surface remaining.

*Anthrax Edema.*—An area of edema surrounds a malignant pustule and often spreads widely, but in cases of external anthrax without a pustule there is edema alone. This lesion occurs in connective tissue, especially loose tissue. It is a spreading, livid edema, with an ill-defined margin. There is no pain and usually no vesication and no fever. In severe cases, however, there is fever, vesicles form, and gangrene may arise. Anthrax edema differs from cellulitis in the absence of pus formation, and from malignant edema by the less disposition to result in gangrene. Two of the cases I have seen were anthrax edema. In Horwitz's case in the Philadelphia Hospital the forearm, arm, and shoulder were enormously edematous. In Keen's case in the Jefferson College Hospital the forearm and arm were edematous.

**Bacilli in the Blood in All Forms of Anthrax.**—In some cases they are found, in others they are not. To find them is extremely ominous, as most cases with

bacilli-laden blood die. Bacilli are seldom found until thirty-six hours before death (Mitchell, in "Brit. Med. Jour.," April 1, 1911). In fatal cases the blood always contains bacilli.

**Prognosis.**—When bacilli are found in the blood the prognosis is very bad. Becker knows only of 3 such cases which survived ("Münchener medicinische Wochen.," Jan. 23, 1912). Of Becker's 44 cases which had no bacilli in the blood, 43 recovered. There were 11 with bacilli in the blood and all died. A case which is going to be fatal always shows bacilli in the blood, at least within thirty-six hours of death. Even apparently bad cases with negative blood findings are apt to recover (Becker, Ibid.). During six years the Board of the Bradford woolen industry reported that in 71 cases of anthrax 24 died (15 internal and 9 external). The former estimate of the death-rate from external anthrax was from 25 to 30 per cent. If a lesion is upon the face the prognosis is much worse than if it is upon an extremity, and if upon the upper extremity it is worse than if it is upon the lower. It is claimed that death-rate has been notably reduced by modern treatment, and under serum treatment is said to be but little over 6 per cent. In a series of 15 cases of external anthrax reported by Royer and Holmes there were 3 deaths ("Therapeutic Gazette," Jan., 1908). Eleven of these cases received serum, and of the 11, 2 died.

Pulmonary anthrax and intestinal anthrax have been regarded as invariably fatal, but vastly better results may be looked for hereafter.

**Prevention of Human Anthrax.**—Spores are the great danger. Unfortunately, there is no known disinfectant for wool which will kill spores and not injure fabric (London letter in "Jour. Am. Med. Assoc.," Feb 17, 1912). Blood-stained wools are the great peril. The blood contains the spores and formalin will not penetrate dried blood. Wet salted hides are entirely safe, and governmental regulation to prevent the importation of any other kind would prevent anthrax among those who work in hide, skin, and leather industries (Ponder, in "Lancet," Nov. 4 1911).

**Treatment of External Anthrax.**—If a person is wounded by an object suspected of carrying the infection, cauterize the wound by the hot iron or fuming nitric acid. A sufferer from anthrax must be isolated in a well-ventilated room. All dressings are to be burned, all discharges asepticized, and after the removal of the patient the bed-clothes are burned and the room disinfected. If there are no bacilli in the blood a malignant pustule should be entirely excised, and the wound mopped out with pure carbolic acid or burned with the hot iron. If there is an extensive area of edema, it should be freely incised down to the deep fascia at several points. The area about an anthrax edema or an excised pustule should be injected every sixth hour with a 5 per cent. solution of carbolic acid. The wound and the edematous area should be dressed with hot antiseptic fomentations, and, if dealing with an extremity, a splint is applied. Excision should be practised for pustule even when glands are enlarged. When excision cannot be performed make crucial incisions through the lesion, mop the wounds with pure carbolic acid, and inject about and in the pustule carbolic acid (1: 20) every six hours until the disease abates or toxic symptoms appear. Dress the part as directed above. In a successful case the adherent eschar is finally separated by the influence of the fomentations. Davaine advised the following plan: Inject the pustule and the tissues about it at many points every eight or ten hours with 1 part of tincture of iodin diluted with 2 parts of water or with a 10 per cent. solution of carbolic acid, or with a 0.1 per cent. solution of corrosive sublimate. Dress with wet antiseptic gauze and apply an ice-bag. Personally I would not use an ice-bag on an area of infection, but would prefer heat. In Keen's very severe case of anthrax edema multiple incisions were made, carbolic acid was injected into sound tissues above the edema, and the part was dressed with hot antiseptic

fomentations. Recovery followed. Constitutional treatment in anthrax edema, malignant pustule, or internal anthrax must be sustaining and stimulating. Maffucci gives carbolic acid internally, and also uses it externally. Davies-Colley uses ipecac locally and gives 5 gr. by the mouth every four hours. Statistics indicate that the serum treatment is of the greatest value. The material is known as Sclavo's serum; it is obtained from the immunized ass, and it was introduced into practice in 1897. It is perfectly harmless and may be given in a vein or subcutaneously. Sclavo injects 40 c.c. in different regions of the wall of the abdomen. Usually the temperature begins to fall in an hour; if improvement is not obvious in twenty-four hours, the dose is repeated. Intravenous injection is reserved for severe cases, the dose being 10 c.c. into a subcutaneous vein of the dorsal surface of the hand. The serum can do no harm and should always be given. If given early, all cases but very severe ones will recover (Legge's Nilray Lectures, "Brit. Med. Jour.," March 18, 1905). Becker ("Münch. med. Woch.," Jan. 23, 1912) has given salvarsan, and he believes it saved the life of a very ill man. The persistence of anthrax infection in a room was well shown in the record of Keen's case. The infection lingered on the floor of the room in which the patient had been operated upon for a long time. Three disinfections were necessary before it became impossible to obtain anthrax bacilli from the contaminated floor. This indicates that such a case should be operated upon in a room not regularly used for operations.

**Hydrophobia, Rabies, or Lyssa.**—Hydrophobia is a spasmodic and paralytic disease due to inoculation with virus from a rabid animal. Inoculation is nearly always through a wound, but cases occur after the licking of the hand by a diseased dog. The disease does not appear to arise except as the result of inoculation. It is most common in dogs and wolves, but it may develop in cats, horses, goats, foxes, cattle, sheep, and pigs. Cats are said to cause 6 per cent. of the cases (Cumming, in "Jour. Am. Med. Assoc.," May 18, 1912). Lack of water is never a cause. It is far more common in the carnivora than the herbivora. In Russia wolves are responsible for many cases. It is said that poultry may suffer from it. Human hydrophobia in most instances follows dog bites. Roux estimates that about 14 per cent. of the people bitten by mad animals develop the disease. If the bite is on an exposed part, it is far more apt to cause rabies than if the rabid animal's teeth passed through clothing. The saliva is the usual vehicle of contagion, but other fluids and tissues contain the virus, especially the brain and cord. The blood and urine do not contain it. Hydrophobia has been known for centuries. It is not spoken of by Hippocrates, but is described in animals by Aristotle, Pliny, and Celsus, and is alluded to by Ovid, Horace, Virgil, and Plutarch. Celsus first described the disease in man and first used the term "hydrophobia." At the present day some ardent antivivisectionists dispute its existence. The fact that an infant bitten by a rabid animal may develop rabies proves that the disease is not due to the imagination. Hydrophobia is almost invariably fatal. No causative micro-organism has been demonstrated. One must exist, but it probably escapes detection because of its very small size.[1] Negri has discovered in the central nervous system bodies which are probably protozoa and are perhaps the cause of the disease. They are called Negri bodies. The poison cannot gain entrance through sound mucous membrane. It used to be thought that the disease was particularly apt to arise in hot weather, but it is now known that it may occur any time of the year. There was a veritable epidemic of it among the animals in Greenland in 1860, and at this time the temperature averaged 25 degrees below zero (F. W. Dudley, in "Jour. Am. Med. Assoc.," Dec. 19, 1908). It is common in Russia. No

[1] Since the above was written Noguchi has announced the discovery of the causative protozoön.

portion of the world is completely exempt. No constant postmortem lesions have been certainly demonstrated in those dead of rabies. Gowers believes that in the spinal cord there is hyperemia, but no infiltration with cells, whereas in the medulla, especially about the respiratory center, there are hyperemia and cellular infiltration of the perivascular spaces; but such perivascular infiltration can occur in some other acute conditions and hence is not characteristic. What is known as the *rabic tubercle* is found in the medulla and about the motor cells of the upper part of the spinal cord. Each tubercle consists of an aggregation of embryonal cells, which destroy and finally replace the nerve-cells which they surround. Babés thinks the tubercle characteristic. Infiltration of the ganglia with epithelioid cells and round cells has been held by some to be characteristic, but both the rabic tubercle and ganglion infiltration occur in other conditions. The disease is extremely rare in the United States and the author has not seen a single case.

If a dog is poisoned with barium carbonate the symptoms are similar to those of rabies ("Penna. Health Bulletin," Aug., 1909).

**Symptoms.**—The period of incubation of human hydrophobia is from a few weeks to several months, and it has been alleged that it may even be two years, but it is very doubtful if there is ever a period of incubation of over six or seven months. The average incubation period in man is forty days (Ravenel). The initial symptoms are mental depression, anxiety, sleeplessness, restlessness, headache, malaise, and often pain or even congestion in the cicatrix. The anxiety which is usually present may be deepened into actual fear. In dogs the condition of fear is so evident that Cælius Aurelianus centuries ago called the disease *pantophobia* (fear of everything). The previously mentioned symptoms are quickly followed by dysphagia. It is not only water that is difficult to swallow, but everything the patient tries to drink or eat. The difficulty in swallowing results apparently from apnea produced instantly when an attempt is made to swallow. Curtis points out that the difficulty is not spasm of the pharynx and larynx, but a sense of immediate suffocation due to reflex stimulation of respiratory inhibition. If spasms occur—and they may occur—they are secondary to this suffocative state, a state in which the action of the diaphragm ceases for a time. The air-passages become congested and the sufferer makes frequent and painful efforts to expel thick mucus, and the efforts produce paroxysms of suffocation. Between the paroxysms the patient is evidently somewhat breathless, and Warren tells us that his speech is not unlike that "of a child who has recently been crying and is endeavoring to control itself" ("Surgical Pathology and Therapeutics"). As the condition grows worse, suffocative attacks, which were at first induced by attempts at swallowing, come to be caused also by bright lights, sudden or loud noises, irritations of the skin, or even thinking of swallowing. At length suffocative paroxysms occur spontaneously and the patient jumps, or hurls himself about, or the muscles of the entire body are thrown into clonic spasm. Tonic spasm does not occur. A condition of general hyperesthesia exists. The mind is usually clear, although during the periods of excitement there may be maniacal furor with hallucinations which pass away in the stage of relaxation. The temperature is moderately elevated (101° to 103° F. or higher). The spasmodic stage lasts from one to three days, and the patient may die during this stage from exhaustion or from asphyxia. If he lives through this period, the convulsions gradually cease, the power of swallowing returns, and the patient succumbs to exhaustion in less than twenty-four hours, or he develops ascending paralysis which soon causes cardiac and respiratory failure. In what is known as *paralytic rabies*, a very rare form of the disease in human beings, the attack comes on with the same early symptoms met with in the commoner form,

but paralysis soon begins about the bitten part and spreads to all the limbs and to the trunk.

In hydrophobia death is almost inevitable. Practically all cases in which it is alleged that recovery ensued were not true hydrophobia, but hysteria. An exception must be made of Murri's case. Wood says that in hysteria, especially among boys, "beast-mimicry" is common, the sufferer snarling like a dog; and in the form known as "*spurious hydrophobia*," in which there may or may not be convulsions, there are a dread of water, emotional excitement, snarling, and attempts to bite the bystanders (in genuine hydrophobia no attempts are made to bite, and no sounds are uttered like those made by a dog).

Lyssa is separated from lockjaw by the paroyxsms of suffocation and the absence of tonic spasms in the former, as contrasted with the fixation of the jaws and the tonic spasms with clonic exacerbations of lockjaw.

**Treatment.**—When a person is bitten by a supposed rabid animal and is seen soon after the injury, constriction should be applied if possible above the wound, the wounded area should be incised, allowed to bleed freely, and should then be washed with a 5 per cent. solution of formaldehyd and dressed for twelve hours with a like solution. It is held that formaldehyd is a specific disinfectant of the virus (Cumming, in "Jour. Am. Med. Assoc.," May, 8, 1912). If not seen for a day or two, open the wound and scrub with a 5 per cent. solution of formaldehyd. Many physicians advocate excising the wounded area and cauterizing with pure nitric acid, a hot iron, or the Paquelin cautery. Fuming nitric acid is warmly commended by Paul Bartholow and others. Ravenel believes it may save a man even after twenty-four hours have elapsed since the bite. After the wound has been treated with formaldehyd or cauterized it is to be dressed antiseptically. If the patient is not seen for a day or two after the injury, cauterization is useless; it is not only useless, but it may delude the patient and his friends into a feeling of security. In any case, early or late, send the patient at once to a Pasteur institute. If the animal which inflicted the injury was not hydrophobic, no harm will result from inoculations; if it was hydrophobic, preventive treatment may save the patient. The method known as the preventive treatment was devised by Pasteur, who discovered the following remarkable facts: If the virus of a rabid dog (street rabies) be placed beneath the dura of another dog, it *always* causes hydrophobia in from sixteen to twenty days, and invariably causes death. If the virus is passed through a series of rabbits it gets stronger (laboratory virus), and if inserted beneath the dura of a dog it causes the disease in from five to six days, and kills in four or five days. The virus can be attenuated by passing it through a series of monkeys or by keeping it for a definite time. To obtain attenuated preparations in a convenient form Pasteur made emulsions from the spinal cords of hydrophobic rabbits, the animals having been dead two or three weeks. He found that the emulsion obtained from the rabbit longest dead is the weakest. He injected a dog with emulsions of progressively increasing strength and made it immune to hydrophobia. The patient is injected with an emulsion made from the dried spinal cords of hydrophobic rabbits. In this emulsion the virus is attenuated, and day by day the strength of the injected virus is increased. These emulsions cause the body-cells to form antitoxin, and either the virus of street rabies does not develop at all or by the time it begins to develop a quantity of antitoxin is present to antagonize it. In the New York Pasteur Institute patients remain under treatment for fifteen days, two inoculations being given daily. In cases in which treatment is begun late, or in which the head or face was bitten, from four to six inoculations are given each day. The report of the Parisian Pasteur Institute shows that since its foundation there has been a mortality of 0.5 per cent. The lowest estimated number of those attacked by hydrophobia before this method was used was 5

per cent. of those bitten, and all attacked died; hence, the Pasteur treatment as applied in the Parisian Institute shows one-twenty-fifth of the mortality which attends other preventive methods. In the Paris Pasteur Institute during 1910 401 patients were treated without a death. Ravenel, in 1901, estimated that 55,000 persons have been treated by the Pasteur method and that less than 1 per cent. have died. The value of this plan seems definitely established. The general public believes that the dog which did the biting should be killed. The dog should, if possible, be locked up and watched rather than killed. It may be proved in this way that it did not have hydrophobia. If it were necessary to kill the dog, or if the dog was killed at once or soon after, the physicians of the New York Pasteur Institute advise that the dog's head be cut from the body with an aseptic knife and a piece of the medulla oblongata be abstracted. The bit of medulla should be placed in a mixture of equal parts of glycerin and water which was previously sterilized by boiling. The bottle should be sealed and sent to the institute, in order that inoculations may be made upon animals to prove the existence or absence of hydrophobia. Babés tubercles and Negri's bodies are at once sought for in the specimen, and if they are found, treatment should be started at once. In the paroxysm of hydrophobia the treatment in the past was purely palliative. If we employ only palliative methods, keep the patient in a dark, quiet room, relieve thirst by enemata, saturate him with morphin, empty the bowels by enemata, attend to the bladder by regular catheterization, and during the paroxysms anesthetize. Murri, of Bologna, cured a case of hydrophobia by injecting emulsions of cords of rabbits dead six, five, four, and three days respectively. It would be proper to try this remedy if hydrophobia develops. A serum has been prepared by Tizzoni and Centani which they claim is successful in treating the disease as experimentally induced in the laboratory. The remarkable suggestion has come from Tizzoni that rabies be treated with rays of radium, it having been shown that rabic virus can be destroyed by radium.

**Glanders, Malleus, Farcy, or Equinia.**—Glanders is an infectious eruptive fever occurring in horses, mules, and asses, sometimes noted in goats, hogs, dogs, cats, and some other animals, and communicable to man. Cattle, house mice, house rats, white mice, and white rats are immune. The disease is most common in the horse and is due to the Bacillus mallei (see page 52). These bacilli are found chiefly in the nasal discharge and the recent nodules. There are few in older ulcerations. They are not found in the blood. Human glanders is by no means as uncommon as was once thought. Not a few cases die undiagnosticated. In a recent study 156 cases of chronic glanders were discussed (Robin, "Studies from the Royal Victoria Hospital of Montreal," 1906). If the nodules occur in the nares, the disease is called "glanders"; if beneath the skin, it is termed "farcy." The Bacillus mallei is communicated to man through an abraded surface or a mucous membrane. Bernstein and Carling reported 6 cases of human glanders. They say: "In none of our cases was there definite evidence of the point of inoculation or path of infection, and the general evidence, clinical and experimental, on both of these questions is so conflicting that the time is hardly ripe for the expression of decided opinions. The hypothetic paths are by direct inoculation through a wound or by the hair-follicles, by inhalation or by ingestion ("Brit. Med. Jour.," Feb. 6, 1909). The characteristic lesions are infective granulomata in the nares, skin, lungs, and subcutaneous tissue. In the nares granulomata result in ulcers and under the skin break down into abscesses. In some cases there is no nasal discharge. Multiple abscesses anywhere should excite suspicion of the existence of glanders. From the site of inoculation the bacilli are disseminated and the cutaneous and muscular structures and lungs become involved. There is usually a remittent fever. There is no known method of immunizing animals and mallein

has no immunizing power. One attack does not prevent another. Man may be infected from a diseased animal, and as the common source of infection is the horse, the usual victims are those who use or work about horses, and yet people who have never been about horses may develop the disease. The period of incubation after infection is four or five days.

**Acute and Chronic Glanders.**—In acute glanders there is septic inflammation at the point of inoculation; nodules may form in the nose and ulcerate; there is profuse nasal discharge; the glands of the neck enlarge; there is weakness, frontal headache, chilliness, pain in the back and limbs; often diarrhea; after a time the muscles become painful; there is fever, the evening temperature being 100° F. or higher and the morning temperature being lower. Chills may occur. There may be chest pains, severe muscular pain, bronchitis, and signs of pulmonary congestion. The lungs may be infected by inspiration of the bacilli in the discharge of the ulcers. It may not be suspected that the patient has glanders and the diagnosis of typhoid may perhaps be made. Twelve to fourteen days after the beginning of the trouble little hard lumps arise in the muscles and just beneath the skin. In a few days the lumps soften, break down, and discharge a bloody fluid which contains the bacilli of glanders. In a number of cases an eruption resembling small-pox appears on the face and about the joints, and this exanthem is usually prophetic of approaching death. It differs from small-pox in not being umbilicated. Leukocytosis may or may not exist. In chronic glanders there are like symptoms which last six weeks or more. Acute glanders is nearly always fatal. Chronic glanders lasts for months, is rarely diagnosticated, being mistaken for catarrh, may be recovered from, but if not soon eradicated will sooner or later eventuate in a fatal acute condition. The mortality is about 90 per cent.

**Acute and Chronic Farcy.**—Acute farcy arises at the site of a skin inoculation; it begins as an intense inflammation, from which emerge inflamed lymphatics that present nodules or "*farcy-buds*." Abscesses form, but the pus differs in appearance from ordinary pus, is often gelatinous, and may be red in color. There are joint-pains and the constitutional symptoms of sepsis, but no involvement of the nares. In it nodules occur upon the extremities, which nodules break down into abscesses and eventuate in ulcers resembling those of tuberculosis or, perhaps, of syphilis. The ulcers form rapidly and often heal.

**Diagnosis of Glanders and Farcy.**—Mallein is a concentration of the glycerin broth in which the bacilli of glanders were grown. Injection of mallein into an infected animal produces a significant reaction (fever, malaise, restlessness, perhaps a distinct rigor, sometimes vomiting, marked swelling at the point of inoculation). The dose used is 10 to 15 min. (Bernstein and Carling, in "Brit. Med. Jour.," Feb. 6, 1909).

Mallein has proved highly valuable in the hands of veterinarians. The reaction when it is injected into an animal with glanders is like the reaction produced by tuberculin on a tuberculous animal.

Animal injection is very valuable in diagnosis. The highly susceptible guinea-pig is used for this purpose. The doubtful material (nasal mucus or pus or a tissue fragment) is injected into the peritoneal sac of a male guinea-pig. Usually in three or four days the testicles enlarge and later suppurate.

The pig is killed and the pus examined for the bacillus of glanders. Testicular enlargement does not always occur even when the inoculated material was from a lesion of glanders. In some cases it does not occur for weeks. As a rule, it does occur.

In the lesions of glanders the bacilli of the disease are scanty and in many lesions are mingled with other bacteria, hence the bacteriologic diagnosis is always difficult and is often impossible without animal inoculation. The

value of an agglutination test is as yet undetermined, but it is regarded with confidence by some.

*Treatment.*—In treating this disease the point of infection is at once to be incised and cauterized, dusted with iodoform, and dressed antiseptically. The skin over enlarged glands and swollen lymphatics is to be painted with iodin and smeared with ichthyol. Bandages are applied to edematous extremities. Ulcers are curetted, touched with pure carbolic acid, dusted with iodoform, and dressed antiseptically. In glanders the nostrils should be sprayed at frequent intervals with peroxid of hydrogen and syringed with a solution of sulphurous acid. The mouth must be rinsed repeatedly with solutions of chlorate of potassium. Abscesses are to be opened, mopped with pure carbolic acid, and dressed antiseptically. Stimulants and nourishing diet are imperatively demanded. Morphin is necessary for the muscular pain, restlessness, and insomnia. Digitalis is given to stimulate the circulation and kidney secretion. Sulphur iodid and arsenite of strychnin have been used. Diseased horses ought at once to be killed and their stalls should be torn to pieces, purified, and entirely rebuilt. A man with chronic glanders should be removed to the seaside. The nasal passages must be kept clean and the ulcers must be cauterized and dressed with iodoform gauze. Nutritious foods, tonics, and stimulants are necessary.

Treatment of recent cases by frequent small doses of mallein finds advocates. It has been suggested that injection of the serum of the blood of an immune animal (the ox, for instance) might be serviceable. Most observers assert that serum of naturally immune animals and the serum of animals which have been repeatedly injected with mallein are equally worthless.

**Actinomycosis** (*streptotrichosis*) is a specific infectious disorder characterized by chronic infiltrating, granulomatous inflammation, and is due to the presence in the tissues of some variety of streptothrix. It was long believed that the *actinomyces bovis* or ray-fungus was the only cause of the disease. We now believe that other members of the streptothrix group may be responsible. As stated on page 19 the streptothrices are usually regarded as molds, but they possibly constitute a transition stage between filamentous fungi and bacteria. At present many pathologists use the term "actinomyces" as the generic name for the various forms of these parasites. Other pathologists designate them as streptothrix forms, of which the actinomyces bovis is one. In 1877 Bollinger recognized and described these parasites as causative of disease in cattle. In 1878 Israel described human actinomycosis. Acland, in 1884, reported the first English case of human actinomycosis. Some of the varieties of the organism are pathogenic, others do not seem to be. Some forms are anaërobic, others are aërobic. When dried they are not killed at once, but months after may develop if placed under favorable conditions. When growing in the tissues they usually form numerous distinct yellow, reddish, black, or gray aggregations, each about the size of a sand grain, and called from their color *sulphur grains* or *red pepper grains*. They grow from mycelia. Usually the growths lie in thick and sticky purulent matter. If purulent matter containing growths is rubbed between the fingers, it will give a gritty sensation like sand if the growth is not very recent. The grit is due to lime-salts. The growth of the fungi causes the formation of an infective granuloma, and great masses of granulation tissue may form with collections of necrotic or purulent matter here and there, and zones of fibrous tissue. The fungi are easily discovered in the sulphur grains by the microscope, but if the fungi are mycelial and are scattered instead of being gathered into granules it is difficult to discover them. This disease occurs in cattle (*lumpy jaw*) and in pigs, and can be transmitted to man. It is believed by many that the fungi exist normally inside the husks of barley and other grasses and that animals are inoculated by eating

the contaminated grasses, scratches on the mucous membrane of the tongue, mouth, or pharynx being inoculated. In man the fungus may be taken in with food, by chewing the mold-bearing grass or straw, or by inhalation of contaminated dust. Its development seems dependent upon processes of decay. It may lodge and develop in a breach of mucous continuity, a crypt of the tonsil, or a hollow tooth.

Lord, of Boston, has found the parasite in carious teeth of those free from actinomycosis, and has produced actinomycosis in guinea-pigs by inoculating them with the contents of carious teeth ("Publications of the Massachusetts General Hospital," Oct., 1911). Lord reaches the conclusion that true actinomycosis (the disease due to ray-fungi) arises from within the individual, carious teeth being the cause.

The disease is at first local; later there may be general blood infection and abscesses may form in the lung, liver, kidney, etc.

The fungi may pass into the lungs, causing pulmonary actinomycosis; into the intestines, causing intestinal actinomycosis; into the skin, the bones, the subcutaneous tissues, the heart, the brain, the liver, the urinary organs, etc. Abdominal anthrax is the commonest form and comprises nearly 50 per cent. of cases. Cases of human actinomycosis until recently were looked upon as sarcomata. Many sinuses form, ulcers develop, but large abscesses do not arise.

Cutaneous actinomycosis may be secondary to visceral infection with the disease, may be a purely local condition due to inoculation of a wound, or may be associated with some adjacent area of bone infection. The gummatous form of actinomycosis resembles a gummatous syphilitic area, and in it many small purulent pockets open by fistulæ.

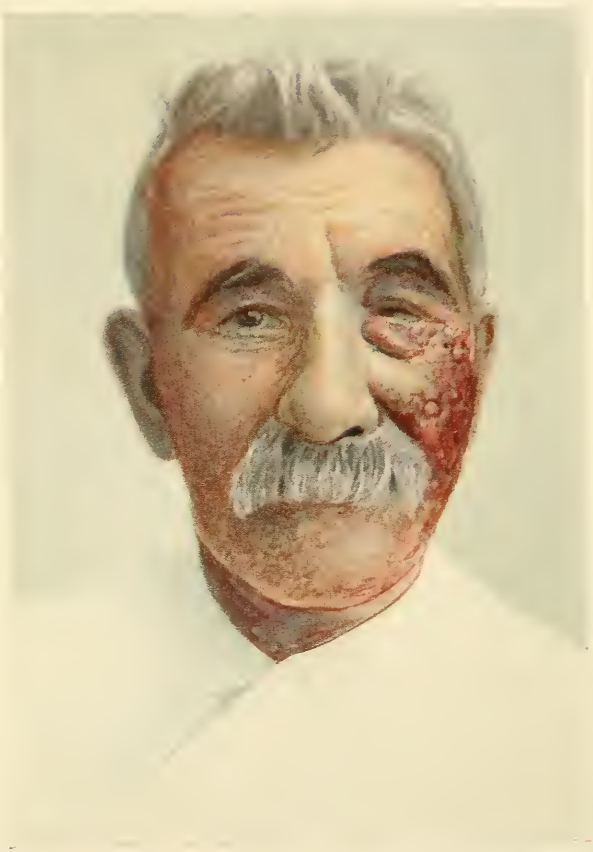
In actinomycosis there is no spread by lymphatics until very late in the case. In fact, the adjacent lymph-glands are very seldom involved unless there is secondary pyogenic infection, and if metastasis occurs it takes place by the veins. The condition causes but slight pain. A diagnosis must be made from syphilis, sarcoma, carcinoma, and tuberculosis. The formation of a tumor, followed by sinuses and ulceration, the ulcer having thin, non-indurated, undermined edges and edematous granulations, and adjacent pus cavities joining by sinuses, the appearance of the pus and the microscopic study of the discharge are significant. An actinomycotic ulcer may partially heal here and there. It has been stated that an individual with actinomycosis may react to tuberculin like a person with tuberculosis (see page 227). The muscular and connective tissues become infiltrated and hard, as though a coagulating material had been injected into them (Poncet and Bérard, in "Lyon Médicale," March 27, 1904). Edema and induration extend wide of the active focus of disease (Plate 2). Actinomycosis may last for years or it may prove fatal.

In the anthracoid form there are no distinct purulent collections, but many fistulæ discharge pus at various points (Monestié).

An area of cutaneous actinomycosis is characterized by the existence of violet, blue, gray, or black maculæ, varying in size from that of a pin's head to that of a bean, the center of each macule being white and containing a minute quantity of pus (Derville).

In actinomycosis of bone the bone enlarges and becomes painful, the parts adjacent swell from infiltration and soften, pus forms and reaches the surface through fistulæ, and the skin becomes involved secondarily.

Abdominal actinomycosis takes origin from the gastro-intestinal tract, an actinomycotic nodule of the intestine having ulcerated, adhesions having formed, and an actinomycotic abscess having arisen, or actinomycotic disease of the intestine having spread. In over 50 per cent. of cases of abdominal actinomycosis the cecum is the part attacked. A fecal fistula may form and the liver may be involved. At least 150 cases of actinomycosis of the appendix



Author's case of facial actinomycosis.

have been reported. A mass containing putrid pus develops. If not evacuated fistulæ form. It is a very chronic condition, and although fistulæ may heal, they break open again and again (Short, in "Lancet," Sept. 14, 1907). The fungi may be found in the stools.

The mortality of actinomycosis depends upon the site of infection, the question of secondary infection, and the plan of treatment. If pyogenic infection occurs, fatal pyemia may arise. The prognosis is reasonably good in many cases. The majority of cutaneous cases (nearly 90 per cent.) and many osseous cases can be cured. The mortality in the abdominal cases is large. Grill says that of 77 abdominal cases treated surgically 45 died, 22 recovered, and 10 were improved. Frazier ("Keen's System of Surgery") tells us that the mortality of the reported cases of actinomycosis in the United States was 47 per cent., and quotes Jiron as follows regarding the mortality of the various forms: Face and neck, 11 per cent.; thorax, 83 per cent.; abdomen, 71 per cent.; brain, 100 per cent. Actinomycosis has a strong tendency to redevelop even after apparently thorough excision. A case of cutaneous actinomycosis of the arm, seen by the author, was operated on twenty times. Ulceration took place into the axillary artery and death was narrowly averted. Recovery finally ensued. I have seen 4 cases of human actinomycosis: one was the patient just referred to; another was a mattress stuffer (straw being used), his lesions were on the chest and jaw and recovery followed operation; the third was a stable hand, who died from lesions of the face, jaw, and neck; the fourth was a railroad watchman who had nothing to do with horses (Plate 2).

**Treatment.**—Free excision if possible; otherwise incision, scraping, cauterization with pure carbolic acid or silver nitrate, and packing with iodoform gauze. If possible, remove the entire area; if not possible, remove all that can be safely taken away. Sinuses must be widely opened, each collection of pus must be drained, and granulation tissue if not extirpated must be scraped away with a sharp spoon. Give internally large doses of iodid of potassium. This drug alone has cured many cases. It is given for a week or two and is then discontinued for one week. It is curious that though iodid of potash seems to influence actinomycosis favorably, the fungus will live unharmed in a 2 per cent. solution of that drug (Harbitz and Grandahl, in "Amer. Jour. Med. Sci.," Sept., 1911). In a fistula from intestinal or appendical actinomycosis give potassium iodid and sulphate of copper (Bevan) and irrigate the sinuses with diluted tincture of iodin. Cases of actinomycosis should be placed under the best hygienic conditions, should live as much as possible in the sunlight and open air, and should be given nutritious diet, tonics, and often stimulants.

**Blastomycosis.**—May, in 1894, called attention to the disease. In 1894 Busse described a fatal infection due to a pathogenic yeast. The very existence of this disease has been denied, but numerous cases have been reported and most observers regard it as an entity. Many cases have been reported from Chicago and its neighborhood by Bevan, Hektoen, Hyde, and others. It may be a local infection (cutaneous blastomycosis), but in some cases the disease is generalized (systemic blastomycosis). The disease is found in North America, South America, Europe, and the East. In generalized blastomycosis the lungs are usually primarily infected. Generalized blastomycosis is usually fatal. The yeasts when found in the tissues are in the budding stage.

Cutaneous blastomycosis (blastomycetic dermatitis or oïdiomycosis) was first described by Gilchrist in 1894. It is due to infection with a variety of yeast fungus. In the skin it begins as a papule or an indurated pustule, which becomes crusted and finally warty, enlarges at the periphery, and becomes surrounded by more recently developed lesions. The area becomes studded with minute abscesses, crusted foci, and areas of bleeding granulations. Here and there healing may occur.

The disease may arise on any portion of the body, but the hands and face are most liable to it. It is slow in progress and lasts for many months. If it progresses, it will finally produce systemic blastomycosis or secondary infection will produce fatal septicemia. Bevan (Lexer's "General Surgery," edited by Arthur Dean Bevan) says the evidences of general infection are: "irregular temperature, loss of appetite, general weakness, emaciation, cough, rapid, feeble pulse, acceleration of the respiration, at times albumin in the urine, multiple subcutaneous nodules and abscesses resulting in superficial, irregular ulcers, abnormal physical findings in the lungs, edema of the extremities, and various grades of anemia." Coccidial disease is caused by an organism strongly resembling the yeast of blastomycosis. Bevan places the two diseases in the same group. Some observers regard them as identical. The symptoms are indistinguishable. Montgomery regards iodid of potassium as a test agent—it is without effect on coccidial disease, but acts strongly and favorably upon blastomycosis.

**Treatment.**—In some cases excision, in others the *x*-rays. Potassium iodid in very large doses is given. It greatly benefits most cases and cures many. Its employment in this disease is due to Bevan. He also gives copper sulphate internally ($\frac{1}{4}$ gr. three times a day), and applies a 1 per cent. solution of that chemical to local lesions.

## XVI. BURNS AND SCALDS; EFFECTS OF COLD

Burns and scalds are injuries due to the action of caloric. Scalds are due to heated fluids or vapors. There is no true pathological difference between burns and scalds. Dupuytren classified burns into six degrees, as follows: (1) Characterized by erythema; (2) characterized by dermatitis with the formation of vesicles; (3) characterized by partial destruction of the skin, which structure is not, however, entirely burned through; (4) characterized by destruction of the skin to the subcutaneous tissue; (5) characterized by destruction of all superficial structures and of part of the muscular layer; (6) characterized by "carbonization" of the whole thickness of the muscles.

The **symptoms** of a severe burn are local and constitutional. *Local symptoms* are pain and inflammation, which vary in nature, in intensity, or in degree according to the extent of tissue damage. *Constitutional symptoms* are very weak pulse, shallow respiration, and subnormal temperature—in other words, the condition of shock exists. The patient may die without reacting from shock, but in most cases there is reaction, followed by a severe reactionary fever, with a strong tendency to congestion of internal parts. During the existence of fever there may be vomiting, diarrhea, hemoglobinuria, albuminuria and enlargement of the liver, spleen, lymph-glands, and tonsils. Marked blood changes follow burns (see "Clinical Hematology," by J. C. DaCosta, Jr.). There is a marked and rapid increase in red blood-cells (polycythemia). This is due in part to venous stasis and in part to loss of blood-plasma. Leukocytosis is rapid and pronounced and there is a notable increase in blood-plaques.

The blood has a marked disposition to clot, and clots may damage various structures or organs. Further, the altered blood damages the organs of excretion, and the liver and kidneys may cease to perform their functions properly. After a severe burn there are imperfect oxygenation and a tendency to universal fatty degeneration. The symptomatic stages are often designated as *prostration*, *reaction*, and *suppuration*. During the first forty-eight hours after a burn there are congestion in and about the burned area,

severe pain, and possibly internal congestions. There may be shock and possibly toxic delirium or convulsions. From the end of the second to the end of the eighth or ninth day there are severe inflammation of the burned area, formation of sloughs, and a strong tendency to inflammation of the brain in head burns, of the lungs in chest burns, of the abdominal organs in abdominal burns. Duodenal inflammation may arise after any burn. Septic emboli in very unusual cases cause *Curling's ulcer* of the duodenum (see page 162). Duodenitis and Curling's ulcer are possibly due, as Wm. Hunter suggested, to the bile having become irritant by the excretion in it of toxic matter. After the eighth or ninth day the sloughs separate from the burned area and healing begins. The raw surface is slow to heal, hemorrhages may occur, the granulations are apt to be exuberant and edematous, and the scars are very contractile and often produce hideous or disabling deformity. If over one-half of the body surface is badly burned, death will almost certainly occur, and probably within two days. The danger of a burn depends upon its extent, its depth, and its situation. Burning of a large area superficially is much more dangerous than burning a small area deeply. Burns of the extremities are not so dangerous as are burns of the head, chest, or abdomen. Death after severe burns is positively not due to loss of body-heat in the burned area. Some think it is produced by auto-intoxication with retained body secretions. High temperature produces blood changes—viz., disintegration of red corpuscles. Thrombosis may occur, and irritation of the kidneys and other organs is produced by "products of corpuscular degeneration."[1]

The blood of burned animals contains toxins (Kijanitzen), and so does the urine (Reis). It seems probable that the constitutional symptoms and death, if it occurs, are due partly to corpuscular disorganization and partly to the absorption of toxic matter from the seat of injury, this matter having been formed by the action of heat on the body-cells and fluids. Sepsis is not infrequent. Death may be directly due to shock, to sepsis, to exhaustion, to tetanus, to embolism or thrombosis, to congestion of the brain, lungs or kidneys, or to Curling's ulcer of the duodenum.

**Treatment.**—The *local treatment* of slight burns is to moisten the parts frequently with a saturated solution of bicarbonate of sodium or with normal salt solution. In burns of moderate degree a mixture of zinc ointment with iodoform, though not antiseptic, is a comfortable dressing.

If a large surface is burned, remove the clothing with great care, and before applying dressings give a hypodermatic injection of morphin, administer stimulants, and if the patient has a chill, place him in a warm bath. Use all ordinary means to secure reaction from shock. Waterhouse makes a recommendation which I have frequently used with advantage, viz., dress the burn with a 1 per cent. solution of acetate of aluminum, wrap the patient in blankets, hold up the other bedclothes with a cradle, and put under the bedclothes an electric light of 32 candlepower. This will make the temperature under the clothes from 100° to 105° F. ("Brit. Med. Jour.," July 9, 1910). If we desire to dress a large burn aseptically, anesthetize the patient, spray the burnt area with peroxid of hydrogen, irrigate it with a solution of boric acid, dry with sterile cotton, dust with *Senn's powder* (3 parts of boric acid and 1 part of salicylic acid), and dress with salicylated cotton. Senn's powder is better than iodoform. Iodoform may allay pain, but is apt to produce dermatitis. Change the dressing no oftener than is required, and at each change proceed as above described, although it will not be necessary to anesthetize. Peroxid of hydrogen softens and loosens the dressings, and they can be readily removed. The custom in the Jefferson Medical College Hospital is to give morphin and stimulants, to cut away the clothing, to wrap the unburned parts

[1] Bardeen, in "Johns Hopkins Hospital Bulletin," April, 1897.

with blankets, and place about them cans or bags of hot water. The burned region is sprayed with diluted peroxid of hydrogen contained in an atomizer, and is irrigated with salt solution. Portions of epidermis which remain are retained. Any blisters are opened with a sterile needle, and the part is dressed with several layers of sterile lint or tarlatan soaked in normal salt solution, and the dressing is kept moist. During the second or inflammatory stage use stimulants and concentrated food, allay pain by opium or morphin, favor elimination by the skin, bowels and kidneys, and combat any tendency to internal congestion or inflammation. In very extensive burns complete and continuous immersion of the part in warm salt solution is an excellent treatment.

The *picric acid treatment*, first suggested by Thiery, has many advocates. It greatly mitigates the pain. It is used early only in limited burns of the first and second degrees, but it can be employed in late stages of deep burns to stimulate the formation of epidermis. If used early in a large or a deep burn, it may poison the patient. It may poison a child when used upon a burn of the second degree. A case was reported by Dr. J. Stuart Rose ("Scottish Med. and Surg. Jour.," Dec., 1903), occurring in a boy of nine, who was treated with picric acid for a scald of the first degree, there being only one or two small blisters in addition to the redness. Ointment of picric acid was used (½ dr. to 1 oz. of vaselin). Symptoms were noted three days after beginning the treatment. The symptoms of poisoning are dark-colored urine (carboluria), albuminuria, marked yellowness of the skin, yellowness perhaps of hair at the scalp margins, diarrhea, and elevated temperature. Rose considers a 1 per cent. solution safe. It is applied as follows: The part should be disinfected, gauze saturated with a 1 per cent. watery solution of picric acid should be laid upon the burned area, and be covered with absorbent cotton and a bandage. This dressing is not changed for three to five days, and the next dressing can be left in place until the burn is healed. D'Arcy Power has carefully studied the real status of picric acid as a remedy for burns, and some of his conclusions have been set forth above.

Périer dresses a burn with a tarlatan compress, folded six times and soaked in the following solution: boric acid, 2½ dr.; antipyrin, 1½ dr.; sterile water, 8 oz. The following ointment is used by Reclus: iodoform, 15 gr.; antipyrin, 75 gr.; boric acid, 75 gr.; vaselin, 1½ oz.

Carron oil consists of equal parts of linseed oil and lime-water. It allays the pain of a burn, but it is a filthy preparation, and its use is followed by much pus formation. Cosmolin gives comfort as a dressing, but should not be used on the face, lest it cause pigmentation. The elder Gross used lead paint. A solution of nitrate of potassium allays the pain. Bismuth paste is a very satisfactory dressing. In every burn of the fingers and toes keep the burnt digits separated by gauze, lint, or rubber tissue during healing, otherwise adjacent fingers will adhere and "*webbing*" will result. When extensive destruction of tissue has taken place and healing has begun, use splints and extension to limit contractures, and skin-graft as soon as possible. If granulation is slow, stimulate with copper sulphate or mild silver nitrate solutions. Exuberant granulations require burning down. Flabby granulations require pressure. If healing is slow, or if the burn is extensive, skin-graft. Skin-grafting should be done early in an extensive burn. If performed before much cicatricial tissue has formed, the graft will be more apt to adhere, and if the graft does adhere, further formation of scar tissue will be greatly limited. When an extremity has been carbonized, amputation must be performed. The *constitutional treatment* of a severe burn is to bring about reaction; combat pain with opium, and keep the bowels and kidneys active. If suppuration

occurs, give tonics, stimulants, and concentrated foods. Complications are treated according to general rules.

**Burns and Scalds of the Tongue, Pharynx, Glottis, and Epiglottis.**—A child or lunatic may drink boiling fluid or inhale steam from a tea-kettle. Firemen occasionally suffer from scalds of the tongue and pharynx after being suddenly enveloped in a cloud of hot steam, and from burns by the inhalation of hot vapor or flame. Caustic may be taken into the mouth or swallowed. The tongue and pharyngeal mucous membrane swell greatly, large vesicles form, there are shock, severe pain, dysphagia, and dyspnea. Edema of the glottis may arise.

**Treatment.**—Combat shock; give morphin for pain; puncture vesicles, and have the patient almost constantly suck bits of ice. If great swelling occurs, make multiple longitudinal incisions through the mucous membrane of the dorsum of the tongue. If edema of the glottis begins, scarify it. If this fails, perform intubation or tracheotomy.

**Burns of the Esophagus.**—The esophagus is seldom scalded, as a boiling fluid rarely gets below the pharynx. The swallowing of an acid or alkali produces severe burns at the constricted portions of the gullet (see page 933). Such an accident produces shock, dyspnea, violent pain, vomiting of blood, and thirst. Death may occur from shock or perforation of the stomach. In many cases severe gastritis follows a burn of the esophagus. As the acute symptoms of a burn of the gullet gradually abate, sloughs are cast off, ulcers form, cicatrization begins, and the signs of stricture develop (see page 933).

**Treatment.**—Give a remedy to neutralize the caustic. Administer several large drafts of water and wash out the stomach. Combat shock. Give morphin for pain. Feed by the rectum as long as the patient's strength does not begin to fail. On beginning mouth-feeding, use at first milk and then beef-juice, jelly, and ice-cream. In from two to four weeks after the infliction of the burn begin the use of bougies to limit contraction.

**Effects of Cold.**—*Local Effects.*—Cold produces numbness, pricking, a feeling of weight, redness of the surface followed by stiffness, local insensibility, and mottling or pallor. Sudden intense cold causes the formation of blebs, the coagulation of blood in the superficial veins, and violent pain in the part. Cold locally produces frost-bite (see page 177).

The *constitutional effects* of cold are at first stimulating, then depressing, and are exhibited by uneasiness, pain, and an intense drowsiness which, if yielded to, is the road to death by way of internal congestion. Death from prolonged cold resembles in appearance death from apoplexy. Death from sudden and overwhelming cold is caused by anemia of the brain from weak circulation and capillary embolism. To bring a partly frozen person into a warm room may cause death by embolism.

**Treatment.**—Frost-bite is treated as outlined on page 177. When a person is nearly frozen to death place him in a *cool* room, but under no circumstance in a cold bath; make artificial respiration, rub him briskly with flannel soaked in alcohol or in whisky, and follow this by rubbing by dry hands. After a time wrap the patient in warm blankets and give an enema of brandy. Mustard-plasters are to be applied over the heart and spine. As soon as swallowing is possible, brandy is administered by the mouth. As the condition improves gradually raise the temperature of the room and give *hot* drinks.

**Chilblain** or **pernio** is a secondary effect of cold. It is really an area of local asphyxia (see page 173). It usually appears as a local congestion upon the toes, the ears, the fingers, or the nose, and now and then inflames and ulcerates. A chilblain is apt to become congested on the victim approaching a fire

or on taking exercise, and when congested it itches, tingles, and stings. Frequent attacks of congestion produce crops of vesicles; these vesicles rupture and expose ulcers, which in rare instances slough.

**Treatment.**—If chilblain affects the toes, prevent congestion of the legs and feet. Order large shoes and woolen stockings and forbid tight garters. The patient with pernio must take regular outdoor exercise and must not loiter around a hot fire. Every morning and evening he should take a general cold sponge-bath, followed by rubbing with alcohol and friction by a coarse towel, and in winter he should sleep wearing warm stockings or with his feet upon a warm-water bag. When a chilblain is only a congested spot, it should be washed twice a day in cold salt water, rubbed dry with flannel, and subjected to applications of tincture of iodin and soap liniment (1 : 2), tincture of cantharides and soap liniment (1 : 6), or equal parts of turpentine and olive oil (W. H. A. Jacobson). Jacobson says itching is relieved by painting belladonna liniment upon the part and allowing it to dry. Tincture of iodin may relieve it, and so may a mustard foot-bath. A valuable preparation for itching is composed of 1 dr. of powdered camphor and 4 oz. of cosmolin. A little of this ointment is rubbed in twice a day. The following prescription, the source of which I do not remember, is very valuable for itching: 1 dr. of powdered camphor, 1½ dr. of ichthyol, ½ dr. of lanolin, and 4 oz. of cosmolin, rubbed into the part and covered with cotton-wool. If vesicles form, paint with contractile collodion; if ulcers form, dress antiseptically. If ulcers are sluggish, use equal parts of resin cerate and spirits of turpentine. A good antiseptic and protective is the following: oxid of zinc, 6 gr.; chlorid of zinc, 20 gr.; gelatin, 2 oz.; distilled water, 1 oz.

## XVII. SYPHILIS, OR POX

The name comes from the title of a poem which was published in Italy in 1530, "Syphilis sive Morbus Gallicus," by Hieronymus Fracastorius. For centuries there has been fierce dispute as to the origin of syphilis. Many assert that Europe was free of it until the discovery of America and that the sailors of Columbus brought it from Hayti. Others claim that the disease has always existed in Europe and the East. We know there was a violent outbreak of it in 1494 among the French soldiers besieging Naples.

**Definition.**—Syphilis is a chronic contagious, and sometimes hereditary, constitutional disease. It is one of the most common of diseases and were facts known would rank high as a cause of death. Lesser states that in Berlin, of men over twenty-five years of age who come to autopsy, 9 per cent. are syphilitic. In the United States it is twice as frequent among negroes as compared with whites, although in them the disease is apt to be more curable and less disastrous. It was long believed that only members of the human family could take syphilis, but Metchnikoff and Roux have succeeded in inoculating chimpanzees ("Annals of Pasteur Institute," Dec., 1903). These two observers have inoculated many animals and have shown that the nearer the animal is to man, the more nearly the disease resembles human syphilis. Anthropoid apes can be successfully inoculated, but only in the chimpanzee are human symptoms accurately produced. At first the causitive protozoöns are localized. Metchnikoff in 1906 inoculated a student and a monkey with syphilitic virus. An hour after inoculating the man the seat of inoculation was rubbed for five minutes with mercurial ointment and the man escaped syphilis. The ointment was not used on the monkey and a chancre developed. Its first lesion is an infecting area or chancre, which is followed by lymphatic enlargements, eruptions upon the skin and mucous membranes, affections of the appendages of the skin

(hair and nails), "chronic inflammation and infiltration of the cellulovascular tissue, bones, and periosteum" (White), and, later, often by gummata. The disease is due to a protozoön, the Spirochæta pallida. This fact was demonstrated by Schaudinn in 1905 (see page 55). The Spirochæta pallida is demonstrated in recent scrapings from specific lesions. The more contagious the lesion, the more of the organisms are found. The later the lesion, the fewer the organisms found. Spirochetes are always discoverable in the chancre. They can usually be found in the secondary lesions and sometimes in the blood during the secondary stage. Spirochetes are present in tertiary lesions, although in less number than in secondary lesions. The presence of a great number of spirochetes means a malignant case. They tend to disappear under mercurial treatment (see pp. 336 and 340). Syphilitic fever is due to absorption of toxins. Skin eruptions and eruptions on the mucous membranes in the secondary stage arise from local deposit and multiplication of the spirochetes. The spirochetes continue to exist in the body after the cessation of secondary symptoms, and may die out or may awaken into activity, producing *reminders*.

Up to about twelve days after the appearance of a chancre a person can still be infected by a fresh exposure to inoculation. After twelve days he cannot be unless inoculated with a very large amount of virus (Pinard). In other words, after twelve days there is immunity to fresh inoculation, but the immunity is not powerful enough to kill the spirochetes already in the body. During the primary stage, from the twelfth day after the appearance of the chancre, and during the secondary stage fresh poison cannot infect, and this is true for a long time after the disappearance of secondary symptoms. As a matter of fact, the immunity generally lasts for life and reinfection is one of the rarest of occurrences. Most supposed cases of reinfection were really instances of a fresh outbreak of an old disease, but a few undoubted cases of reinfection have been reported. Immunity in the primary stage is due to products absorbed from the infected area. Colles's immunity is that acquired by mothers who have borne syphilitic children, but who themselves show no sign of the disease. Profeta's immunity is the immunity against infection possessed by children born of syphilitic parents. Some of these children have never shown signs of disease, but nevertheless they are immune. It is claimed that a person long free from active syphilis, but still immune, can transmit immunity to his children. Tertiary syphilis is not nearly as readily transmissible as secondary syphilis, but it secures immunity.

**Transmission of Syphilis.**—This disease can be transmitted: (1) by contact with the virus—*acquired* syphilis, and (2) by hereditary transmission—*hereditary* syphilis. The poison cannot enter through an intact epidermis or epithelial layer, and abrasion or solution of continuity is requisite for infection. Syphilis is usually, but not always, a venereal disease. It may be caught by infection of the genitals during coition, by infection of the tongue or lips in kissing, by smoking poisoned pipes, by drinking out of infected vessels, or by beastly practices. Syphilis not due to sexual relations is called *syphilis of the innocent.* The barber is a danger, and cases are reported as following razor cuts and particularly the application of the alum stick to arrest bleeding. This stick is used over and over again and dried blood is often to be found upon it. I was consulted by a man who had been thus infected. I have treated two young girls infected by dentists' instruments, a policeman infected by a pipe, a glassblower infected from the blowpipe, a street car driver who got the disease from a borrowed whistle, a police officer who got it striking a prisoner on the mouth and cutting his own knuckle on the teeth, a hospital orderly who infected his nose picking it by a contaminated finger, and three physicians who caught it from

patients. Schamberg makes a valuable contribution to the accumulation of facts which demonstrates the danger of promiscuous osculations ("Jour. Am. Med. Assoc.," Sept. 2, 1911). A party of young men and women indulged in a kissing game. One of the young men had a sore lip. As a result of the party 6 men and 1 woman developed labial chancres. A girl kissed at another party by the contaminator of the first party got a chancre. The solitary male victim of the first party evidently got the disease by kissing a girl recently kissed by the syphilitic man. Bulkley (Ibid., March 4, 1905) collected 1863 cases following vaccination; 179 following circumcision; 82 following tattooing, and 745 following cupping or venesection. The initial lesion of syphilis may be found on the finger, penis, eyelid, lip, tongue, cheek, palate, tonsil, labium, vagina, anus, nipple, etc. A person may be a host for syphilis, carry it, give it to another, and yet escape it himself (a surgeon may carry it under his nails and a woman may have it lodged in her vagina). Syphilis can be transmitted by vaccination with human lymph which contains the pus of a syphilitic eruption or the blood of a syphilitic person. Vaccine lymph, even after passage through a person with pox, will not convey syphilis if it is free from blood and the pus of specific lesions; it is not the lymph that poisons, but some other substance which the lymph may carry. When syphilis is caught from one of a different race the disease is apt to run a peculiarly severe course. The apprehensions of the sailor regarding "Chinese pox" are probably well founded. John Knott says ("New York Med. Jour.," Oct. 31, 1908): "The Swan Alley sore of the days of Benjamin Travers was immeasurably more severe and more rapidly destructive in its progress than any average London chancre; for it was the fruit of the continuous patronizing culture of the foreign sailors and refugees who were derived from the veriest social and moral dregs of the population of all foreign countries."

**Effect of Syphilis Upon Longevity.**—This is a difficult matter to determine. Many deaths result from diseases caused by syphilis and yet they are not certified as due to syphilis. My own belief is that the mortality directly due to syphilis is very small indeed (except in cases of congenital syphilis), but that the mortality indirectly due to it is very large. I think that the life insurance companies are justified in requiring those who have had syphilis to pay higher premiums than those who have never had the disease.

**Syphilis and Tuberculosis.**—Syphilis and tuberculosis may coexist. The syphilis may be severe and the tuberculosis mild, or vice versâ. In some cases neither is severe and in some cases both are. As a rule, they have an unfavorable influence on each other. Tuberculosis is not very unusual in the course of secondary syphilis. It is apt to run a rapid course because of the patient's debility and worry and because of the treatment employed for the syphilis. Old syphilitics may develop tuberculosis, but do not seem particularly prone to do so. A child born syphilitic is very apt to become tuberculous, more apt even than is a child born of tuberculous parents. If a patient who is thought to be well of syphilis becomes tuberculous the syphilis may crop out again. If a tuberculous patient becomes syphilitic the tuberculosis usually gets worse. It is well known that syphilitic ulcers may become tuberculous. This sometimes occurs in the larynx. (See chapter on Syphilis in Bonney's treatise on "Pulmonary Tuberculosis").

**Syphilitic Stages.**—Syphilis was divided by Ricord into three stages: (1) The *primary* stage—chancre and indolent bubo; (2) the *secondary* stage—disease of the upper layer of the skin and mucous membranes; and (3) the *tertiary* stage—affections of connective tissues, bones, fibrous and serous membranes, and parenchymatous organs. This division, which is useful clinically, is still largely employed, but it is not so sharp and distinct as was believed by Ricord; it is only artificial. For instance, ozena may

develop during a secondary eruption, and bone disease may appear early in the case.

**Syphilitic Periods.**—White divides the pox into the following periods: (1) Period of *primary incubation*—the time between exposure and the appearance of the chancre, from ten to ninety days, the average being twenty-five days; (2) period of *primary symptoms*—chancre and bubo of adjacent lymph-glands; (3) period of *secondary incubation*—the time between the appearance of the chancre and the advent of secondary symptoms, about six weeks as a rule; (4) period of *secondary symptoms*—lasting from one to three years; (5) *intermediate* period—there may be no symptoms or there may be light symptoms which are less symmetrical and more general than those of the secondary period: it lasts from two to four years, and ends in recovery or tertiary syphilis; (6) period of *tertiary symptoms*—indefinite in duration. The fifth and sixth periods may never occur, the disease having been cured.

**Primary Syphilis.**—The primary stage comprises the chancre or infecting sore and bubo. A chancre or initial lesion is an infective granuloma resulting from the poison of syphilis and is most usually met with upon the genital organs. A chancre may be derived from the discharges of another chancre, from the secretion of mucous patches and moist papules, from syphilitic blood, or from the pus or secretion of any secondary lesion. Tertiary lesions seldom cause chancre. It appears at the point of inoculation (see page 316), and is the first lesion of the disease. During the three weeks or more requisite to develop a chancre the poison is continuously entering the system, and when the chancre develops the system already contains a large amount of poison. A chancre is not a local lesion from which syphilis springs, but is a local manifestation of an existing constitutional disease, hence excision is entirely useless. If twelve days or more after the appearance of a chancre we take some of the discharge and insert it at some indifferent point, into the person from whom we took it, a new indurated chancre will not be formed. This means that the subject has become immune to inoculation, but the immunity is not sufficiently powerful a factor to kill the spirochetes already in the body. When inoculation fails it means that the individual already has constitutional syphilis. If a syphilitic is inoculated with the discharge of a chancre twelve days or more after the chancre began, no indurated sore develops, but if the chancre furnishing the virus was *irritated*, a *non-indurated* sore may arise at the point of inoculation. If we take the discharge of a chancre and insert it into a healthy person, an indurated chancre follows. Hence we say that primary syphilis is not auto-inoculable, but is hetero-inoculable. A soft sore can be produced in the lower animals by inoculation with the virus of a chancre, but a hard sore cannot except in monkeys. Some observers, notably Kaposi, of Vienna, advocate the *unity theory*. This theory maintains that both hard and soft sores are due to the same virus, the infective power of the soft chancre simply being less than that of the hard sore, the possibillty of constitutional infection depending, not upon differences in the poison, but rather upon differences in the soil and in the local processes. The unicists advocate excision of chancres, soft or hard, to prevent, if possible, constitutional involvement. Most syphilographers believe in the *duality theory*, which we have previously set forth. This theory took origin from the classical investigations of Bassereau and Rollet. The duality theory maintains that the soft sore is caused by a poison different from the one which originates the hard sore, and that a true soft sore never infects the system. The discovery of the micro-organism causing syphilis proves the dualists to be correct.

**Initial Lesions.**—An initial lesion, hard chancre, or infecting sore never appears until at least ten days after exposure; it may not appear for many weeks, but it usually arises in about twenty-five days. There are three

chief forms of initial lesion: (1) A purple patch exposed by peeling epidermis, without induration and ulceration—a rare form; (2) an indurated area under the epidermis, without ulceration—a very common form; and (3) a round, indurated, cartilaginous area with an elevated edge, which ulcerates, exposing a velvety surface looking like raw ham; it bleeds easily, rarely suppurates, does not spread, and the discharge is thin and watery. This is the *Hunterian chancre*, which is rarer than the second variety, but commoner than the first, and which ulcerates because of dirt, caustic applications, or friction.

A chancre is rarely multiple, but if it is so, all the sores usually appear together as a result of the primary inoculation; they seldom follow one another because of auto-infection, although during the first twelve days such a result is possible. A hard sore does not suppurate unless irritated by caustics, friction, or dirt, or unless there be mixed infection with chancroid; its nature is not to suppurate. The hardness may affect only the base and margins of an ulcer or it may affect considerable areas, but it has well-defined margins and feels like cartilage encapsuled, so that it can be picked up between the fingers. This hardness or sclerosis is due to gradual inflammatory exudation into "the tissue at the base of the ulcer and to growth of the nodule" (von Zeissl). It feels distinct from the surrounding tissues, like a foreign body lying in the part. A chancre untreated may last many months. The induration usually disappears soon after the appearance of secondary symptoms. A copper-colored spot remains, and does not disappear until the disease is cured. Induration may again appear before the outbreak of some distant lesion.

**Mixed Infection of Chancre and Chancroid.**—Von Zeissl says: "If syphilitic contagion is mixed with pus, a chancre begins as a circumscribed area of hyperemia and swelling, which undergoes ulceration, and does not develop hardness for a period of from ten days to several weeks, and may develop a nodule after the first ulcer has entirely healed." This condition is seen when mixed infection occurs, the chancroid poison being quick, and the syphilitic poison being slow, to act. If chancroid poison is deposited some time after the syphilitic poison has been absorbed, the induration may appear in a few days after the chancroid begins. A soft chancre may arise upon an existing syphilitic nodule and may eat out the induration.

**Diagnosis of Chancre.**—It is necessary to distinguish a chancre from a chancroid and from ulcerated herpes. A chancroid appears in from two to five days after contagion (always less than ten days); it may be multiple from the start, but, even if beginning as one sore, other sores appear by auto-inoculation; it begins as a pustule, which bursts and exposes an ulcer; the ulcer is circular, has thin, sharp-cut, or undermined edges, a sloughy, non-granulating base, and gives origin to a thin, purulent, offensive discharge which is both auto- and hetero-inoculable. These soft sores have no true sclerotic area, do not bleed, produce no constitutional symptoms, and are apt to be followed by acute inflammatory buboes which tend to suppurate. A chancroid causes pain, and the original ulcer enlarges greatly. A chancre appears in about twenty-five days after inoculation (never before ten days); it is generally single, but if multiple sores exist they usually all appear together, for their discharge is not auto-inoculable after the twelfth day if the sore is not irritated; an auto-inoculation of the products of an irritated chancre may produce a soft purulent ulcer. A chancre begins as a desquamating area, an excoriation, or a nodule; if an ulcer forms, its floor is covered with granulations and it is red and smooth; the discharge is thin and scanty and not offensive; the edges are thick and sloping; it is surrounded by an area of induration, and bleeds when touched; there appear about the same time with the induration or very soon after it indolent multiple, unfused enlargements of the adjacent glands, which rarely

suppurate, and it is followed by secondary symptoms. A chancre causes little pain, and after it has existed for a few days rarely shows any tendency to spread. For the first few days after the appearance of a chancre the Wassermann reaction is negative, then, in nearly three-fourths of the cases, it becomes positive. There is, however, dispute as to the value of the reaction in the diagnosis of primary syphilis. Finding the Spirochæta pallida in scrapings from or the discharge of the sore is proof that the lesion is a chancre. A urethral chancre appears after the usual period of incubation; it is situated near the meatus, one lip of which is usually indurated; the discharge is slight, often bloody, seldom purulent; indurated multiple buboes arise; the sore can be seen, and constitutional symptoms follow.

Herpetic ulceration has no period of incubation; it may follow fever, but usually arises from friction or irritation due to dirt or acrid discharges. It appears as a group of vesicles, all of which may dry up, or some may dry up and others ulcerate, or they may run together and ulcerate. The edges of a herpetic ulcer are in segments of circles; the ulcer is superficial, has but little discharge, and does not have much tendency to spread; it has no induration; it is painful; it is not accompanied by bubo unless suppuration is marked. Herpes is not followed by constitutional involvement.

A chancre may be mistaken for cancer of the tongue. "A chancre of this region is brownish red, a cancer being bright red. A chancre is soft in the center; a cancer presents uniformity of induration. A chancre gives origin to a thin, purulent discharge, free from blood; a cancer furnishes a non-purulent, bloody discharge. A chancre is soon followed by indolent lymphatic enlargements under the jaw; a cancer is followed by painful enlargements." A cancer is slower in evolution, is not followed by constitutional symptoms, and the lymphatic enlargements are much later in appearing than in chancre.

**Phagedena.**—A chancre or a chancroid may be attacked by phagedena, a destructive form of ulceration which was once common, but at present is rare. The ulceration often spreads on all sides and also deeply into the tissues. In some cases it spreads at the edge in one direction (*serpiginous ulceration*), in some cases sloughing occurs. Phagedena occurs only in the debilitated (anemics, drunkards, strumous subjects, sufferers from diabetes, Bright's disease, etc.; salivation can cause it). The phagedenic ulcer is irregular, with congested and edematous edges, and a foul, sloughy floor.

**Chancre Redux.**—Some observers believe that reinfection with syphilis is not very unusual. Most authorities maintain that it is very rare. The latter school maintains that the region once occupied by a chancre may, after months or several or even many years, become indurated anew. Occasionally such a relapse occurs during full treatment. Fournier pointed out this fact thirty years ago. Such a reinduration is called *chancre redux*, or relapsing chancre.

If syphilitic manifestations follow such an induration, we must conclude that reinfection has truly occurred. If they do not follow, and this is the rule, the lesion is not really a chancre, but is probably a gumma in an early stage of development. Mauriac pointed out this last fact.[1]

In **syphilitic bubo** anatomically related lymphatic glands enlarge about the same time as or at least very soon after induration of the initial lesion begins. In the very beginning these glands may be a little painful, but the pain is slight and of temporary duration. These enlargements are called "indolent buboes"; they may be as small as peas or as large as walnuts, are freely movable, and very rarely suppurate. The lesion of the glands is hyperplasia of all the gland-elements and of their capsules, due to absorption of the virus. If the patient is tuberculous, the bubo is apt to become enormous,

[1] Mracek, in "Wien. klin. Rundschau," 1896; H. G. Antony, in "Chicago Medical Recorder," April, 1899.

lobulated, and persistent. If the chancre appears on the penis, the superficial inguinal and femoral glands enlarge, usually on the same side of the body as the sore. If the sore is on the frenum, both groins are involved. If a chancre appears on the lip or tongue, the bubo is beneath the jaw. These buboes may remain for many months; they do not break down unless the sore suppurates or unless the patient is of the tuberculous type; and they finally disappear by absorption or fatty degeneration. About six weeks after buboes have formed in the glands related to the lesion all the lymphatics of the body enlarge. General lymphatic involvement arises about the time the secondary eruption appears. The enlargement of the postcervical and epitrochlear glands is diagnostically important. Glandular enlargements persist until after the eruptions have disappeared.

Glandular enlargement always occurs in syphilis, but the bubo exists in only one-third of the chancroid cases. The bubo of syphilis is multiple, consisting of a chain of movable glands (the glandulæ Pleiades of Ricord); the bubo of chancroid is one inflamed and immovable mass. The bubo of syphilis is indurated, painless, small, and slow in growth; the bubo of chancroid shows inflammatory hardness, is painful, large, and rapid in growth; the first rarely suppurates, the second often does. The skin over a syphilitic bubo is normal; that over a chancroidal bubo may become red and adherent. A syphilitic bubo is not cured by local treatment, but is cured by the internal use of mercury and is followed by secondary symptoms. A chancroidal bubo requires local treatment, is not cured by mercury, and is not followed by secondaries. Herpes, balanitis, and gonorrhea rarely cause bubo, but when they do the bubo in each case is similar to that caused by chancroid. A positive diagnosis of syphilis can be made when an indurated sore on the penis is followed by multiple indolent buboes in the groin and by enlargement of distant glands.

**General Syphilis.**—As the general lymphatic enlargement becomes manifest a group of symptoms known as *syphilitic fever* may appear. In many mild cases, however, fever is absent and the eruption is the first sign of constitutional involvement. The patient usually thinks he has a severe cold, is feverish and restless; complains of headache, lassitude, sleeplessness, and anorexia; his face is pale; he has intermitting rheumatoid pains in the joints and muscles, especially of the shoulders, arms, chest, and back, which pains change their location constantly and prevent sleep; night-sweats occur, and the pulse is quite frequent. The fever usually reaches its height in forty-eight hours, and falls as the eruption develops. The eruption develops usually in from forty-eight to seventy-two hours after the onset of the fever, but may not do so for one week or even more. The fever and the discomfort are worse at night. In type the fever may be intermittent, remittent, or continued. It is usually intermittent. There may or may not be chills, and chills may occur every day or irregularly. Prolonged syphilitic fever with delay in the appearance of the eruption gives rise sometimes to great errors in diagnosis. Prolonged and irregular fever is apt to arise in visceral syphilis, especially syphilis of the liver. In syphilitic fever there are anemia, trivial leukocytosis, and a marked fall in hemoglobin. Syphilitic fever may reappear during the progress of the disease.

**Secondary Syphilis.**—The phenomena of secondary syphilis are due to poisoned blood. They are "local reactions against the spirochetes which have now become disseminated by the blood-stream" (H. G. Adamson, in the "Lancet," April 6, 1912). During untreated active secondary syphilis the Wassermann reaction is practically always positive. Scrapings from a lesion may show the causal organisms. Secondary syphilis is characterized by plastic inflammation, by the formation of fibrous tissue, and by thickening of tissue.

Superficial ulcerations may occur. Structural overgrowths appear (for instance, warts).

**Syphilitic Skin Diseases.**—*Syphilodermata* (*syphilids*) are due to circumscribed inflammation, and may be dry or purulent. There is no one eruption characteristic of syphilis. This disease may counterfeit any skin disease, but it is an imitation which is not perfect and is never a counterpart. Syphilitic eruptions are often circumscribed; they terminate suddenly at their edges, and do not gradually shade into the sound skin. In color they are apt to be brownish red, like tarnished copper; especially is this the case in late syphilids. Hutchinson cautions us to remember that an ordinary non-specific eruption may be copper colored, especially in people with dark complexions and when it occurs on the legs. Eruptions are apt to leave a brownish stain. Early syphilitic eruptions are symmetrical. Syphilitic eruptions have an affection for particular regions, such as the forehead, the abdomen and chest, the neck and scalp, about the lips and the alæ of the nose, the navel, anus, groins, between the toes, and upon the palms and soles. Early secondary eruptions rarely appear on the face or hands. Specific eruptions are polymorphous, various forms of eruption being often present at the same time, so that roseola is seen here, papules there, etc. These syphilids do not cause as much itching as do non-specific eruptions, except when they occur upon the scalp, about the anus, or between the toes. The late secondary eruptions tend to an arrangement in curved lines.

**Forms of Eruption.**—The chief forms of eruption are: (1) erythema, (2) papular syphilids, (3) pustular syphilids, and (4) tubercular syphilids. Besides these eruptions pigmentation may occur (pigmentary syphilid), and blood may extravasate (purpuric syphilid).

Prince A. Morrow does not believe in erecting the vesicular syphilids into a special group. He tells us that vesicles sometimes form on erythemato-papular lesions, but their presence is an accident and not a regular phenomenon. So, too, the bullous syphilid is a rare accident in a case, and even when it occurs soon becomes pustular. The pemphigoid syphilid is found almost exclusively in hereditary disease.[1]

1. **Erythema** (*Maculæ, Roseola,* or *Spots*).—This eruption usually comes on gradually, crop after crop of spots appearing, and many days passing before an extensive area is covered. Occasionally, however, it arises suddenly (after a hot bath, after taking violent exercise, or after eating an indigestible meal). This eruption consists of circumscribed, irregularly round, hyperemic spots, about ½ inch in diameter, whose color does not entirely disappear on pressure in an old eruption, but does in a recent one. The color is at first light pink, but it becomes red, purple, or even brown. In the papular form of erythema the spots are slightly elevated. Erythema is rare upon the face and the dorsum of the hands and feet. It attacks especially the chest and belly, but appears often on the forehead, the bend of the elbow, and the inner portion of the thigh, the neck, and the flexor surface of the forearms and arms. It appears first on the abdomen and last on the legs. Usually erythema follows syphilitic fever, about six weeks after the chancre appears, and the number and distinctness of the spots are in proportion to the violence of the fever. No fever or slight fever means there will be but few spots and they will soon disappear. In rare cases the eruption is very transitory, lasting but a few hours, but it usually continues for several weeks if untreated. It may pass away or may be converted into a papular eruption. Mercury will cause it to disappear in a couple of weeks. In examining for this form of eruption in a doubtful case, let cold air blow upon the chest and belly; this blanches the sound skin and makes clear any discoloration. No desquam-

[1] Morrow's "System of Genito-urinary Diseases, Syphilology, and Dermatology."

ation attends the macular eruption, but a brownish stain remains for a variable time after the eruption fades. Erythema means, as a rule, a mild and curable attack. Maculæ may be combined with the next form, constituting a maculopapular eruption.

The maculopapular syphilids are evolved from the macular syphilids. They are slightly elevated, are situated upon hyperemic bases, and the summits of some of them may undergo slight desquamation. A roseolar area may show one or several of these macular papules. They are apt to arrange themselves in segments of circles and are symmetrically distributed. This eruption usually appears early, but may appear late. It may fade and reappear several times in the same patient. The eruption lasts a few weeks.

Fig. 134.—Condylomata (Horwitz).

2. **Papular syphilids,** which are papules or elevations covered with dry skin, may or may not desquamate. If they do desquamate, the process begins over the center. They usually appear from the third to the sixth month of the disease. They may be preceded by fever, and often reappear again and again. They are at first small and red, but become larger and brownish. They are firm in feel and vary in size from the head of a pin to a five-cent piece or larger. Very large papules constitute nummular syphilids. They all tend to scale. The epiderm becomes thin, red, and glistening, splits in the center and desquamates, and a fringe of epidermis surrounds the desquamated area. This process may be repeated once or oftener. When lenticular papules recur in the late secondary stage they are apt to group themselves in circles limited to particular regions (annular or circinate syphiloderm). They may be present as miliary papules, lenticular papules, moist papules, and papules with marked epidermic proliferation resembling psoriasis (papulosquamous eruption). Papules on fading leave very persistent coppery-looking stains. Papules upon the palms and soles constitute the so-called "palmar and plantar *psoriasis*," which appears from three months to one year after the appearance of the chancre. Papules just below the line of the hair on the forehead constitute the *corona veneris*. Papular syphilids appear especially upon the forehead, the neck, the abdomen, and the extremities. The papular or squamous syphilid of the palms and soles begins as a red spot, which becomes elevated and brownish; the epidermis thickens and is cast off, and there then remains a central red spot surrounded by undermined skin. If papules are in regions where they are kept moist (as about the anus), they become covered with a sodden gray film, which after a time is cast off and leaves the papule without epidermis. The sodden papules are called *flat condylomata*, moist or humid papules or plates (Fig. 134). Papules which are at first small may become large. The small or miliary papules constitute *syphil-*

*itic lichen.* The lenticular papules are most common, and strongly tend to scale off. The papular syphilids give a worse prognosis for the constitutional disease than do spots. The syphilitic negro is particularly apt to develop the annular syphiloderm.

3. **Pustular syphilids** arise from papules. The condition is known as *acne* when the apex of the papule softens, *impetigo* when the whole papule suppurates, and *ecthyma* or *rupia* when the corium is also deeply involved. Vesicles occasionally precede pustules. The pustular eruption appears a number of months after infection and later than the papular. The pustular eruption gives a very bad prognosis for the constitutional disease. Rupia is formed by a pustule rupturing or a papule ulcerating, the secretion drying and forming a conical crust which continually increases in height and diameter, while the ulceration extends at the edges. When the crust is pulled off there is seen a foul ulcer with congested, jagged, and undermined edges. Rupia may be secondary or tertiary, and it invariably leaves scars. It appears only after at least six months have passed since the chancre began. Secondary rupia is symmetrical. Tertiary rupia is asymmetrical.

4. **Tubercular syphilids** are greatly enlarged papules intermediate between ordinary papules and gummata.

*Diagnosis Between Secondary and Tertiary Syphilids.*—A secondary eruption is distinguished from a tertiary eruption by the following: the first tends to disappear, the second tends to persist and to spread; the first is general and symmetrical, the second is local and asymmetrical; the first does not spread at its edge, the second tends to spread at its edge, and this tendency, which is designated *serpiginous*, produces an ulcer shaped like a horseshoe (Jonathan Hutchinson). Secondary lesions appear within certain limits of time, develop regularly, and are dispersed by mercurial treatment. Tertiary lesions appear at no fixed time, develop irregularly, and are not cleared up by mercury.

**Affections of the Mucous Membranes.**—The chief lesions in syphilitic affections of the mucous membranes are mucous patches, warts, and condylomata. The first phenomena of secondary syphilis are, as a rule, symmetrical ulcers of the tonsils, painless, of temporary duration, and superficial (Hutchinson). The borders of the ulcers are gray and the areas are reniform in shape. Catarrhal inflammations often occur. Eruptions appear on the mucous membranes as upon the skin. *Mucous patches* are papules deprived of epithelium; they are gray in color, are moist, and give off an offensive and virulent discharge. They usually appear as areas of congestion, swelling, and abrasion of the epidermis upon the lips, palate, gums, tongue, cheeks, vagina, labia, vulva, scrotum, anus, and under the prepuce. A moist papule of the skin is really a mucous patch. These patches, which are always circular or oval, are among the most constant lesions of the secondary stage, appearing from time to time during many months. If a patch has the papillæ destroyed, it is called a *bald patch.* If the papules present hypertrophied papillæ fused together, there appear enlargements with flat tops, termed *condylomata* (Fig. 134); if the papillæ of the papules hypertrophy and do not fuse, the growths are called *warts.* Mucous lesions of the mouth are commonest in smokers and in those with bad or neglected teeth. Hutchinson says that persistence in smoking during syphilis may cause leukomata, or persistent white patches. The vagina and lips of the vulva during the secondary stage are often covered with mucous patches. The uterus may contain mucous lesions which poison the uterine discharge. The larynx may suffer from inflammation, eruptions, and ulceration (hence the hoarse voice which is so usual). The nasal mucous membrane may also suffer. The rectal mucous membrane may be attacked by patches, and so may the glans penis, the inner surface of the prepuce,

and the urethra. Early in the secondary stage in some cases there is a slight mucopurulent urethral discharge, and examination with an endoscope shows redness of the mucous membrane of the anterior urethra. The discharge is contagious. The condition may be followed by constriction of the urethral caliber. Distinct ulceration may take place.

**Affections of the Hair.**—In syphilis the hair is usually shed to a great extent. This loss may be widespread (beard, mustache, hair of head, eyebrows, pubic hair, etc.) or it may be limited. Complete baldness sometimes ensues, but it is rarely permanent. The hairs of the head are first noticed to come out on the comb; on pulling them they are found loose in their sheaths—so loose that Ricord has said "a man would drown if a rescuer could pull only upon the hair of the head." The falling out of the hair, which is known as *alopecia*, usually begins soon after the fever or about the time of the eruption, but it may be postponed until much later. The skin of a syphilitic bald spot is never smooth, but is scaly. The hair may thin generally, baldness may appear in twisting lines, or it may be complete only in limited areas. Alopecia results from shrinking of the hair-pulp, death of the hair, and casting off of the sheath.

**Affections of the Nails.**—*Paronychia* is inflammation and ulceration of the skin in contact with a nail and extending to the matrix. The nail is cast off partially or entirely. *Onychia* is inflammation of the matrix, and is manifested by white spots, brittleness or extended opacity, twisting, and breaking off of the nail. The parts around are not affected. The damaged nail drops off and another diseased nail appears.

**Affections of the Ear.**—Temporary impairment of hearing in one or both ears is not uncommon in syphilitic affections of the ear. Rarely, permanent symmetrical deafness is produced. Ménière's disease is sometimes caused by syphilis.

**Affections of the Bones and Joints.**—In syphilis there may be slight and temporary periostitis. Pain and tenderness arise in various bones, the pain being worse at night (*osteocopic pains*). Osteoperiostitis usually arises with or after the onset of the secondary eruption, but in rare instances precedes the syphilids. The bones usually involved are the tibiæ, clavicles, and skull. Intense headache may be due to periostitis of the inner surface of a cranial bone. Local periostitis may form a *soft node*, which by ossification becomes a *hard node*. Pain like that of rheumatism may affect the joints. It is not increased by motion and is worse at night. Such pains are by no means uncommon and in some cases are very severe. The joints are not stiff except perhaps on rising. Paton reminds us that such arthralgia is an early symptom and may actually antedate the secondary eruption ("Brit. Med. Jour.," Nov. 28, 1903). More common than the above condition is synovitis, acute or chronic. It often comes on rapidly without other symptoms and is announced by swelling, tenderness, and pain. In some cases the pain is severe, and the patient is feverish or actually ill. Such cases constitute what is called *syphilitic rheumatism*, but the profuse sweats of acute rheumatism are absent, the heart is never attacked, the skin is not red, the fever is not high, and the condition is not migrating (Ibid.). Hydrarthrosis may arise in the knee as a sequence of either of the above conditions, or, late in the secondary stage, it may arise without such an antecedent trouble (Paton). Symmetrical synovitis has been noted. Secondary syphilitic disease of bone, periosteum, and joints lasts only a short time and is never destructive.

**Affections of the Eye.**—*Iritis* is the commonest eye trouble which may arise during secondary syphilis. It appears from three to six months after the chancre, and begins in one eye, the other eye soon becoming affected. The symptoms are a pink zone in the sclerotic, a congested, red or muddy iris, irregularity of the pupil accentuated by atropin, the existence of pain and photo-

phobia, and sometimes hazy or even clouded pupil. Rheumatic iritis causes much pain and photophobia, syphilitic iritis comparatively little; there is less swelling in the first than in the second; the former tends to recur, the latter does not. Iritis is usually recovered from, good vision being retained. Diffuse retinitis and disseminated choroiditis never occur until a number of months have passed since the infection. The symptoms are failure of sight, muscæ volitantes, and very little photophobia. The diagnosis of retinitis and choroiditis is made by the ophthalmoscope.

**Affections of the Testes.**—**Syphilitic Sarcocele.**—The testicle enlarges because of plastic inflammation. Both glands usually suffer, but not always. Fluid distends the tunica vaginalis. The epididymis escapes. The testicle is not the seat of pain, is troublesome because of its weight, and has very little of the proper sensation on squeezing. The plastic exudate is generally largely absorbed, but it may organize into fibrous tissue, the organ passing into atrophic cirrhosis.

**Nervous System.**—Syphilis of the nervous system may arise as early as the sixth month after infection, although the nervous system is far more apt to suffer in the intermediate period or in the tertiary stage. Actual deposits in the brain or cord do at times take place in the secondary stage. These deposits call for prompt and active treatment or they will cause permanent damage. Such lesions are particularly common in untreated cases and in cases in which secondary manifestations were slight or perhaps even unobserved.

**The Albuminuria of Secondary Syphilis.**—It is not very unusual for nephritis with albuminuria to develop early in the secondary stage. There may be the ordinary symptoms of nephritis, but in many cases there is albuminuria and nothing more. Large amounts of albumin run away from the kidneys and the high percentage of albumin in the urine is a notable feature. Many of these cases recover completely, some become chronic, and in some death occurs. It seems probable that mercurial treatment is, in part at least, responsible for some of these cases. The syphilitic poison causes the others. Those in which there is albuminuria and nothing more are due to syphilis rather than to mercury. Those in which there is dropsy are aggravated and perhaps caused by mercury (Fleissinger, in "Journal des Practicens," August 3, 1907).

Fig. 135.—Serpiginous ulcers.

**Intermediate Period.**—Secondary lesions cease to appear in from eighteen months to three years. In the intermediate period no symptoms may appear, yet the disease may be still for some time latent and not cured. The Wassermann reaction may be negative. Symptoms may arise from time to time. These symptoms, which are called *reminders*, are not so severe as tertiary symptoms, are apt to be symmetrical, and do not closely resemble secondary lesions. Among the reminders we may name palmar psoriasis and sarcocele. Sarcocele in this stage is bilateral and rarely painful. Bilateral indolent epididymitis occasionally occurs. Sores on the tongue, a papular skin eruption, and choroiditis may arise. Gummata occasionally occur in this stage, but they are apt to be symmetrical and non-persistent. Sym-

metrical superficial dactylitis may occur. Arteritis may develop, beginning in the intima or adventitia, and causing, it may be, aneurysm, thrombosis, or embolism. Obliterative endarteritis may cause gangrene. Vascular changes are notably common in the vessels of the brain, and thrombosis may occur, in which case paralysis usually comes on gradually, preceded by numbness, although sudden paralysis may take place. The paralysis may be limited, extensive, transitory, or permanent. The nervous system often suffers in this stage (anesthetic areas and retinitis). The viscera are often congested and infiltrated (liver, spleen, kidneys, and lungs).

**Tertiary Syphilis.**—This stage is not often reached, the disease being cured before it has been attained. About 85 per cent. of syphilitics escape it entirely. In this stage there is greatly impaired nutrition the result of the prolonged disease. Until recently it was generally thought that tertiary lesions are not contagious. This statement is now known to be untrue. They are not nearly as contagious as the primary lesions, as secondary lesions, or as blood of the secondary stage, but they are contagious, though feebly so. A tertiary lesion contains spirochetes, but not nearly so many as a secondary lesion. The primary stage disappears without treatment, the secondary stage tends ultimately to spontaneous disappearance, but tertiary lesions tend to persist and to recur. Tertiary lesions may be single or may be widely scattered; when multiple they are not symmetrical except by accident. These lesions may attack any tissue, even after many years of apparent cure; they all tend to spread locally, they all leave permanent atrophy or thickening, they all tend to relapse, and a local influence is often an exciting cause. Tertiary syphilis may cause marked anemia and it is sometimes the cause of pernicious anemia (Dumas and Pirrot, in "La Presse Médicale," xv, Nos, 39 and 40).

**Tertiary skin eruptions** are liable to ulcerate. Various eruptions may occur: papular syphilids, pustular syphilids, gummatous syphilids, serpiginous syphilids, and pigmentary syphilids. The characteristic syphilid is *rupia*, which is formed by a pustule rupturing or a papule ulcerating. A brown or black crust forms because of the drying of the discharge, ulceration continues under the crust, new crusts form, and, as the ulcer is constantly increasing peripherally, the new crusts are larger in diameter than the old ones and the mass assumes the form of a cone. An ulcer which has destroyed the deeper layers of the skin is exposed by tearing off the crust. On healing, a rupial ulcer always leaves a permanent scar.

**Serpiginous ulcers** (Fig. 135) are common in tertiary syphilis, and are especially common about the knees, nostrils, forehead, and lips. Serpiginous ulceration is spoken of as *syphilitic lupus*. It is preceded by a widespread brown-colored nodular cutaneous infiltration. The nodules suppurate, run together, crust, and produce an ulcer which spreads rapidly and assumes the shape of a horseshoe.

The **gumma** (Fig. 136) is the typical tertiary lesion. In some cases there is a solitary gumma; in others, two or three or even many gummata. A gumma is a mass of granulation tissue, grayish-yellow in color, containing many cells and few fibers. Organization of the gumma fails to take place because of a want of sufficient blood-supply, the cellular mass is apt to undergo caseation, and when this occurs an ulcer forms. One portion of the mass may caseate, another portion may become fibrous. In some cases the entire gumma becomes fibrous. A gumma varies in diameter from $\frac{1}{8}$ inch to 2 or 3 inches, presents a center of gummy degeneration, a surrounding area of immature fibrous tissue, and an outer zone of embryonic tissue and leukocytes. A gumma, when it is spontaneously evacuated, exhibits a small opening or many openings with very thin red and undermined edges; the ulcer is slow to heal, and forms a thin scar, white in the center, but pig-

mented at the margins and usually depressed (Jonathan Hutchinson, Jr.). The *gummatous ulcer* is deep, circular in outline, with undermined edges and an uneven floor, which is usually covered with a thick, white, adherent slough. Sometimes there is no slough, but an extensive area is infiltrated. A gummatous

Fig. 136.—Gumma of the clavicle.

ulcer may coalesce with one or more adjacent ulcers. The discharge is scanty and tenacious. These ulcers are often seen upon the legs, and when once healed rarely recur. A gumma in the internal organs may become a fibrous mass. Gummata form in the skin, subcutaneous tissues, submucous structures, muscles, tongue, joints, bones, bursæ, testes, spinal cord, brain, and internal organs. In tertiary syphilis an inflammation may not form a circumscribed gumma, but, instead, may produce a diffuse degenerating mass. This type of inflammation, which is seen in bones, is called "gummatous." In the nasal cavity a gumma is rapidly followed by an ulcer and there is a strong tendency to necrosis of the vomer and sometimes of the turbinated bones. This condition produces a foul discharge and is known as syphilitic *ozena*. Advanced necrosis of the nasal bones causes the nose to "fall in," which is a hideous deformity (Fig. 137). The commonest laryngeal lesion is multiple ulceration following minute gummata. A healing gumma in a mucous canal such as the rectum or larynx causes thickening and stricture. Tertiary syphilis is a common cause of amyloid degeneration and the most frequent cause of arterial and nervous sclerosis.

Fig. 137.—Destruction of the nose in tertiary syphilis.

**Various Lesions.**—Hutchinson enumerates the lesions of tertiary syphilis as follows: *Periostitis*, forming nodes or causing sclerotic hypertrophy, suppuration, or necrosis; gummata in various parts; disease of the skin of the type of rupia or lupus; gumma or inflammation of the tongue, causing sclerosis;

structural changes in the nervous system, causing ataxia, ophthalmoplegia externa and interna, general paresis, optic atrophy, and paralyses of cerebral nerves; amyloid degenerations; and chronic inflammation of certain mucous membranes (of the mouth, pharynx, vagina, rectum, etc.), with thickening and ulceration. Gummatous infiltration of the eyelid is sometimes observed. Gummatous osteoperiostitis of the vertebræ may arise, and this may be associated with disease of the membranes or cord. Syphilitic inflammation of vertebræ is called syphilitic spondylitis. Unilateral enlargement of the epididymis is sometimes noted, the mass feeling heavy, aching a little, but not being very tender. Unilateral sarcocele may be met with. Gummata may arise in the iris, the larynx, the rectum, and the nose.

**Tertiary Syphilis of Bones.**—The bones particularly liable to disease are the skull, sternum, clavicle, nasal septum, and tibia. There may be productive periostitis. This arises in the deeper layer of the periosteum. It may be one limited area, several, or many areas; may be circumferential, or may involve the length of the shaft of a long bone. In most cases the bone is also involved (osteoperiostitis). The bone thickens under the periosteum or toward the medullary cavity, or in both directions. The cuticular spongy substance may be the seat of disease. The lesion may be small and circumscribed or extensive. The thick bone becomes dense (sclerosis). In protracted cases of bone overgrowth in a long bone, if growth occurs in the direction of the long axis, the bone will become bent. As a matter of fact, each of the above lesions is a gumma which undergoes resolution or organization. Syphilitic periostitis is a superficial gumma. Syphilitic osteitis is a deep gumma. Syphilitic osteomyelitis is a deep gumma which has not undergone resolution or organization. The bone about a deep gumma is thickened. There is usually but one gumma in a bone, but there may be two or several. A large gumma weakens bone so that fracture may take place from slight force. Caries or necrosis may arise. A sequestrum seldom forms unless there is mixed infection. Periostitis affects particularly the superficial bones (tibia, clavicle, sternum, ulna, etc.). It begins in the deeper layer of the periosteum, swelling arises, gummy changes occur, and the bone beneath is more or less destroyed. In the skull the bone may be completely penetrated. Not unusually syphilitic periostitis arises at the seat of a trivial injury. Syphilitic osteomyelitis occurs particularly in the phalanges and skull. An area of syphilitic bone disease may undergo repair, osteosclerosis usually and osteoporosis sometimes resulting. After perforation of the skull there is no bony repair. Syphilis of the bones of the nose is necrosis resulting from gummatous ulceration. The *x*-ray picture is usually most valuable in reaching a diagnosis of tertiary syphilis of bone. A syphilitic lesion, if visible, usually has a distinct outline. A pure periosteal mass may not show at all. The bone lesion shows the deep shadow of thickening throughout or, as in the gumma, thickening around a much lighter region. Tuberculosis produces absorption of bone and light areas in the plate. Sarcoma of the periosteum always shows. Sarcoma has not a distinct margin like syphilis.

**Tertiary Dactylitis** (Fig. 138).—This condition is a gummatous formation in a finger or toe. There is a superficial form in which the deposit begins in the subcutaneous tissue and subsequently involves the joint ligaments. In a toe the entire digit is usually involved, in a finger the condition is usually limited to the proximal phalanx. Superficial dactylitis is a very early tertiary phenomenon. A painless swelling gradually forms and it is most distinct on the dorsal surface. The swelling becomes purplish or reddish-blue in color and the joint becomes preternaturally mobile. The swelling may ulcerate.

Deep dactylitis is a very late tertiary manifestation and is osteomyelitis or periostitis of the fingers or perhaps of the toes. One or more proximal pha-

langes are apt to suffer. The skin seldom suffers. Caries and necrosis may occur and the joint may be destroyed, or the bone may be partially absorbed and shortened from dry caries. Ulceration of the skin is rare.

**Tertiary Syphilis of Joints.**—(See the careful study of E. Percy Paton, in "Brit. Med. Jour.," Nov. 28, 1903.) The knee-joint is most commonly affected. Chronic synovitis may arise with considerable or even great swelling (hydrarthrosis), trivial pain, slight functional impairment, some thickening of synovial membrane, and some harshness or grating on movement. Gummatous synovitis may arise, a condition which sometimes follows the ordinary synovitis, but more often exhibits very little swelling. The synovial membrane exhibits irregular areas of thickening and the symptoms resemble those of a tuberculous joint (Paton).

In some syphilitic joints the disease begins in the bone and cartilage. In such a condition there is rigidity, marked limitation of movement, pains, not often severe, and some deformity (Ibid.). Again, as Paton points out, a joint may be involved by an adjacent syphilitic area, synovitis arising, or, if a gumma breaks into a joint, secondary pyogenic infection may follow. Ankylosis may follow joint syphilis.

**Visceral Syphilis.**—Amyloid changes may occur in any of the viscera of an individual with tertiary syphilis, and such changes may be found in people in whom suppuration never occurred. The lungs may undergo fibroid induration (*syphilitic phthisis*). Syphilitic phthisis is often a non-febrile malady. The sputum does not contain the bacilli of tuberculosis, night-sweats and diarrhea are unusual, and emaciation and exhaustion are less decided than in tuberculosis. Gummata may form in the heart, liver, spleen, or kidneys. The capsule and fibrous septa of the liver may thicken, the organ being puckered by contraction. Albuminuria may occur in tertiary syphilis. It may be caused by fibroid changes in the kidneys, by the formation of gummata, or by amyloid degeneration. Its occurrence should be watched for. Mercury and iodid of potassium have been regarded as causative of albuminuria in some cases. When albuminuria is associated with arterial disease and elevated tension, the condition is to be regarded as parasyphilitic rather than syphilitic. When albuminuria results from a true syphilitic lesion of the kidney, there is enlargement of the liver, that organ is often painful, there is ascites, and sometimes jaundice (Fiessinger).

Syphilis may cause disease of the stomach, and probably does so more frequently than was formerly supposed, because it is difficult to distinguish from more common diseases. The condition may be gummatous infiltration of the walls of the stomach, multiple and minute gummata, ulcerations resulting from breaking down of gummata, or syphilitic endarteritis of the gastric vessels. When ulcers heal, cicatricial contraction results. Sometimes a large mass can be palpated. The symptoms last for years. There is pain after eating, but hemorrhage does not occur unless ulcer forms. Syphilitic ulcers and gummata of the stomach may be cured by efficient antisyphilitic treatment. Like lesions may form in the intestines. Flexner, Fränkel, Fournier, and others have discussed this subject.[1]

**Nervous syphilis** may be manifested by disorders of the brain, cord, or nerves. It is rare after severe secondaries, and is most common when secondaries were light or so trivial as to have escaped observation. Severe secondaries seem to cast off, mitigate, or exhaust the poison. Nervous syphilis may result directly from the specific disease, and such lesions are truly syphilitic. Paresis, locomotor ataxia, myelitis, meningitis, neuritis, arteritis may be directly due to the presence of spirochetes causing the formation of syphilitic

[1] See editorial in "Jour. Amer. Med. Assoc.," March 24, 1900, and Roudnitzky, quoted in 'Progressive Medicine," June, 1908, from "Prakt. Vratch," August and September, 1907.

tissue. Nervous syphilis may result indirectly from the specific disease, but not be directly caused by spirochetes having formed syphilitic tissue. Such lesions are called *parasyphilitic.* For instance, a gumma of the brain is a true syphilitic lesion, but locomotor ataxia following syphilis is often a parasyphilitic lesion. As a matter of fact, the spirochetes may act directly on nerve matter without forming syphilitic tissue. Such a condition, though degenerative, is syphilitic, not parasyphilitic. Syphilitic lesions are improved or cured by antisyphilitic treatment, parasyphilitic conditions are not. The diagnosis between syphilitic and parasyphilitic lesions is often impossible without the therapeutic test (mercury, salvarsan). The former are far more apt to show positive Wassermann reactions than the latter. We must remember that brain syphilis is usually a late phenomenon (from one to thirty years after infection). The lesion may be gumma of the membranes (tumor), gummatous meningitis, arterial atheroma, or obliterative endarteritis. A gumma may eventuate in a scar, a cyst, or a calcareous mass. The symptoms of brain syphilis depend on the nature, seat, and rate of development of the lesions. It is to be noted that syphilitic palsy is apt to be limited, progressive, and incomplete. Epilepsy appearing after the thirtieth year is very probably specific if alcohol as a cause can be ruled out. Persistent headache, tremor, insomnia or somnolence, transitory, limited, and erratic palsies, unnatural slowness of utterance, amnesia, vertigo, and epilepsy are very suggestive of syphilis. Sudden ptosis is very significant; so is sudden palsy of one or more of the extrinsic eye-muscles. In syphilitic insomnia the patient cannot get to sleep at night for a long while, but when he once gets to sleep he reposes well. The type of insanity which is most apt to arise is a likeness or counterpart of general paralysis, and, like ordinary paresis, it is not curable. Most paretics have a syphilitic history. Spinal syphilis may cause sclerosis, a condition like Landry's paralysis, softening, and tumor. Neuritis is not uncommon in syphilis.

**Justus's Test for Syphilis.**—The test described by Justus, in 1894, consists in first estimating the amount of hemoglobin present, then making a single mercurial inunction, and again estimating the hemoglobin. It is claimed that the corpuscles of an untreated syphilitic are unduly sensitive, and if the disease is present a mercurial inunction will cause a loss of 10 to 20 per cent. of hemoglobin within twenty-four hours, which fall persists a few hours and is then followed by a rise to a level above that which existed when the test was applied. It is often demonstrable in secondary, tertiary, or congenital syphilis. It usually fails in latent cases, when an initial lesion is recent, and in early secondary syphilis, and in some diseases other than syphilis the reaction can be obtained.

**The Serum Diagnosis of Syphilis (Wassermann's Test).**—This test can only be employed in institutions possessed of the best laboratory facilities. It is technical in the extreme. In order to understand it certain facts must be known.

Every normal serum contains an activating material known as *complement,* and complement is destroyed by heat. When bacteria or alien corpuscles are injected into a living animal, the tissues of that animal react and *amboceptor* is formed. Amboceptor includes all antibodies. Amboceptor brings together complement and the bacterial cell and it is not destroyed by heat. If we inject the corpuscles of sheep's blood into a rabbit, amboceptor forms and appears in the rabbit's serum. Amboceptor unites with complement and with alien corpuscles and the sheep's corpuscles are dissolved, and the blood of the rabbit now contains a distinct excess of amboceptor. If some of this blood is drawn and the serum is placed in a test-tube, it will dissolve in the tube corpuscles of sheep's blood if they are added to it. If, however, the rabbit serum is heated for one-half hour to 50° C. before being placed in a test-tube, the heat

destroys the complement, and then the rabbit serum will be unable to dissolve the corpuscles of sheep's blood, if they are added, because amboceptor without complement is incapable of effecting the solution.

If, however, after destroying the complement by heat, any other serum is added, the added serum furnishes the necessary complement and the mixture is now able to dissolve sheep's corpuscles.

On these facts the serum diagnosis of syphilis depends.

Wassermann proved that if an extract made of a syphilitic organ is placed in the serum of a syphilitic individual, the amboceptor or antibody will unite with the complement and the organ extract, although the union cannot be recognized by inspection. In order to be able to identify the occurrence of such a union a process must be gone through.

The serum of the patient thought to be syphilitic is heated and complement is thus destroyed. It is then mixed with the organ extract, which unites with the amboceptor of the serum, one arm of the amboceptor being still unsaturated and open for union. Guinea-pig serum is now added to furnish complement. If the patient is syphilitic, the serum complement just added will unite with the unsaturated arm of the antibody or amboceptor. To find out if this has taken place we add the heated serum of a rabbit, which will destroy sheep's corpuscles if fresh serum is added to it.

"Sheep's corpuscles are also added. If the complement contained in the guinea-pig serum that was added was taken up or united with by the syphilitic antibody, there will be none left over, and consequently the added sheep's corpuscles will not be dissolved. If, however, the serum was not syphilitic, the complement will not have been taken up, but will be left over for union with the hemolytic amboceptor of the inactivated rabbit serum, which latter unites with the blood-corpuscles, and the combination causes the solution of the latter" (Wm. J. Butler, "N. Y. Med. Journal," Jan. 30, 1909).

The test is usually made with blood of the suspected individual, but may be made with cerebrospinal fluid. The test can be made with milk taken from the breast of a lactating syphilitic.

Major Harrison (quoted by D'Arcy Power, in "Brit. Med. Jour.," Dec. 7, 1912) applied the test in 489 cases. It was positive in 71.8 per cent. of cases of primary syphilis, in 90 per cent. of cases of secondary syphilis, and in 83.5 per cent. of cases of tertiary syphilis. It was present in 50 per cent. of those in a stage of latency. In paretics it was found in over 80 per cent. of cases; in tabetics in over 50 per cent. of cases.

A positive Wassermann reaction may be obtained in noma, leprosy, scarlet fever, sleeping sickness, and some cases of malaria. Practically, a positive reaction obtained from a patient in the United States nearly always means syphilis. A negative Wassermann does not prove that syphilis is absent, unless it is constantly negative and the patient is not taking mercury. In latent stages of syphilis the reaction often becomes negative for a considerable time. Active mercurial treatment or an injection of salvarsan should cause a positive reaction to become negative. The earlier mercury is given in a case of syphilis, the sooner is a negative reaction obtained.

It seems certain that the serum test has high diagnostic value. It may enable us to be sure of the diagnosis long before the appearance of secondaries. A positive reaction indicates that the poison is active and calls upon the physician to apply active treatment. This should be the rule, no matter how long it has been since there were any external manifestations of the disease. Wet nurses should be tested by this method before being allowed to assume charge of an infant.

**Noguchi's Cutaneous Reaction** ("Jour. of Exper. Med.," 1911).—Noguchi gives the name luetin to an emulsion of dead cells of pure culture of

Spirochæta pallida. Rubbed into the skin it produces a marked reaction in tertiary and hereditary syphilis, but seldom gives a reaction in primary or secondary syphilis (Noguchi, in "Jour. Am. Med. Assoc.," 1912, vol. lviii).

**Diagnosis by Finding the Spirochæta Pallida.**—This method is of the greatest value. The organism, if carefully searched for, is found in chancre, in all the lesions of early secondary syphilis, and in congenital syphilis. Spirochetes, though comparatively few, are found in tertiary lesions. Hence, the old idea that tertiary lesions are not contagious must be cast aside. It is not found in lesions other than syphilis. Spirochetes can be found in a few minutes in material from a syphilitic papule or sore. Williams ("Archives of Diagnosis," Jan., 1910) scrapes the papule or sore lightly with a scalpel, drops a little warm salt solution on it, and examines the salt solution at once.

**Prevention of Syphilis.**—I shall not discuss the various plans at present under consideration for the diminution or extinction of syphilis in communities. It is sufficient to say that no method has yet been decided upon. Desirable as it would be to entirely prevent syphilis, it is at present impossible, as promiscuous sexual intercourse and prostitution are still with us, and destined, for some time at least, to remain.

The individual in endeavoring to avoid syphilis should avoid, as far as possible, all the acts spoken of on pages 317 and 318, which may be responsible for infection. Metchnikoff discovered that if within twenty-four hours of inoculation calomel ointment is rubbed in the area syphilis can be prevented. He used 1 part of calomel and 2 of lanolin. This fact is widely utilized. In the United States Navy when a sailor returns from shore leave and admits to a suspicious connection, the penis and foreskin are rubbed with a mixture containing 33 parts of calomel, 10 parts of vaselin, and 67 parts of lanolin.

If before connection the glans and prepuce are smeared with soap or calomel ointment, the liability to tearing and abrasion is lessened and crypts and follicles are blocked up. If this plan is followed, and if after connection the parts are washed and bathed with a solution of corrosive sublimate (1:2000) or permanganate of potash (1:3000), the danger of infection is greatly lessened. To apply the calomel ointment gives additional assurances of safety.

**Abortive Treatment.**—I do not believe that syphilis can be aborted by cauterization, by excision, or by the administration of mercury, luetin, or salvarsan. Several observers, even during recent years, have claimed that ablation of the chancre will sometimes prevent the disease. In the reported cases there is some doubt as to the diagnosis. Neisser's experiments upon apes demonstrate the futility of excision. He found spirochetes in adjacent glands before the sore had indurated. Injection of another ape with material from these glands was followed by the development of syphilis. Excision causes pain and diagnostic uncertainty and is invariably useless.

**Treatment of the Primary Stage.**—It has long been taught that a chancre should not be excised because the disease is constitutional when the chancre appears, and excision and cauterization inflict needless pain and do no good. The initial lesion should never be cauterized unless it is phagedenic or becoming so. Order the patient to soak the penis for five minutes twice daily in warm salt water (a teaspoonful of salt to a cupful of water), and then to spray the sore with peroxid of hydrogen diluted with an equal bulk of water. The ulcer is then dried with absorbent cotton and on it is dusted a powder composed of equal parts of bismuth and calomel or, better, is dressed with calomel ointment. The buboes in the groin require no local treatment unless they tend to suppurate. If they persist or become large, paint them with iodin or rub ichthyol ointment or mercurial ointment into them, and apply a spica bandage to the groin. Some authorities give mercury in this stage in order to prevent secondaries. The younger Gross opposed this strongly, and affirmed

a wish to see the secondary eruption—first, because it proves the diagnosis; and, second, because it affords valuable prognostic indications (an erythematous eruption means a light case, an early pustular eruption means a grave case with serious complications); I long followed the plan of my old master, and did not order mercury until constitutional symptoms developed. We now know that the development of a positive Wassermann reaction may be regarded as confirmatory of the diagnosis, the finding of spirochetes proves it, and early treatment may prevent disastrous lesions. We make the diagnosis early and at once begin constitutional treatment (see page 336). If phagedena arises, place the patient promptly upon stimulants and nutritious diet, secure sleep, and destroy the ulcer by the use of nitric acid or the cautery while the patient is anesthetized. After cauterization dust the sore with iodoform and dress with wet antiseptic gauze. Several times a day change the dressings, and at each change spray the sore with peroxid of hydrogen, irrigate with bichlorid of mercury solution, and dust with iodoform. It may be necessary to cauterize several times. In some cases it will be necessary to employ continuous irrigation by an antiseptic fluid. These cases are sometimes fatal and usually produce great destruction of tissue. In chancre redux watch carefully for the symptoms in order to determine if the condition is really one of reinfection or if we are dealing with a gumma which resembles a chancre in appearance.

A chancre usually heals promptly after the administration of salvarsan, and quite rapidly when the patient is placed on mercury. It is not wise to give iodid of potash during the primary stage and it is not probable that it is helpful at all. Some teach that iodid of potash helps the absorption of granulation tissue and so lessens induration and promotes healing. It certainly is not as efficient as mercury for this purpose; it does not kill spirochetes, the tissues of the patient soon become "habituated to its presence and excrete it as rapidly as it is ingested" (D'Arcy Power, in "Brit. Med. Jour.," Dec. 7, 1912).

**Treatment of the Secondary Stage.**—The chance of cure in most cases is excellent if the patient follows advice. The prognosis is much worse if the patient is a hard drinker or is the victim of Bright's disease, diabetes, tuberculosis, or other chronic exhausting malady. In the secondary stage the aim is to cure the disease. That it can be cured is known because reinfection occurs in some persons. The old axiom, "Syphilis once, syphilis ever," is not true.

**Diet and General Care.**—In the beginning of treatment the patient must see his physician every day or two until the proper dose of mercury has been ascertained. For the following six months he should see his physician once a week, and during the next six months once every other week. During the second year he needs to see him once every month. Of course, if complications arise at any period the visits must be more frequent. At the beginning of the attack he must have his teeth put in perfect order. Tobacco is absolutely forbidden because its use favors the development of mucous patches in the mouth. Alcohol as a beverage is prohibited. It is used only as a medicine. The teeth should be gently scrubbed with a soft brush in the morning, in the evening, and after each meal, and a mild astringent or antiseptic mouth-wash and gargle is to be used several times a day. If the gums become red and tender, chlorate of potash is used as a gargle and mouth-wash (1 oz. of the drug to 1 pint of water). The patient should wear flannel in winter. The author believes Guiteras's rules are sound, and in accordance with them directs the patient to refrain from kissing any one on the lips and from using a common towel, wash-rag, cup, glass, pipe, or razor. He is told to sleep alone in bed, to wash his hands often, to wear gloves, and to keep his fingers out of his mouth. Every morning he should take a warm bath, being especially careful to cleanse the anus, perineum, axillæ, groins, and between the toes; and after the bath these parts should be dusted with borated talc powder.

A Turkish bath once a week is ordered by Guiteras when no skin eruption exists. The patient must avoid drafts, cold, and wet; must take a moderate amount of gentle outdoor exercise, and must sleep eight hours out of the twenty-four. The diet is of importance, and in this, too, the author follows Guiteras and orders the patient to avoid eating anything fried, or any meat or fish which has been canned, salted, or preserved. Fruits, pickles, tea, condiments, alcoholic beverages, clams, pork, veal, and pastry are not to be taken. (See article by Luke Beggs, in "Phila. Med. Jour.," June 7, 1901.)

**Medical Treatment.**—Mercury cures syphilis. We no longer give it only to remove symptoms, we give it to produce cure. It is given in small doses for a long period. We were taught this by Fournier, Lang, and Sir Jonathan Hutchinson (D'Arcy Power, in "Brit. Med. Jour.," Dec. 7, 1912). Mercury kills the spirochetes. This is proved by the experiments upon apes made by Metchnikoff and Roux. Iodid of potash is seldom used in the secondary stage for the same reasons that it is avoided in the primary stage. Mercury must be used, the form being a matter of choice. Fournier advocated intermittent treatment. In this plan give $\frac{1}{3}$ gr. of protiodid of mercury daily for six months, then stop for a month; then give mercury for three months, then stop two months. During the first year the patient is under treatment nine months, and during the second year eight months. Some prefer the intermittent and others the continuous plan of treatment. The author prefers the continuous plan. In following the continuous plan find the patient's tolerance to mercury, and keep him for two years on daily doses below the amount he will tolerate. Gross's rule for continuous treatment is to order pills of green iodid of mercury, each pill containing $\frac{1}{5}$ gr. The patient is ordered one pill after each meal to begin with; the next day the after-breakfast dose is increased to two pills; the following day the after-dinner dose is two pills, and so on, one pill being added every day. This advance is continued until there is slight diarrhea, griping, a metallic taste, or tenderness on snapping the teeth together, whereupon one pill is taken off each day until all unfavorable symptoms disappear. Then the dose is reduced one-half and this amount is called the tonic dose. This experimentation finds a dose on which the patient can be kept with entire safety for a long time; but if it is found that colic or diarrhea is apt to recur, there must be added to each pill $\frac{1}{12}$ gr. of opium. The patient is given mercury in this way for two years. Every time new symptoms appear the dose is raised, and as soon as they disappear it is lowered to the standard. If the protiodid is not tolerated, give the bichlorid:

℞. Hydrarg. chlor. corros., gr. j;
Syr. sarsaparillæ comp., f℥iij.—M.
Sig.—One fluidram, in water, after meals.

Mercury with chalk in 1- or 2-gr. doses four times a day, with or without Dover's powder in 1-gr. doses, may be used. Mercurial inunctions produce a rapid effect, but irritate the skin. The drug should be rubbed in with a gloved hand. There can be used once a day $\frac{1}{2}$ dr. of oleate of mercury (10 per cent.) or 1 dr. of mercurial ointment, rubbed into the skin. The first day it is rubbed into the inside of one thigh; the second day into the inside of the other thigh; the third day into the inside of one arm; the fourth day into the other arm; next, into one groin and then into the other groin, and then inunction is again made at the point of original application, and so on. After the rubbing the patient puts on underclothes and goes to bed, and in the morning takes a bath. The ointment may be smeared on a rag, which is then worn between the stocking and sole of the foot during the day.

Fumigation is performed by volatilizing each night 1 dr. of calomel. The patient sits naked on a cane-seat chair, and is wrapped up to the neck in a

blanket which drops tent-like to the floor; the calomel is put upon an iron plate under the chair, and is heated by an alcohol lamp beneath the plate. The skin becomes coated with calomel, and the subject, after putting on woolen drawers and an undershirt, gets into bed. Hypodermatic injections of mercury are used by some physicians. They cause an eruption to disappear rapidly, but may produce abscesses, and relapses are prone to occur. The injection method will not abort the disease; should never be a routine treatment; in suitable cases it is very valuable for symptomatic use, as when lesions on the face or in important structures make a rapid impression desirable or necessary; in cases which obstinately relapse under other treatment, and in syphilis of the nervous system. J. William White, after a large experience with this method, says that hypodermatic injections of corrosive sublimate are painful and are strongly objected to by many patients; that this method of treatment is occasionally dangerous and even fatal; that it is liable to be followed by local complications (erythema, nodosities, cellulitis, abscess, sloughing); that it cannot be carried out by the patient, but requires the surgeon's constant intervention. This syphilographer concludes that hypodermatic medication does not offer advantages justifying its use as a systematic method of treatment, and that it encourages insufficient treatment—those "short heroic courses" which Hutchinson shows are followed by the gravest tertiary lesions. "The claim that by a few injections the time of treatment can be measured by months or even by weeks, instead of by years, would seem, as Mauriac has said, to involve the idea that mercury given hypodermically acquires some new and powerful curative property which, given in other ways, it does not possess."[1] The usual plan is to give daily a hypodermatic injection of corrosive sublimate deep into the back or buttock, the dose being $\frac{1}{4}$ gr. of the drug. Thirty such injections are used unless some contra-indication demands their discontinuance sooner. The treatment is then stopped. If the symptoms recur, however, the patient is given another course, the daily dosage being $\frac{1}{6}$ gr., the treatment being again stopped after thirty injections, but being continued anew in $\frac{1}{8}$-gr. doses if the symptoms recur. The following preparation is used by some syphilographers: 0.5 part of corrosive sublimate, 3 parts of guaiacol, and 97 parts of sterile olive oil. Thirty minims contain $\frac{1}{16}$ gr. of corrosive sublimate. This mixture should be thrown deeply into the buttock and it causes no pain. The use of gray oil hypodermatically has warm advocates. It is claimed that it provokes little pain and irritation, and that it is a very efficient remedy. The oil should not be thick like an ointment, because such a preparation could not be used without warming, and heat causes the mercury to aggregate in lumps. Olive oil should not be used, as it becomes rancid. Dumesnil's formula is the best ("Brit. Med. Jour.," Jan. 18, 1908):

"The ingredients must be sterilized before they are incorporated, as it is impossible to sterilize the product. If the directions given below are carefully followed no risk of septic poisoning is to be apprehended. The formula proposed by M. Dumesnil has been accepted by a committee especially appointed by the Société de Pharmacie of Paris, to investigate the methods of preparing gray oil. Twenty-six gm. of anhydrous wool-fat and 60 gm. of pure liquid paraffin (*huile de vaselin médicinale*) are sterilized separately in glass flasks at 120° C. for twenty minutes. A pestle and mortar are sterilized by means of burning alcohol and placed therein are 40 gm. of mercury and then the wool-fat. The metallic particles are triturated until they are sufficiently minute when examined under a magnification of 480 diameters, and then the liquid paraffin is added in small portions. The product should weigh 126 gm. and

[1] J. William White, in Morrow's "System of Genito-urinary Diseases, Syphilology, and Dermatology."

measure 100 c.c. and should be transferred immediately to phials of 2-, 5-, and 10-c.c. capacity, previously sterilized at 180° C."

An injection is given twice during the first week, once during the second week, and after this once a week or once every other week for an indefinite period of time. It may be given oftener if symptoms arise or persist.

Taylor believes that gray oil may give rise to unpleasant and sometimes even dangerous symptoms, and that it should be used with extreme care and only in selected cases in which other remedies are contra-indicated. He says that in reading about the hypodermatic method he has been struck with the fact that "the most serious results have almost invariably followed injections in which fatty matters have been the vehicle of suspension."[1]

Some surgeons employ intravenous injections of mercury. Lane injects, at first every other day and later daily, 20 min. of a 1 per cent. solution of cyanid of mercury. The skin in front of the elbow is rendered aseptic, a fillet is tied around the arm, the needle is inserted into a vein, the fillet is loosened, the fluid is injected, and the needle is withdrawn. This method of using mercury is painless and produces a rapid effect. It may be used in nervous syphilis, but should not be used as a routine. In whatever way mercury is given, do not allow it to produce salivation (hydrargyrism or ptyalism). Always remember that mercury may cause albuminuria and examine the urine at regular intervals during a course of the drug. If albumin appears in the urine, cut down the dose of mercury or stop the drug for a time. In the beginning of a case of syphilis, if the kidneys are found to be diseased, give the mercury cautiously, and never fail to examine the urine at regular intervals. An individual can take more mercury in summer than in winter because during the warm weather perspiration favors elimination.

Sometimes, when a patient has a secondary eruption, the eruption grows temporarily worse when mercury is administered. This is called *Herxheimer's reaction.* It is due, according to Adamson, to having killed some spirochetes and thus caused the liberation of more endotoxins, the endotoxins causing "a further local defensive reaction" ("Lancet," April 6, 1912).

In order to cure syphilis mercury should be given for two years, and the mercurial course must be followed by at least a six months' course of iodid of potash. Reminders require both iodid of potash and mercury (mixed treatment). Throughout the mercurial course the patient should be weighed once a week, and if it is at any time found that the weight is decreasing, tonics, concentrated food, and cod-liver oil are ordered. If the weight continues to grow less and the health begins obviously to fail, stop the mercury for a time, continue the cod-liver oil, tonics and nourishing food, and order hot baths, fresh air, iron, and chlorid of gold and sodium. If during the mercurial course albumin appears in the urine and some edema is noted, the mercury should be stopped for several or a number of weeks and the patient should be given a milk diet. If marked albuminuria is noted, but no other symptoms exist, mercury need not be discontinued, but the patient is watched most carefully for the advent of any other symptom (Fiessinger, in "Journal des Practiciens," August 3, 1907).

**Acute Ptyalism, or Salivation.**—In acute ptyalism the saliva becomes thick and excessive in amount; the gums become spongy and tender and liable to bleed. Tenderness can be detected early by snapping the teeth. A metallic taste is complained of; the breath becomes fetid; the oral structures swell; the teeth loosen; the saliva is produced in great quantity; and there are purging, colic, and exhaustion. Sometimes there are fever and a diffuse scarlatiniform eruption upon the skin. A chronic hydrargyrism may be shown by salivation, gastro-intestinal disorder, emaciation, mental depression,

[1] "Venereal Diseases," by Robert W. Taylor.

weakness, albuminuria, and tremor. To avoid salivation, advance the dose with great caution and instruct the patient as to the first sign of the trouble. He should use a soft tooth-brush and an astringent mouth-wash (48 gr. of boric acid to 4 oz. each of listerine and water). When ptyalism is noted, discontinue the administration of the drug. Employ the above mouth-wash or one composed of a saturated solution of chlorate of potassium. Order $\frac{1}{120}$ gr. of atropin twice a day, and in bad cases spray the mouth with peroxid of hydrogen and use silver nitrate locally (20 gr. to 1 oz.). Give stimulants (iron, quinin, and strychnin) and nutritious food. A weekly Turkish bath is of great service. In chronic hydrargyrism stop the administration of the drug, use tonics, stimulants, open-air exercise, Turkish baths, and nutritious food. The chlorid of gold and sodium forms a substitute for mercury. The use of iodid of potassium is of questionable value in ptyalism.

**Treatment of Complications in the Secondary Stage.**—The complications of the secondary stage usually require local applications in addition to general remedies. Mucous patches in the mouth should be touched with bluestone every day, an astringent mouth-wash being employed several times daily. If the patches ulcerate, they should be touched once a day with lunar caustic; if these areas proliferate, they should be excised and cauterized. Vegetations or growing papules on the skin must, if calomel powder fails to remove them, be cut away with scissors and be cauterized with chromic acid or with the Paquelin cautery. Condylomata demand washing with ethereal soap several times daily, thorough drying, dusting with equal parts of calomel and subnitrate of bismuth or with borated talcum, and covering with dry bichlorid gauze. If these simple procedures fail, excise and cauterize.

For psoriasis of the palms and soles diachylon ointment, mercurial plaster, or painting with tincture of iodin should be employed. Ulcers of paronychia are dressed with iodoform and corrosive sublimate gauze. Deep cutaneous ulcers are cleaned once a day with ethereal soap, sprayed with peroxid of hydrogen, dressed with iodoform and corrosive sublimate gauze, and bandaged. When the process of granulation is well established dress with 1 part of unguent. hydrarg. nitratis to 7 parts of cosmolin. In sarcocele mercurial ointment should be rubbed into the skin of the scrotum or the testicle be strapped. In alopecia the hair should be kept short, and every night the scalp should be cleaned with equal parts of green soap and alcohol rubbed into a lather with water. After the soap has been washed out some hair tonic should be rubbed into the scalp with a sponge. A favorite preparation of Erasmus Wilson's consisted of the following ingredients:

℞. Ol. amygd. dil.,
Liq. ammoniæ, āā f℥j;
Sp. rosemarini,
Aquæ mellis, āā f℥iij.—M.
Ft. lotio.

One part of tincture of cantharides to 8 parts of castor oil may be rubbed into the scalp. Solutions of quinin are esteemed by some. A useful wash for the scalp is the following: 1 dr. of borate of sodium, 1 dr. of spirits of camphor, 2 dr. of glycerin, and sufficient orange-flower water to make 4 fl.oz.

In treating persistent skin-lesions, inunctions, injections, fumigations, or mercurial baths may be used. Baths are suited to patients with delicate skins, to those whose digestion fails when mercury is given by the mouth, and to those whose lungs will not tolerate fumigations. Corrosive sublimate, ½ oz., and 4 scruples of sal ammoniac are mixed in about 4 oz. of water; this is added to a bath at a temperature of 95° F. The patient gets into this bath,

covers the tub with a blanket, leaving only his head exposed, and remains in the bath an hour or so. Mercurial baths may rapidly cause salivation.

**Tertiary Stage.**—If at any time during the case there appear tertiary symptoms, the patient should be put on mixed treatment, that is, mercury and an iodid. In any case, after two years of mercury add iodid of potassium to the treatment. If any tertiary symptoms appear, the rule is to use mixed treatment and to continue it for at least six months after all symptoms disappear, the six months' course dating from their disappearance. This emphasizes the fact that the iodids alone will not cure tertiary syphilis. Iodid of potash does not cure, but is very valuable in removing symptoms. In late syphilis both iodid and mercury are given. It is the mercury that cures. The mercury is given in small or tonic doses. Since the days of Ricord iodid of potash has been held in high esteem. In obstinate tertiary lesions and in nervous syphilis the iodids should be run up to an enormous amount (from 30 to 250 gr. per day). Sometimes people can take large doses of iodid when small doses produce iodism. Cyon explains this curious fact as follows: small doses combine with some products of the thyroid gland and form toxic iodothyrin. Large doses are diuretic, form soluble salts, and are rapidly eliminated. An easy way to give iodid is to order a saturated solution each drop of which equals about 1 gr. of the drug. Each dose of the iodid is given one hour after meals and in at least half a glass of water. If the iodid disagrees, it may be given in water containing 1 dr. of aromatic spirit of ammonia, or 5 drops of fluidextract of ergot, or it may be given in milk. The iodid of sodium may be tolerated better than the potassium salt, as it is less depressing to the circulation, or the iodids of sodium, potassium, and ammonium may be combined. Gotheil sometimes gives tincture of iodin in 10-drop doses, well diluted. Iodid may be given as an enema in milk. In giving the iodids, begin with a small dose. During a course of the iodids always give tonics and insist on plenty of fresh air. Arsenic given daily tends to prevent skin eruptions. The iodids may disagree for a time, but tolerance may be established as the administration is continued. The value of the newer organic iodin preparations is as yet uncertain. Some of them are given hypodermatically. An iodized oil, known as iodipin, can be given by the mouth or by intramuscular injection. If given by the stomach it may be taken in capsules, in tablets, or in milk (Coates, in "Brit. Med. Jour.," May 7, 1910). The iodids when they radically disagree produce *iodism*—a condition which is made manifest by a flow of mucus from the nose, conjunctival irritation, a bad taste in the mouth, exhaustion, anorexia, nausea, and tremor. In some subjects there are outbreaks of acne, vesicular eruptions, or even bullæ or hemorrhages. Iodism calls for the abandonment of the drug, and the administration of increasing doses of Fowler's solution of arsenic, of laxatives, of diuretic waters, or, if there is great exhaustion, of stimulants. In some cases belladonna is of service. Some patients who cannot take the alkaline iodids may take syrup of hydriodic acid. After the patient has been for six months under mixed treatment without a symptom, stop all treatment and await developments. If during one year no symptoms recur, the patient is probably cured; if symptoms do recur, there must be six months more of mixed treatment and another year of watching. It would be wise were every person who has had syphilis to take a six weeks' course of mercury and iodid twice a year for the balance of life. It is probable that such a plan would save many from visceral syphilis and late nervous syphilis. Syphilitic ulcers are treated locally by cleanliness, antiseptic applications, and, if the situation admits of it, by the daily use of the hot-air apparatus or by the induction of hyperemia by means of the rubber bandage or the cupping-glass. If albuminuria arises during the tertiary stage and there is arterial disease with high tension, mercury

will do harm. In albuminuria without high tension it may be given (see pages 327 and 338).

*The Question of Marriage.*—Fournier has insisted that it is a great wrong to tell a syphilitic that he can never marry. He must not marry until he is cured, and he is not cured until, after the cessation of the use of mercury and iodid, he goes one year without treatment and without symptoms.

*Treatment of Syphilis by Salvarsan or "606" (Hydrochlorid of Dioxydiamido-arsenobenzol).*—Various arsenic compounds have been tried in syphilis (atoxyl, cacodylate of sodium, arsacetin, soamin), but salvarsan and neosalvarsan are the most important. Ehrlich introduced "606" (salvarsan) in 1909 and "914" (neosalvarsan) early in 1912. It was hoped that a single intravenous injection would kill all the spirochetes and immediately cure the disease. This hope has not been realized, though the remedy has great power for good. It kills multitudes of spirochetes by directly poisoning them, not by causing the tissues to form antibodies. It causes symptoms to rapidly pass away, but if the administration is not continued at intervals or if mercury is not given, relapse, and probably disastrous relapse, is almost certain to occur. In many cases, but not in all, the Wassermann reaction becomes negative after the injection. The test is rendered negative more quickly than by mercury, but it may soon become positive again. The beneficial effects of these arsenic preparations are particularly manifest in early syphilis, and are very evident in tertiary syphilis. The drugs are of little or no use in parasyphilitic lesions.

In some cases "a spirochete infection which has been in abeyance" can be roused by salvarsan "into sufficient activity to cause the Wassermann reaction to become positive," when it was before negative. Hence, salvarsan and neosalvarsan may be used as tests for syphilis and as tests to determine if syphilis has been cured (D'Arcy Power, in "Brit. Med. Jour.," Dec. 7, 1912).

Salvarsan is not to be given as routine treatment of syphilis because it does not cure; and mercury does. It is not a substitute for mercury, but is used in particular cases for particular reasons. Even when salvarsan is given, mercury and iodid should be given in the same doses and for the same time as they would have been given had salvarsan not been administered. Salvarsan should be given when the patient is intolerant to mercury or when mercury fails to control the disease; when an important structure is threatened with damage or destruction; when the sore becomes phagedenic; for malignant syphilis; for persistent or spreading ulcerations; for laryngeal syphilis; for tertiary syphilis; for glossitis and ulceration of the tongue with leukoplakia, and for periostitis and osteitis in congenital or acquired syphilis. It does no good to syphilitic necrosis, probably because pyogenic organisms are active in such cases, and it is useless for symmetrical synovitis of the knees, possibly because the child victims have also tuberculosis (Ibid.).

A man with persistent mucous patches in the mouth and every prostitute with syphilis should be given salvarsan because of the danger to the community of such syphilitics. A characteristic and disfiguring eruption calls for it. A married man with a chancre should receive it promptly. Salvarsan is contra-indicated when there is advanced disease of the heart and arteries; in degenerative conditions of the brain and cord, when there is optic atrophy; in diabetes; in diseases of the liver or kidneys (even if syphilitic); in pulmonary tuberculosis, and in children before the age of three.

It is estimated by some that salvarsan is responsible for 1 death in every 1000 cases treated. Others set the rate at a much higher figure. Major French ("Lancet," Nov. 18, 1911) refers to 41 deaths as having occurred in less than one year. Levy-Bing, of Paris, says that the deaths number 70 or 80 and that death may occur in a healthy person. Hallopeau, Gaucher and Ra-

vant reported 3 deaths ("Bull. de l'Acad. de Med.," Oct. and Nov., 1911). A man of nineteen developed arsenical poisoning and died of uremia on the sixth day after the injection. A girl of eighteen died in a similar manner. Géronne, Finger, and others seem to prove that the symptoms which may follow the administration of salvarsan are manifestations of acute intoxication by arsenic, and are not, as Neisser claims, produced by endotoxins from destroyed spirochetes. Finger had a fatal case due to arsenical poisoning ("Brit. Med. Jour.," Jan. 27, 1912). Auditory and ocular complications may occur. In 9 per cent. of patients to whom Finger gave salvarsan nerve complications developed. Most of the deaths followed a second injection, hence the claim that anaphylaxis was the cause.

The drug may be given by intramuscular injection. It causes pain which is often severe and prolonged inflammation. This method is employed in children from three to seven years of age, because the veins are small and hard to find. In adults the intravenous method is always to be preferred. It is given in a vein in front of the elbow. One capsule containing .6 gm. of salvarsan is opened, and the drug, which is an acid salt, is dissolved in a hot alkaline solution. A pint of recently sterilized salt solution is used and ten minutes are occupied in running the fluid into the vein. The fluid is given at a temperature a little above blood heat. Neosalvarsan to the amount of .9 gm. is dissolved in 6 oz. of freshly distilled water. It gives a neutral solution and is very soluble. The fluid is injected at a temperature of 60 to 70° F.

Several hours after the injection of salvarsan symptoms may arise (chills, elevation of temperature, diarrhea, vomiting, headache). In a few hours, as a rule, the symptoms pass away. One must be on the watch for dangerous symptoms (cardiac depression, dyspnea, great restlessness, excitement, edema of the face, cyanosis of the face, persistent vomiting, diarrhea, albuminuria, spasm of the limb muscles, and collapse).

The effect of salvarsan on the Wassermann reaction has been much discussed. Theoretically, it should always make a positive reaction negative. Practically, it often does not. It may require a second injection to do it. After the administration of salvarsan the reaction may become negative, remain so for several months, and again become positive. Neisser claims that in only 10 per cent. of latent cases is a negative reaction converted into a positive one. It was stated above that in latent syphilis with a negative reaction salvarsan may actually cause a positive reaction. Evidently, a negative Wassermann is not to be regarded as absolute and unassailable proof that a patient is cured.

**Hereditary Syphilis.**—**Transmitted congenital syphilis** is hereditary syphilis manifest at birth. Acquired syphilis (except in the case of a woman who obtains the disease from a fetus) always presents the chancre as an initial lesion; hereditary syphilis never does. Hereditary syphilis may present itself at birth, and usually shows itself within, at most, the first six months of extra-uterine life. In rare cases (tardy hereditary syphilis) the disease does not become manifest until puberty.

*Rules of Inheritance.*—According to von Zeissl,[1] the rules of inheritance are as follows:

1. If one parent is syphilitic at the time of procreation, the child may be syphilitic.

2. Syphilitic parents may bring forth healthy children.

3. If a mother, healthy at procreation, bears a child syphilitic from the father, the mother must have latent pox, having become infected through the placental circulation, or must be immune. She often shows no symptoms, having received the poison gradually in the blood, and having thus received,

[1] "Pathology and Treatment of Syphilis."

it may be said, preventive inoculations. Certain it is that mothers are almost never infected by suckling their syphilitic children (Colles's law).

4. If both parents were healthy at the time of procreation, and the mother afterward contracts syphilis, the child may become syphilitic, and the earlier in the pregnancy the mother is diseased, the more certain is the child to be tainted. This is known as "infection *in utero*."

5. The more recent the parental syphilis, the more certain is infection of the offspring. The children are often stillborn.

6. When the disease is latent in the parents it is apt to be tardy in the children.

7. The longer the time which has passed since the disappearance of parental symptoms, the more improbable is infection of the children.

8. In most instances parental syphilis grows weaker, and after the parents beget some tainted children they bring forth healthy ones.

Syphilis in the mother is more dangerous to the offspring than syphilis in the father. The frequent immunity of the mother is due to the fact that her tissues produce antitoxins under the influence of the slowly absorbed virus. The milk of a syphilitic mother contains quantities of antibodies (Bab, "Zeitschrift f. Geb. und Gynäkologie," vol. x, No. 2).

Many women affected with hereditary syphilis are sterile. Many syphilitic women abort before the eighth month, most commonly in the fifth month. The fetus very often dies at an early period of gestation. This may be due to a gummatous placenta or to a degeneration of placental follicles. Bab (Ibid.) reports that out of 33 infants, victims of congenital syphilis, 16 were born alive and 7 of these died within a few days. Hyde says that about 90 per cent. of those born living subsequently die of the disease.

*Evidences of Hereditary Syphilis Manifest At or, Oftener, Soon After Birth.*—Hutchinson says that at birth the skin is almost invariably clear. In from six to eight weeks "snuffles" begin, which are soon followed by a skin eruption, by body wasting, and by a chain of secondary symptoms (iritis, mucous patches, pains, condylomata, etc.). The child looks like a withered-up old man. Eruptions are met with on the palms and soles. Intertrigo is usual. Cracks occur at the angles of the mouth and leave permanent radiating scars. The abdomen is tumid and there is apt to be exhausting diarrhea. The secreting and absorbing glands of the intestinal tract atrophy.[1] It is seldom that distinct gummatous tumors form in hereditary syphilis. The type of disease induced is a diffuse interstitial cellular change in the viscera, and the viscera are much more apt to suffer than in acquired syphilis. The liver, spleen, and pancreas often enlarge from interstitial changes, and the lungs sometimes are attacked in the same manner. Synovitis or arthritis may arise, the condition being similar to that met with in acquired syphilis. A form encountered between the third month and end of the second year, according to Paton, is characterized by growth into the joint of fungating granulation tissue, the joint is useless, and the parts about are swollen and edematous. Atrophic lesions may appear in the bones. In the skull the bone may be softened by removal of its lime salts or be thinned by the pressure of the brain. In the long bones the epiphyseal lines suffer, the attachment of the epiphysis to the shaft is weak, and separation is easily induced. Epiphysitis is common, seldom causes pain, and rarely leads to suppuration, except in children who are old enough to walk (Coutts). Osteophytic lesions of the skull are shown by symmetrical spots of thickening upon the parietal and frontal bones (*natiform skulls*). In the long bones osteophytes are frequently formed. In some cases osteophytes grow from the epiphysis, and in consequence deformity and impaired function are noted and a certain amount

[1] Coutts, in "Brit. Med. Jour.," 1894, p. 1643.

of ankylosis may occur. This condition of osteophytic growth from an epiphysis was called by Fournier *arthropathie deformant.* A child with precocious hereditary syphilis is apt to die, but if it lives from six months to one year the symptoms for a time disappear, and for years the disease may be latent. Diagnosis is difficult after the third or fourth year, especially if the disease be associated with rickets or tuberculosis. When later symptoms arise they may be various, namely: noises in the ears, often followed by deafness; interstitial keratitis; synovitis in any joint, particularly painless but marked symmetrical effusion in the knee-joints, with trivial functional disturbance; ossifying nodes; developmental osseous defects; suppurative periostitis; ulcerations; death of bone; falling in of the nose; nervous maladies; occasionally sarcocele, dactylitis, etc.

Fig. 138.—Dactylitis.

**Dactylitis** (Fig. 138) is common in hereditary syphilis. There is a superficial and a deep form (see p. 330). Commonly, a swelling gradually forms. It is fusiform in shape and becomes purple in color. It lasts for months. One or more fingers may be involved and the fingers are more apt to suffer than the toes. The proximal phalanx is most liable to the lesion. The superficial form is apt to soften and ulcerate. Sinuses seldom form. The deep form not infrequently causes tissue destruction and shortening of the digit from rarefying osteitis or dry caries. Some cases of superficial dactylitis are symmetrical and of short duration and are to be regarded as late secondary lesions.

In hereditary syphilis the eye symptoms are of great diagnostic importance. In 212 cases of congenital syphilis Fournier found eye trouble in 101. Keratitis and choroiditis are the most usual forms. Bone trouble occurs in almost half of the cases, but is not often severe enough to cause symptoms. The tongue often shows a smooth base (Virchow's sign). It is due to lymphoid atrophy and fibrosis (Symmers, in "Amer. Jour. Med. Sci., Dec., 1910). Hirschberg believed choroiditis to be pathognomonic. The descendants of syphilitic parents may exhibit certain pathological conditions which are not directly syphilitic. Fournier calls such phenomena parasyphilitic. Among these phenomena are arrest of development of the body at large or of special structures, weakness of constitution, and other stigmata of degeneration.

Fig. 139.—Hutchinson teeth.

In the **diagnosis** of hereditary syphilis the condition of the teeth is of considerable importance: the temporary teeth decay soon, but present no characteristic defect. If the upper permanent central incisors are examined, they are often, but by no means always, found defective. Other teeth may show defects, but in these alone are characteristic defects likely to appear. In hereditary syphilis they may present an appearance of marked deviation from health, and are then called *Hutchinson teeth* (Fig. 139). Hutchinson stated that if they are dwarfed, too short and too narrow, and if they display a single central cleft in their free edge, then the diagnosis of syphilis is probable. If the cleft is present and the dwarfing absent, or if the peculiar form of dwarfing be

present without any conspicuous cleft, the diagnosis may still be made. The view that teeth of this nature *prove* the existence of hereditary syphilis and that they occur only in syphilis has been abandoned by Hutchinson himself. In fact, only one-fifth of congenital syphilitics have these teeth, and one-third of the cases of Hutchinson teeth are in individuals free from syphilis. In early infancy the diagnosis of syphilis is made by the snuffles, the broad nose, the skin eruptions, the wasted appearance, the sores at the mouth-angles, the tenderness over bones, condylomata, and the history of the parents. The diagnosis at a later period is made by the existence of symmetrical interstitial keratitis, choroiditis, the smooth base of the tongue, deafness, which comes on without pain or running from the ear, ossifying nodes, white radiating scars about the mouth-angles, sunken nose, natiform skull, deformity of long bones, painless inflammation of epiphyses, and Hutchinson teeth. It must be remembered that a child born apparently healthy and presenting no secondary symptoms may show bone disease, keratitis, or syphilitic deafness at puberty. Finding the spirochetes is of immense importance in arriving at a diagnosis. They can always be found unless the organ examined is decomposed or the fetus is macerated. (See article by Wm. S. Gottheil, "Progressive Medicine," Sept., 1908.)

**Treatment.**—In infants mercurial inunctions are to be used until the symptoms disappear, but mercury must not be forced or be continued too long after the symptoms are gone. There must be rubbed into the sole of each foot or the palm of each hand 5 gr. of mercurial ointment every morning and night. Brodie advised spreading the ointment (in the strength of 1 dr. to the ounce) upon flannel and fastening it around the child's belly. If the skin is so tender that mercury must be administered by the mouth, order that $\frac{1}{12}$ to $\frac{1}{3}$ gr. of mercury with chalk, with 1 gr. of sugar, be taken three times a day after nursing. If tertiary symptoms appear, and in any case when the secondaries have passed away, give $\frac{1}{2}$ to 1 gr. or more of iodid of potassium several times a day in syrup. The mixed treatment should be continued intermittently until puberty. Local lesions require local treatment, as in the adult. A syphilitic child should, if possible, be nursed by its mother, as it will poison a healthy nurse, and also because the mother's milk contains antibodies. In some cases the mother is given salvarsan. If the baby has a sore mouth, it must be fed from a bottle, and if the mother cannot nurse the child, it must be brought up on the bottle. For the cachexia use cod-liver oil, iodid of iron, arsenic, and the phosphates. Salvarsan is not given to a syphilitic child under three years of age. It may be given to children over three and under seven and only by intramuscular injection. To a child over seven it may be given intravenously. The dose for a child of seven is 0.2 gm.

## XVIII. TUMORS OR MORBID GROWTHS

**Division.**—Morbid growths are divided into (1) neoplasms and (2) cysts.

**Neoplasms.**—A neoplasm is a pathological new growth which tends to persist independently of the structures in which it lies, and which performs no physiological function. We say that a tumor performs no physiological function in order to make clear that it is never a useful addition to the economy, but we must not imagine that the cells of every tumor are devoid of physiological activity. As Fütterer ("Medicine," March, 1902) has shown, the cells of a carcinoma of the liver may secrete bile, and even the cells of a secondary focus developing in the course of hepatic carcinoma may also secrete bile. The cells of a tumor may be active, but this activity is not useful and does not con-

stitute physiological function. A hypertrophy is differentiated from a tumor by the facts that it is a result of increased physiological demands or of local nutritive changes, and that it tends to subside after the withdrawal of the exciting stimulus. Further, a hypertrophy does not destroy the natural contour of a part, while a tumor does. Inflammation has marked symptoms: its swelling does not tend to persist, it terminates in resolution, organization, or suppuration, and examination of a section of tissue under the microscope differentiates it from tumor. Inflammation, too, has an assignable exciting cause. A new growth is a mass of newly formed *tissue;* hence it is improper to designate as tumors those swellings due to extravasation of blood (as in hematocele), or of urine (as in ruptured urethra), to displacement of parts (as in hernia, floating kidney, or dislocation of the liver), or to fluid distention of a natural cavity (as in hydrocele or bursitis).

**Classes of Tumors.**—There are two classes of tumors: the first class includes those derived from or composed of ordinary connective tissue or of higher structures. These all originate from cells which are developed from the mesoblast. There are two groups of connective-tissue tumors: (*a*) the typical, innocent, or benign, which mimic or imitate some connective tissue of the healthy adult human body; and (*b*) the atypical or malignant, which find no counterpart in the healthy adult human body, but rather in the immature connective tissues of the embryo.

The second class of tumors includes those which are derived from or composed of epithelium: (*a*) the typical, or innocent, composed of adult epithelium; and (*b*) the atypical, or malignant, composed of embryonic epithelium.

**Müller's law** is that the constituent elements of neoplasms always have their types, counterparts, or close imitations in the tissues, either embryonic or mature, of the human body.

**Virchow's law** is that the cells of a tumor spring from pre-existing cells. There is no special tumor-cell or cancer-cell.

The starting-point of a tumor is a focus of embryonal cells, which focus may have originated before the person was born or may have resulted after birth from some disease or injury. The nature of the tumor depends, first, upon the embryonal layer from which it took origin. Connective-tissue tumors spring from the mesoblast; epithelial tumors spring from the epiblast or the hypoblast. The nature of the tumor depends also upon the stage in which the growth of its cells is arrested. If the cells remain embryonal, the growth is regarded as malignant; if they become fully developed, it is regarded as innocent.

The term "heterologous" is no longer used to signify that the cellular elements of a tumor have no counterpart in the healthy organism, but is employed to signify that a tumor deviates from the type of the structure from which it takes its origin (as a chondroma arising from the parotid gland). Tumors when once formed almost invariably increase and persist, though occasionally warts, exostoses, and fatty tumors disappear spontaneously. Tumors may ulcerate, inflame, slough, be infiltrated with blood, or undergo mucoid, calcareous, or fatty degeneration.

The **causes** of tumors are not positively recognized, those alleged being but theories varying in probability and ingenuity.

The *inclusion theory of Cohnheim* supposes that more embryonic cells exist than are needful to construct the fetal tissues, that masses of them remain in the tissues, and that these embryonic cells may, later in life, be stimulated into active growth perhaps by injury or irritations or hereditary tendency. In other words, Cohnheim believes that all tumors arise from embryonal cells which were included or imprisoned by adult cells during fetal life and were not used during development; or from cells which were "displaced from their proper relations during the process of cell differentiation in the embryo"

(Henry Morris, "Lancet," Dec. 12, 1903). The embryonic hypothesis seems to receive a certain force from the facts that exostoses do sometimes develop from portions of unossified epiphyseal cartilage, and that tumors often arise in regions where there was a suppression of a fetal part, closure of a cleft, or an involution of epithelium (epithelioma is usual at mucocutaneous junctions). This theory does not explain the origin of malignant tumors in scars or recent callus in parts subjected to injury or operation, etc. (Ibid.).

Durante's addition to Cohnheim's theory does explain them. Cohnheim taught that the matrix from which a tumor springs is always an antenatal embryonic area. Durante says a tumor may also spring from a postnatal embryonic area resulting from injury of the mature tissues, lessening their chemical and physiological activities (Morris) and causing them to revert to an embryonic condition.

Objection has been made to the Cohnheim theory on the ground that an embryonal matrix could not remain quiescent, but, as Henry Morris says, certain teeth, the female mammary gland, the larynx, and certain appendages of the skin may not develop until puberty ("Bradshaw Lecture," in "Lancet," Dec. 12, 1903). Branchial cysts which are known to have such an origin are seldom seen until after puberty, and the same is true of many dermoids.

Morris shows that congenital matrices have been shown to exist in the brain, tongue, eye, testicle, ovary, broad ligament, line of coalescence in the trunk, and other places, and such matrices constitute *fetal rests* or *vestiges*. The same author shows that postnatal matrices may arise in the healing of a wound or ulcer, fistula, burns, etc. Portions of epithelium are separated, get placed deeply in the newly forming tissue, become surrounded by connective tissue, and may later take on active growth. As Ribbert points out, any fragment of isolated and imprisoned tissue may become a tumor.

*Heredity* is an extremely uncertain influence, though not an influence to be denied. I believe that there is such a thing as a more or less complete immunity to cancer and that there is such a thing as a predisposition to cancer, and the predisposition as well as the immunity may be hereditary or acquired. Youth constitutes an almost though not quite certain immunity. Cancer is very rare in youth, and when it does occur in a young person it is always very malignant. Its occurrence means unnatural lack of tissue resistance or unusual vigor of cancer-cell. The retrogressive changes of age are predisposing causes. S. W. Gross found direct hereditary influence by no means frequent in cancer of the breast. From 25 to 37 per cent. of cases of cancer of the breast have or had cancerous relatives (see page 387). Heredity in cancer is affirmed by some, denied by others, and doubted by a number. At most, hereditary influence may only predispose. Nevertheless, cases have occurred which cannot be explained by the term "coincidence." In the celebrated "Middlesex Hospital case," a woman and five daughters had cancer of the left breast. A. Pearce Gould had charge of a woman for cancer of the left breast. The mother of this patient, the mother's two sisters, and two of the mother's cousins had died of cancer. Power reports a remarkable instance of family predisposition to cancer. A patient had his right breast removed for cancer in 1896. In 1897 cancerous glands were removed from the axilla. In 1898 he was seen again with an irremovable recurrent growth. His father died of cancer of the breast. He had two brothers, one of whom died of cancer of the throat when sixty-five years of age, the other having died of cancer of the axilla when he was only twenty-four years old. Of his eight sisters, four died of cancer of the breast, and the two who are living both suffer from cancer of the breast. One sister died when an infant and one died after giving birth to a child.[1] The Emperor Napoleon, his father, his brother Lucien, and his sisters Pauline and Caroline

[1] "Brit. Med. Jour.," July 16, 1898.

died of gastric cancer. That there is such a thing as predisposition is rendered probable by the fact that out of many exposed under like conditions a single one may develop cancer. I believe, with Murphy, that there may be such a thing as absence of resistance to the cause of cancer on the part of certain tissues and that such impairment of resistance may be hereditary.

*Injury* and *inflammation* may undoubtedly prove exciting causes. A blow is not infrequently followed by sarcoma; the irritation of a hot pipe-stem may excite cancer of the lip; the scratching of a jagged tooth may cause cancer of the tongue; chimney-sweeps' cancer (which used to be seen in the old days when "the sweep" was an institution) arose from the irritation of dirt in the scrotal creases; and warts often arise from constant contact with acrid materials.

*Physiological activity* favors the development of sarcoma, and *physiological decline* favors the development of carcinoma.

*Parasitic Influences.*—Many believe that parasites cause cancer. This theory does not maintain that the tumor is the parasite, but that it contains the parasite, although Pfeiffer and Adamciewicz did at one time assert that a cancer-cell is not a body-cell, but a parasite resembling an epithelial cell. Butlin in 1905 asserted his belief that the cancer-cells are parasites and act independently like protozoa. Most observers deny this contention because, were it true, there would be only one variety of cancer, because cases could only arise by direct contact, and because it would leave unanswered how the original growth arose, as it could not have come from a pre-existing cancer-cell (Brand, in "Lancet," Jan. 11, 1908). Some facts render a parasitic origin of malignant growths not improbable; as, for instance, the likeness of some tumors to infective granulomata, the tendency to secondary development in distant parts of the body, the resemblance of the secondary to the primary growths, and the tenacity of their persistence. A parasitic origin of cancer is possibly suggested by its geographical distribution, the disease being very common in low and marshy districts, and Haviland ("Lancet," April 27, 1894) and others maintain that certain houses become infected, the disease appearing in these houses among successive families inhabiting them. They speak of such abodes as *cancer-houses*.

Some surgeons believe that cancer is contagious, but most observers deny it. Hanau found a rat with a cancer and inoculated other rats from it. Moreau in 1894 inoculated mice from a mouse with cancer. Guelliott, of Rheims, believes that cancer is primarily a local infection. He believes this because Moreau and Hanau have inoculated it from one animal to another of the same species, and if this can be brought about experimentally he sees no reason why it cannot happen accidentally. This surgeon says that cancer is very unequally distributed, that genuine cancer-centers and "cancer-houses" exist, and that numerous cases of accidental infection have occurred.[1] Hahn apparently succeeded in grafting cancer from one part to another on the same individual. Jensen and Borrell have inoculated the disease in white mice. Mayet, of Lyons, holds that cancer can be reproduced by grafting or by injection of cancer-fluid. Graf could not find "cancer-houses" after a careful search.[2] Because several people, in the course of years, have died in the same house of cancer is not proof that the house was infected. If such a thing proves contagion there must be contagion in many things now thought free from it, and there must be broken-leg houses, and delirium-tremens houses, and heart-disease houses. Geissler claims to have produced the disease in a dog by planting fragments of cancer in the subcutaneous tissue and vaginal tissue, but Czerny, Rosenbach, and others dispute the claim. Plimmer tells

[1] "Amer. Jour. of Med. Sciences," June, 1895.
[2] "Archiv. f. klin. Chir.," 1895, l, p. 144.

us that an epidemic of cancer arose among the captive white rats in the Freiburg Pathological Institute, and in each case the growth was on the rear part of the body. Roswell Park believes that Gaylord has really produced adenocarcinoma in the lower animals. Hauser disputes the assertion that cancer must be an infectious disease because it is followed by secondary growths. Secondary growths in an infectous disease are caused by the bacterium; secondary growths in cancer are caused by the transference of cells of primary growth.[1] Hauser says with truth that the close connection between innocent and malignant growths renders the parasite view untenable, because to hold it we would be forced to believe that every tumor has a special parasite or that one parasite may cause many kinds of tumors.

There seems to be no doubt that autotransference of cancer can occur, although it rarely does so. Sippel has reported a case in which vaginal carcinoma developed at the point where the vagina was in contact with a preexisting cancer of the portio.[2] Cornil has seen cancer transferred from one of the labia majora to the other, and from one lip to the other. Geissler was unable to transplant cancer, and Gratia also failed in his attempts. Duplay and Bazin say that transmissibility is possible, but only under conditions which are not practically realized. The facts that transplantation can be sometimes carried out, and that contagion is a possible occurrence under exceptional circumstances, do not prove that cancer is a parasitic disease, but simply prove that it can be transplanted. It is not that the cancer carries a parasite which will cause the disease in sound tissues, but rather that the cells of the cancer may themselves take root and grow in sound tissues. The parasitic theory arose from observation of the metastasis which occurs during the progress of the disease, and received support from the fact that inoculation of another part of an individual suffering from cancer may be followed by the development of a tumor like the original growth. For instance, if a cancer is growing upon the lower lip, the upper lip may be inoculated (*contact cancer*). The same is true of the labia. Mr. Harrison Cripps reported the occurrence of cancer of the skin of the arm from contact with an ulcerating scirrhus of the breast. It has also been pointed out that carcinoma is especially common in regions predisposed by their situation to injury and infection, and that, "among the lower animals at least, tumors resembling carcinomata have been transplanted from one to another" ("Recent Studies upon the Etiology of Carcinoma," by Joseph Sailer, "Phila. Med. Jour.," June 7, 1902). But there is great doubt as to the cancerous nature of some of the tumors which have been successfully transplanted from one animal to another.

A transplanted mouse cancer may grow for a time and then completely disappear, and some observers (among them Gaylord) hold that when this occurs the mouse has become immune.

In 1908 the German Pathological Society met in Kiel and discussed various problems of cancer. In this meeting Sticker maintained that there is such a thing as natural immunity to tumors. He showed that a tumor arising spontaneously in an animal can never be transplanted into an animal of another species, and very seldom can a malignant tumor be transplanted into an animal of the same species. He quotes Metchnikoff's utter failure to transplant human tumors into anthropoid apes and reports his own failure to transplant human tumors into various domestic animals. He made over 400 trials and failed every time ("Jour. Am. Med. Assoc.," from Sticker, "Zeitschr. f. Krebsforschung," 1908, vii).

In successful transplantations there has been but slight effort to prove that epithelial cells were not transferred with the supposed parasites, and if they

[1] Hauser, in "Biolog. Centralbl.," Oct. 2, 1895.
[2] "Centralbl. f. Gynak.," No. 4, 1894.

were transferred the success of the experiment does not prove that cancer is due to parasites, but simply proves again what we knew before—that epithelial cells can be transplanted. Many parasites have been regarded as causative by different observers. Bacteria, yeast-cells, and protozoa have been found by different experimenters. It is not thought that bacteria are causative. Yeasts are regarded as causative by some. It is certain that they may exist in cancer, but it is by no means certain that they cause the disease. They may be only a contamination. Gaylord and others regard protozoa as causative, but this statement does not seem to be proved. Many of the supposed parasites of cancer have been shown to be cell degenerations or contaminations. We are justified in concluding that the parasitic origin is not as yet proved, and we agree with the elder Senn that it is improbable.

Tillmanns elaborately discussed the subject of cancer in the Congress of 1895. His conclusions are still most sound and scientific. He says there is no evidence of a bacterial origin of cancer. The parasitic origin has not been proved, and protozoa have not certainly been found. Cancer can be transferred from one part to another of the same individual, or from one individual to another of the same species, but never to one of a different species. It is possible that cancer can spread by contagion; this is very rare, but can happen (as when penile cancer is followed by cervix cancer in a wife). Because it is sometimes possible to transfer cancer, this does not prove that the disease is parasitic or infectious; it simply shows that *tissue* has been successfully transplanted.

*Cancer à deux* is cancer developing in people who live together. Such cases suggest, but do not prove, contagion. Behla collected 19 cases and Guelliot 103 cases. *Conjugal cancer* is classified as cancer à deux. A wife, for instance, may have cancer of the womb and a husband may develop cancer of the penis, supposedly from contact. Conjugal cancer is probably due to irritation or implantation and not to microbic inoculation.

Actinomycosis, long thought to be a true tumor, is now known to arise from the ray-fungus. Some think that psorosperms cause cancer. There can be no doubt that changes in the liver which practically constitute a new growth can arise from the growth of a cell called by Darier the "psorosperm." A disease due to psorosperms is called a "psorospermosis." It is affirmed by some that molluscum contagiosum, follicular keratosis, cancer, and Paget's disease are due to psorosperms. Some claim to find the parasite in all cases of cancer, while others can find it in only 4 or 5 per cent. of the cases.

Heneage Gibbes affirms[1] that dilatation of the bile-ducts of a rabbit's liver is caused by the chronic irritation arising from multiplication of the Coccidium oviforme in them, and not in the columnar cells of the bile-ducts, as has been stated; and, further, that the large majority of glandular cancers show nothing that can be considered parasitic, the suspicious appearances noted in some few cases being due to endogenous cell formation. The Coccidium oviforme is a genus of the sporozoa, class protozoa, the lowest division of the animal kingdom. To this case belong the monera and unfusoria. (For a further discussion of this subject see page 55.)

**Malignant and Innocent Tumors.**—Malignant growths infiltrate the tissues as they grow; benign tumors only push the tissues away; hence malignant tumors are not thoroughly encapsuled, while innocent tumors are encapsuled. Malignant tumors grow rapidly; innocent tumors grow slowly. Malignant tumors become adherent to the skin and cause ulceration; innocent tumors rarely adhere and seldom cause ulceration. Many malignant tumors give rise to secondary growths in adjacent lymphatic glands (cancer, except in the esophagus and antrum of Highmore, always does so; sarcoma rarely

[1] "Amer. Jour. of Med. Sciences," July, 1893.

causes them, unless the growth be melanotic or lymphosarcoma, or unless it arises in the testicle or tonsil). Innocent tumors never cause secondary lymphatic involvement; although the glands near the tumor may enlarge from accidental inflammatory complications. The malignant tumors, especially certain sarcomata and soft cancers, may be followed by secondary growths (metastases) in distant parts and various structures (bones, viscera, brain, muscles, etc.); innocent tumors are not followed by these secondary reproductions, although multiple fatty tumors or multiple lymphomata may exist. Malignant tumors destroy the general health, inducing anemia and cachexia; innocent tumors do not, unless by the accident of position. Malignant tumors tend to recur after removal; innocent tumors do not if operation was thorough. The special histological feature of a malignant growth is the possession by its cells of a power of reproduction which knows no limit, the cells of the tumor living among the body-cells like a parasite and invading and destroying the body-cells.

**The Cachexia of Malignant Disease.**—This condition arises sooner or later in every uncured case of sarcoma and carcinoma.

In sarcoma there is advancing anemia and there are often episodes of elevated temperature due to the absorption of toxic materials from the tumor. The blood examination gives results similar to those found in cancer, but leukocytosis is more frequent. Pain is far less prominent than in cancer unless a nerve is involved or squeezed. Ulceration occurs much later in sarcoma than in carcinoma.

In carcinoma (see page 382) the emaciation is rapid and decided, the loss of strength is significant and notable, and the anemia is marked and progressive. It is due to pain, sleeplessness, ulceration, impaired appetite and digestion, repeated hemorrhages, and the absorption from the tumor of toxic products, which are probably enzymes (especially isohemolysins). Loss of hemoglobin is early and is followed by decrease in the number of red cells. In many cases considerable amounts of sugar exist in the blood. Irregular fever may occur. In both sarcoma and carcinoma the development of secondary growths aggravates the anemia.

**Classification.**—Tumors may be classified as follows:

I. Connective-tissue tumors (those derived from the mesoblast).

1. Innocent tumors, or those composed of mature connective tissue:
   *Lipomata*, or fatty tumors; *fibromata*, or fibrous tumors; *chondromata*, or cartilaginous tumors; *osteomata*, or bony tumors; *odontomata*, or tooth-tumors; *myxomata*, or mucous tumors; *myomata*, or muscle-tumors; *neuromata*, or tumors upon nerves; *gliomata*, or tumors composed of neuroglia; *angiomata*, or tumors formed of blood-vessels; *lymphangiomata*, or tumors formed of lymphatic vessels. The term *lymphoma*, meaning a tumor of a lymphatic gland, was formerly applied to hypertrophy and hyperplasia of a lymphatic gland, no matter whether caused by syphilis, tubercle, Hodgkin's disease, or any other morbid impression. The term has been largely abandoned except as expressing enlargement of a gland, and does not convey any suggestion as to the cause. It is doubtful if there is such a thing as a true lymphoma, understanding by the term a neoplasm arising from and composed of lymphoid cells and resembling lymphatic structure. In the described cases the possibility of infection as a cause has not been eliminated.
2. Malignant tumors, or those composed of embryonic connective tissue:
   *Sarcomata* and adrenal tumors.
   *Endotheliomata* are regarded by some as constituting an independent group and by others as a variety of sarcomata.

II. Epithelial tumors (those derived from the epiblast or hypoblast).
   1. Innocent tumors, or those composed of mature epithelial tissue: *Adenomata*, or tumors whose type is a secreting gland; and *papillomata*, or tumors whose type is found in the papillæ of skin and mucous membranes.
   2. Malignant tumors, or those composed of embryonic epithelial tissue: *Carcinomata*, or cancers.

III. Cystomata are cystic tumors, the cyst-walls of which are new growths and the contents of which are produced by the cells of the newly formed cyst walls.

IV. Teratomata (tumors containing epiblastic, hypoblastic, and mesoblastic elements).

**Innocent Connective-tissue Tumors.**—These growths mimic or imitate some connective tissue or higher tissue of the mature and healthy organism.

**Lipomata** are congenital or acquired tumors composed of fat contained in the cells of connective tissue, which cells are bound together by fibers. If

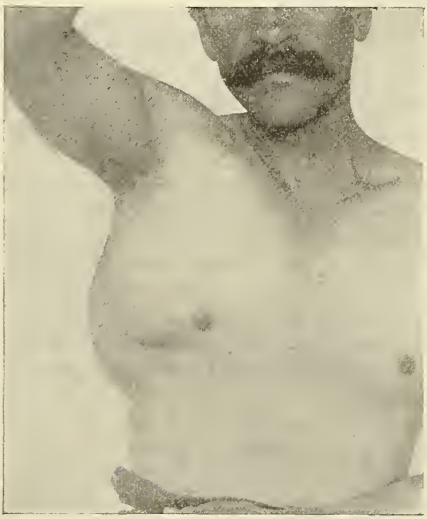


Fig. 140.—Lipoma, wandered from axilla.

Fig. 141.—Diffuse lipoma.

the fibers are excessively abundant, the growth is spoken of as a *fibrofatty tumor*. A fatty tumor has a distinct capsule, tightly adherent to surrounding parts, but loosely attached to the tumor; hence enucleation is easy. Fibrous trabeculæ run from the capsule of a subcutaneous lipoma to the skin; hence movement of the integument over the tumor or of the tumor itself causes dimpling of the skin. An ordinary circumscribed lipoma is of doughy softness, is lobulated, of uniform consistence, and on being tapped imparts to the finger a tremor known as pseudofluctuation. A fatty tumor is mobile, although it may be attached to the skin at points by trabeculæ. Sometimes a fatty tumor gradually shifts its position or wanders (Fig. 140). This is due to gravity. Lipomata are most frequent in middle life, and their commonest situations are in the subcutaneous tissues, especially of the back or of the dorsal surfaces of the limbs; they usually occur singly, but may be multiple and sometimes symmetrical. Senn described the case of a woman who had a fatty tumor in each axilla. A lipoma may grow to an enormous size (in Rhodius's case the tumor weighed 60 pounds), and the growth may be progressive or may be at times stationary and at other times active. The skin over a fatty tumor

sometimes atrophies or even ulcerates; the tumor itself may inflame or partly calcify. When a lipoma has once inflamed, it becomes immovable. Subcutaneous lipoma of the palm of the hand or sole of the foot bears some resemblance clinically to a compound ganglion; it is apt to be congenital. Lipomata of the head and face are rare. In the subcutaneous tissues of the groins, neck, pubes, axillæ, or scrotum a mass of fat may form, unlimited by a capsule and known as a *diffuse lipoma* (Figs. 141 and 143). A diffuse lipoma may dip down among the muscles. Such masses attain large size. The typical diffuse lipoma is occasionally seen on the neck. It begins back of the mastoid process on one side or on both sides. When large, it completely surrounds the neck, a huge double chin forming in front, a great mass hanging on each side, and the posterior portion being divided into two halves by a median depression. A *nevolipoma* is a nevus with much fibrofatty tissue. A very vascular fatty tumor is called *lipoma telangiectodes*. If the tumor stroma contains large veins

Fig. 142.—Lipoma of submaxillary region.

Fig. 143.—Congenital diffuse lipoma of foot and leg. Child seven years of age, second and third toes amputated at thirteen months and large mass moved from sole of foot. Later a large fatty mass moved from calf of leg, surrounding gastrocnemius and soleus muscles and between them (Rugh).

the growth is called a *cavernous lipoma*. A tumor containing much blood can be diminished in size by pressure. Fatty tumors may arise in the subserous tissue, and when such a growth arises in either the femoral or inguinal canal or the linea alba it resembles an omental hernia and is spoken of as a *fat-hernia*. In the retroperitoneal tissues enormous fibrofatty tumors occasionally grow, and these neoplasms tend to become sarcomatous. Lipomata which arise from beneath synovial membranes project into the joints, being still covered by synovial membrane. Fatty tumors occasionally arise in submucous tissues, between or in muscles, from periosteum, and from the meninges of the spinal cord (Sir J. Bland-Sutton). A fatty tumor may undergo metamorphosis. The stroma may be attacked by a myxomatous process or a calcareous degeneration. The fat-cells themselves may become calcareous. Oil-cysts sometimes form. A *xanthoma* is a growth composed of fatty tissue in and about which

there is marked infiltration with small cells. Such a tumor is flattened and slightly elevated. Several or many of these growths occur in the same person. The eyelids are the most common seat of xanthoma. The tumor may undergo involution or may become sarcomatous.

Diabetics are liable to develop xanthomata.

**Treatment.**—A single subcutaneous lipoma should be extirpated. The capsule must be incised, when the tumor is torn out forcibly or, better, is enucleated by dissection; drainage is always employed for twenty-four hours, as butyric fermentation will be apt to occur, and necrosis of small particles of fat predisposes to infection. Multiple subcutaneous lipomata, if very numerous, should not be interfered with unless troublesome because of their size or situation, when the growth or growths causing trouble should be removed. It is difficult to extirpate entire a diffuse lipoma, and several operations may be needed to effect complete removal. Liquor potassæ, once recommended as possessing power, when taken internally, to limit the growth of multiple lipomata or diffuse lipoma, seems to be useless. Subperitoneal

Fig. 144.—Fatty tumor.

lipomata are rarely diagnosticated until the belly has been opened, sometimes not until the growth has been removed.

**Fibromata** are tumors composed of bundles of fibrous tissue. There are two forms, the hard and the soft. A *hard fibroma* consists of wavy fibrous bundles lying in close contact. Here and there connective-tissue corpuscles exist between the fibers. A fibroma has no distinct capsule, though surrounding tissues are so compressed as to simulate a capsule. Fibromata are occasionally congenital, are most usual in young adults, but they may occur at any period of life, and in any part of the body containing connective tissue. Pure fibromata, which are rare, are generally solitary, grow slowly, are of uniform consistence, have not much circulation, and are hard and movable. Fibromata may form upon nerve-sheaths, may arise in the mammary gland, may develop in the lobe of the ear, and may spring from various fibrous membranes, from the periosteum of the base of the skull (*nasopharyngeal fibromata*), and from the gums (*fibrous epulides*). A *soft fibroma* contains much areolar tissue, the spaces of which are filled with fluid, so that the tissue seems

edematous. Soft fibromata grow from the skin, mucous membrane, subcutaneous tissue, intermuscular planes, and periosteum. Soft fibromata possess distinct pedicles and are especially apt to arise from the skin of the scrotum, labia, inner surface of arm and thigh, and from the belly wall of a pregnant woman. They are not unusually multiple, grow slowly, but more rapidly than the hard fibromata, and may become quite large. Fibromata may become cystic, calcareous, osseous, colloidal, or sarcomatous, and may inflame, ulcerate, or even become gangrenous.

A *painful subcutaneous tubercle*, which is a form of fibroma commonest in females, arises in the subcutaneous cellular tissue, usually of the extremities. It is firm, very tender, movable, rarely larger than a pea, and the skin over it seems healthy. Violent pain occurs in paroxysms and radiates over a considerable area, of which the tubercle is the center. These paroxysms may occur only once in many days or many times in one day. Pain is always developed by pressure, and may be linked with spasm. Nerve-fibrillæ are now known to exist in these tubercles, a fact which was long denied.

A *mole* is a fibroma of the skin which is congenital or appears in the early weeks of life. It is rounded or flat, is usually pigmented and of a brown color, is slightly elevated above the cutaneous level, and has a few hairs or an abundant crop of hair growing from it, and varies in size from a pin's head to several inches in diameter, or may even occupy an extensive area of a limb or of the trunk. The tumor rarely grows after the thirteenth or fourteenth year. A mole may become malignant, melanotic carcinoma may arise from its epithelial structures, or melanotic sarcoma from its connective-tissue elements. A mole is an extremely vascular structure; it bleeds freely when cut or scratched, and it sometimes ulcerates. Occasionally several or many moles exist in the same individual. If a mole begins to increase rapily in size, operation is imperative, as rapid growth probably indicates malignant change.

*Fibrous epulis* is a fibroma arising from the gums or periodontal membrane (Sir J. Bland-Sutton) in connection with a carious tooth or retained snag; it is covered by mucous membrane, grows slowly, may attain a large size, and sometimes has a stem, but is more often sessile. It may undergo myxomatous change or may become sarcomatous.

Fibrous tumors may arise from the ovary, the intestine, the larynx, and the submucous tissues of the gastro-intestinal tract. Pure fibromata of the uterus are very rare, but fibromyomata are very common (see Myomata, page 361); hence the term "uterine fibroid" should be abandoned.

*Desmoid tumors* of the abdominal wall are cellular fibromata. A desmoid tumor has a strong disposition to become sarcoma. It has no real capsule. It takes origin from one of the abdominal muscles or muscular insertions or from fascia, particularly from the rectus muscle or its sheath. In most cases the growth is slow; in some it is rapid and the tumor may attain a great size. It may project either anteriorly or toward the abdomen. This form of tumor is vastly more common in women than in men and is especially common in women who have borne children. It may occur at any age, but is most frequent in those between twenty-five and thirty-five. It is a very rare tumor. (See Harvey B. Stone, "Annals of Surgery," August, 1908.)

*Molluscum fibrosum* is an overgrowth of the fibrous tissue of both the skin and subcutaneous structure. Senn excludes this form of growth from consideration with fibromata because of its supposed infective origin. It may be limited or widely extended; it may appear as an infinite number of nodules scattered over the entire body or as hanging folds of fibrous tissue in certain areas.

*Keloid* (Figs. 145, 146) is a fibroma of the true skin. It is a hard and fibrous vascular growth, with a broad base, arising in scar-tissue; it is crossed by pink, white, or discolored ridges, and is named from a fancied likeness to

the crab. It has rarely attacked mucous membrane. It is more common in negroes than in whites, and is most frequent in the cicatrices of burns, though it may arise in the scar of any injury, as the scar from piercing the ears, and in the scars of syphilitic lesions, tuberculous processes, smallpox, or vaccination. I believe that the scars of tuberculous lesions and the scars even of ordinary wounds in tuberculous individuals are particularly apt to become keloidal. It is very common in a person with keloid to be able to find some near or distant tuberculous lesion, or a history of former tuberculosis, or the record of the individual having tuberculous tendencies. The victim of keloid usually reacts to tuberculin. The growth seldom begins in early childhood or in old age. It grows slowly, lasts for many years, and may eventually undergo involution and disappear. The fact that keloid is especially common in the negro race (a race predisposed to tuberculosis) and that it is so frequently met with in the scars of known tuberculous processes, suggests the possibility of a tuberculous cause for the condition. The rapid return of keloid after operation suggests a near or distant infection which furnishes material to a point of least resistance which causes keloid to redevelop. Some cases of keloid

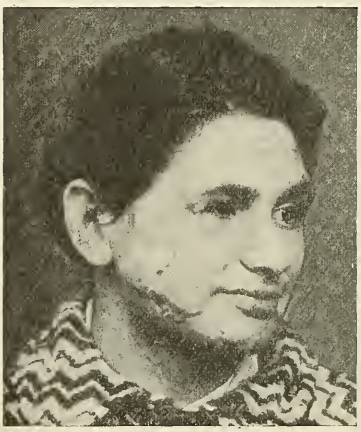


Fig. 145.—Keloid following a burn.

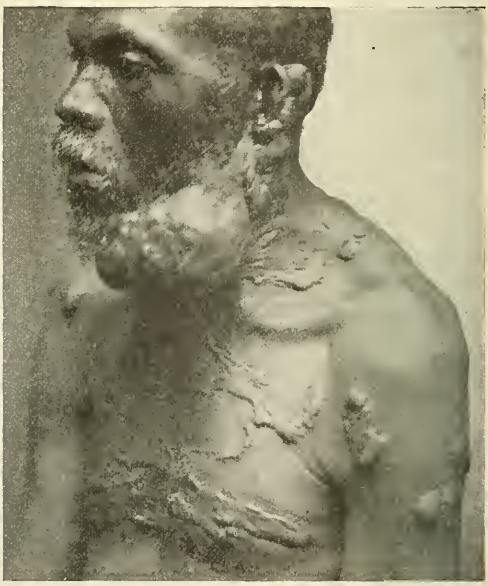


Fig. 146.—Keloid (case of Dr. L. L. Hill, Montgomery, Alabama).

have active tuberculous lesions, others have had them, in still others latent or distant lesions may be found by careful search. In many cases there is a family history of tuberculosis. I am at present investigating this important matter. It is certain that the keloid itself does not contain bacteria. Repeated examinations have failed to find them. It is quite possible that the growth contains toxins of tubercle bacilli, the toxins being the irritant cause. I am now seeking to determine if material from keloid introduced into tuberculous animals will cause a reaction. It is usually believed that it is almost useless to remove keloid by operation, as it will almost certainly return, yet a study of the growth removed shows no reason for the inevitable return. Charles A. Porter has reported a case of massive keloid of the face and hands notably benefited by many operations and skin-grafting. In this case, Porter says, "there has been a gradual but distinct abatement of the tendency to form keloid tissue" ("Annals of Surgery," July, 1909). The fibrous tissue of keloid

springs from the outer walls of the blood-vessels. The papillæ of the skin above the tumor are destroyed or replaced by fibrous tissue.

*Morphea*, spontaneous or true keloid, is a name used to designate a growth of this description which does not arise from a scar; but it seems certain that scar-tissue was present, though possibly in small amount from trivial injury.

Fibrous and papillomatous growths covered with endothelium may spring from any serous membrane. Such a growth of the choroid plexus calcifies early and constitutes a *psammoma* or brain-sand tumor. Such tumors are met with not only in the choroid plexus, but also in the conarium and the dura. All psammomata are not fibrous; some are gliomatous and some are endotheliomatous. A *cholesteatoma* is a fibrous growth covered with endothelium and containing layers of crystalline fat. It occurs especially in the pia mater, but may arise in either of the other membranes or even in the brain substance, and is called a *pearl tumor*.

**Treatment.**—Fibromata should not be let alone, because any fibrous tumor may become a sarcoma. When in accessible regions they should be enucleated. If a hard fibroma of the skin exists the skin is incised and the tumor is "shelled out. "A soft fibroma is removed by an incision carried around the base of its pedicle. A painful subcutaneous tubercle should be excised. If a mole shows the slightest disposition to enlarge, or if it is subjected to pressure or irritation, it should be removed, because if allowed to remain it might develop into a malignant growth. It is often desirable to remove a hairy or pigmented mole, not only because it may become malignant, but also because it is unsightly. A mole, if not too large, may be removed by a 30-second application of solid carbonic acid. This destroys hair and often causes pigment to disappear. Many moles are best treated by excision. Fibrous epulis requires the cutting away of the entire mass, the removal of the related snag or carious tooth, and sometimes the biting away of a portion of the alveolus with rongeur forceps. A nasopharyngeal fibrous polyp often contains sarcomatous elements or develops into a spindle-celled sarcoma. If the polyp is recent and has a pedicle it may be removed by the cautery loop. Most cases require extirpation, the surgeon cutting wide of the growth. In a severe case a part of the superior maxillary bone is removed by osteoplastic resection to permit of extirpation. Keloid should rarely be operated upon: it will almost certainly return, and will also develop in the stitch holes. Trust to time for involution, or use pressure with flexible collodion, by which method J. M. DaCosta cured a case following small-pox. It may be necessary to operate because of ulceration. If it is necessary to operate, remove the keloid and considerable adjacent tissue and fill the gap with Thiersch grafts. The administration of thyroid extract may be of benefit (a 5-gr. tablet three or four times a day). This drug must be given cautiously, as it may cause attacks characterized by fever, dyspnea, and rapid pulse. Thiosinamin hypodermatically has been used, it is claimed, with benefit. A 10 per cent. solution is made, and from 10 to 15 min. can be injected into the gluteal muscles every third day. I have seen two keloids cured by the use of the *x*-rays.

**Chondromata** (*enchondromata*) are tumors formed either of hyaline cartilage, of fibrocartilage, or of both. Chondromata are apt to arise from certain glands, the long bones, the pelvis, the rib cartilages, and the bones of the hands or feet, and often spring from unossified portions of epiphyseal cartilage. They may be single or multiple, and are most commonly met with in the young. They have distinct adherent capsules; they grow slowly, and if of osseous origin progressively hollow out the bones by pressure; they cause no pain; they impart a sensation of firmness to the touch, unless mucoid degeneration forms zones of softness or fluctuation; they are inelastic, smooth or nodular, immovable, and often ossify. A chondroma may grow to an enor-

mous size. A chondroma of the parotid gland or testicle practically always contains sarcomatous elements, and any chondroma may become a sarcoma. Chondromata are notably frequent in persons who had rickets in early life. *Ecchondroses*, which are "small local overgrowths of cartilage" (Sir J. Bland-Sutton), arise from articular cartilages, especially of the knee-joint, and from the cartilages of the larynx and nose. Loose or floating cartilages in the joints may be broken-off ecchondroses or portions of hyaline cartilage which are entirely loose or are held by a narrow stalk, and which arise by chondrification of villous processes of the synovial membrane; only one or vast numbers may exist; one joint may be involved or several; they may produce no symptoms, but usually produce from time to time violent pain and immobility by acting as a joint-wedge. An ecchondroma may arise within the medullary canal of a long bone, from foci of dormant cartilage, and may lead to the development of a *solitary cyst* of large size by softening of the tumor. The femur is the most usual site of solitary cyst. It begins very insidiously and progresses gradually. There are slight lameness, trivial pain, tenderness below the level of the trochanter, apparent shortening, and some bulging of bone. The bone may bend or at some spot may thin so that the cyst can be felt. Such a bone fractures from slight force, and after a fracture, when the effused blood and inflammatory exudate have been absorbed, a tumor can be distinctly detected. A solitary cyst of a long bone is apt to be regarded clinically as a sarcoma (Bergmann-Virchow).

Fig. 147.—Osteoma of femur.

**Treatment.** — Remove chondromata whenever possible, for, if allowed to remain undisturbed, they are apt to resent this hospitality by becoming sarcomatous. A chondroma of the testicle and of a salivary gland is sure to be sarcomatous. In an ordinary chrondroma incise the capsule and take away the growth, using chisels and gouges if necessary. Incomplete removal means inevitable recurrence. Amputation is very rarely demanded. In chondrosarcoma incision must be outside of the capsule. Loose bodies in the joints, if productive of much annoyance, are to be removed, the joint being opened with the strictest antiseptic care. Amputation is sometimes performed for a solitary cyst of a long bone, the surgeon having looked upon the growth as sarcomatous. If a correct diagnosis is arrived at, an attempt should be made to remove the cyst without amputation. Bergmann succeeded in extirpating such a mass from the femur.

**Osteomata** are tumors which are composed of osseous tissue. Sir J. Bland-Sutton says that osteomata are ossifying chondromata. Osteomata take origin from bone, cartilage, connective tissue, especially tissue near the bone, serous membrane, and certain glands and organs. Compact osteomata, which are identical in structure with the compact tissue of bone, arise from the frontal sinus, mastoid process, external auditory meatus, and other regions in those beyond middle life; they are small, smooth, round, densely hard, with small and occasionally cartilaginous bases.

Cancellous osteomata, which comprise the great majority of bone-tumors,

are similar in structure to cancellous bone. They spring from, and are crusted with, cartilage; they may have fibrous capsules, and are often movable when

Fig. 148.—Osteoma of humerus.

recent, but soon become fixed; they have broad bases, are angled, nodular, firm (but not so hard as are the compact osteomata), painless except when pressed, occur particularly at the ends of long bones (Figs. 147 and 148), may grow to large size, and are commonest in youth. Osteomata near joints become overlaid by bursæ, which in rare instances communicate with an adjacent joint.

Osteomata do not tend to become malignant and do not recur after removal. The term *exostosis* or *osteophyte* has been used as being synonymous with osteoma, but wrongly so, as an exostosis is an irregular, local, bony growth which does not tend to progress without limit, and which is, hence, not a tumor. A true exostosis is seen in the ossification of a tendon insertion (Fig. 149), in a limited growth from one of the maxillary bones, and in a local growth from the last phalanx of the big toe, which latter form of growth is known as a *subungual exostosis*. *Osteophytes of the retrocalcaneal bursa* occasionally form when this bursa is inflamed. Inflammation of this bursa is

Fig. 149.—Osteophyte of os calcis.

known as *Achillodynia*, or *Albert's disease*. The bony masses sometimes found in the brain, lungs, testicles, various glands, and tumors are not true osteomata. Osteophytes may arise in a joint in various forms of arthritis.

**Multiple exostoses** (Figs. 150, 151) are rare and depend upon some anomaly in and ossification of temporary cartilage. They arise during the period of active growth of bone. Volkmann and others assigned rickets as the cause. Virchow looked upon the condition as hereditary. Lippert traced the condition through four generations, but hereditary influence is not always evident. Tuberculosis has nothing to do with their development. One, several, or many of the growths may diminish in size or even disappear (Bruns, "Beiträge," xxxiv, 1902). In Oberndorf's case many tumors disappeared, but none after maturity ("New York Med. Jour.," March 5, 1910). This patient had syringomyelia and acromegalic symptoms, causing one to think of Charcot's statement that the gray matter of the cord contains the center for the nutrition of the bone. Exostoses within the cranium or spinal canal may cause symptoms from irritation or from pressure.

**Treatment.**—Osteomata which are non-productive of pain or trouble do not demand removal. If they produce pain by pressure, if they press upon important structures, if they cause annoying deformities, or if they grow rapidly, then remove them by means of chisels, gouges, or the surgical engine.

Fig. 151.—Multiple exostoses. Hundreds of them throughout the body.

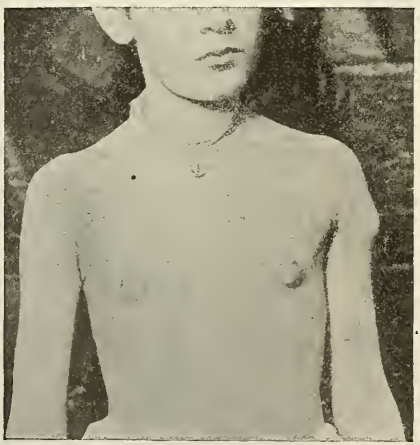


Fig. 150.—Multiple exostoses.

Subungual exostosis should always be removed. The nail should be split and part of it taken away, and the bony mass be gouged away or be cut off with forceps.

**Odontomata**[1] are tumors composed of tooth-tissue. They spring from the germs of teeth or from developing teeth. Sir J. Bland-Sutton divides them into (1) those springing from the follicle; (2) those springing from the papilla, and (3) those springing from the whole germ.

[1] This section is abridged from Sir. J. Bland-Sutton's striking chapter upon Odontomes in his work on "Tumors."

**Epithelial odontomes,** or **multilocular cystic tumors,** arise from the follicle, occur oftenest in the lower jaw, dilate the bone, have capsules, and are made up of masses of cysts which are filled with brown fluid. These cysts are met with most frequently before the age of twenty. *Follicular odontomes,* or *dentigerous cysts* (Fig. 152), oftenest spring from the follicles of the permanent molars. In a dentigerous cyst there exists an expanded follicle which distends the bone, the follicle being filled with thick fluid and containing a portion of a permanent tooth. When a follicular odontome is discovered after the time of second dentition the patient is short one or more permanent teeth. The corresponding milk teeth may be retained. The position of the portion of the tooth is variable. It is usually just beneath the orbit. In a case operated on in the Jefferson Clinic the tooth was in this situation. A *fibrous odontome* is due to thickening of the tooth-sac, which prevents eruption of the tooth; fibrous odontomes are usually multiple, and are apt to occur in rickety children. A *cementome* is due to enlargement, thickening, and ossification of the capsule, the developing tooth being encased in cement. A *compound follicular odontome* is due to ossi-

Fig. 152.—Dentigerous cysts removed from upper jaw of a negro. Both in the right side. Contained portions of undeveloped teeth.

fication of portions only of an enlarged and thickened capsule, and the tumor contains bits of cementum, portions of dentin, or small misshaped teeth. A *radicular odontome* springs from the papilla and arises after the crown of the tooth is formed and while the roots are forming; hence it contains dentin and cement, but no enamel. *Composite odontomes* are formed of irregular, shapeless masses of dentin, cement, and enamel. All the above forms occur in man. They present themselves as hard tumors associated with teeth or in an area where teeth have not erupted. Occasionally an odontome simulates necrosis; it is surrounded by pus, and a sinus forms.

**Treatment.**—The diagnosis is now usually possible by the aid of the *x*-rays. Be in no haste to excise large portions of bone for a doubtful growth; incise first and see if it be an odontome, which usually requires only the removal of an implicated tooth, curetting with a sharp spoon, and packing with iodoform gauze.

**Myxomata** are tumors composed of mucous tissue. They are rare as independent growths, although myxomatous change is frequent in the stroma of other tumors. The tissue type of these tumors is found in the vitreous

humor of the eye and in the perivascular tissues of the umbilical cord (Wharton's jelly). Bowlby states that myxomata are, in reality, soft fibromata whose intercellular substance has been replaced by mucin. The myxomatous state may be a stage in the formation of a fibroma, a stroma not having developed. Myxomata may result from myxomatous degeneration of cartilage, of muscle, or of fibrous tissue. These tumors are soft, elastic, usually pedunculated, tremulous, and vibratory. The stroma is very delicate and carries minute blood-vessels. Cutting into a myxoma causes a straw-colored, clear jelly to exude. Myxomata grow slowly, are encapsuled, have but little circulation, and the diagnosis may be impossible before removal of the growth. Some pathologists place myxomata among the malignant tumors, but most consider them as benign tumors, though they tend strongly to become sarcomatous (*myxosarcomata*). A sarcoma may undergo myxomatous degeneration.

Myxomata may arise from the skin; from the mucous membrane of the nose, the frontal sinus, the antrum, the womb, the auditory meatus, and the tympanum (*gelatinous polypi*); from the parotid and mammary glands; from the subcutaneous tissue, the nerve-sheaths, the intermuscular septa, the rectum, and the bladder (polyps). They may be congenital, but occur most often in young adults, as a result of inflammation. A sudden increase of growth indicates beginning malignancy (sarcomatous change). When a tumor begins to undergo myxomatous transformation we give to it a compound name; for instance, a chondroma undergoing myxomatous change is a chondromyxoma, a fibroma undergoing a like change is a fibromyxoma, etc. *Mucous polypi* grow from the mucous membrane of the nose, particularly from the outer wall near the middle turbinated bone, and often from the roof of the nares. Mucous polypi are soft and jelly-like, of a grayish color, and have stems or peidcles; they may be seen through the anterior nares, may project behind the veil of the palate, and may bulge out from the passages of the nose; they may be, and usually are, multiple; they may be present in one nasal fossa or in both; and they occur most commonly in youths and adults between the ages of fifteen and thirty-five years.

Hydatid moles of pregnancy are due to myxomatous changes in the chorion.

**Treatment.**—In treating myxomata, remove them promptly and thoroughly, because of the danger of sarcomatous change. Polypi of the bladder are removed by means of cutting forceps after suprapubic cystotomy has been performed. Nasal polypi may usually be twisted off or be removed by the wire snare or galvanocautery. Occasionally when the growths are numerous and recur rapidly after removal, the inferior turbinated bones should be removed with a saw (Rouge's operation). This operation secures ready access to the area of disease, which can be attacked radically. A very soft myxoma breaks up when removal is attempted, and the base must be cauterized.

**Myomata** are tumors composed of unstriped muscle-fiber mixed often with fibrous tissue. They are called *leiomyomata*. Tumors composed of striated muscle-fiber and spindle-cells are known as *rhabdomyomata*. They are very rare and are always sarcomatous. Leiomyomata are found in the womb, in the prostate gland, in the walls of the gullet, vagina, stomach, bladder and bowel, in the broad ligament, ovary and round ligament, in the scrotum, and in the skin. Myomata usually begin during or after middle age; they are encapsuled, they grow slowly, they are firm and hard, and produce annoyance by their size and weight or by obstructing a viscus or channel. A leiomyoma of the posterior portion of the middle of the prostate gland is known as a "middle lobe."

The so-called *uterine fibroid* is a myoma or fibromyoma. *Uterine myomata* may originate within the walls of the womb (intramural myomata), from the muscular structure of the mucous lining (submucous myomata), or

from the muscular tissue of the serous covering (subserous myomata). Intramural uterine myomata may be single or multiple and may grow to an enormous size. Submucous myomata project into the cavity of the womb (fleshy polypi), and may project into the vagina. They distend the uterus and are often accompanied by menorrhagia or metrorrhagia. In some rare cases the projecting tumor is detached by nature and the patient is cured; in some cases the myoma becomes gangrenous. A fleshy polyp may produce inversion of the fundus of the womb. Subserous uterine myomata cause trouble only by the inconvenience of weight or the discomfort of pressure. Uterine myomata are commonest in single women, and arise most frequently between the ages of twenty-five and forty-five. Negro women are especially prone to develop such tumors. They may never produce any symptoms. Some of these growths, by enlarging until they ascend above the pelvic brim, produce abdominal distention; some become jammed or impacted in the pelvis, and produce by pressure retention of urine, obstruction to the passage of feces, or hydronephrosis. Impaction may occur temporarily at each menstrual period. Many myomata produce uterine hemorrhage; some cause retroversion of the womb; some protrude from the cervical canal; some are so large that they cause disastrous pressure upon the colon (obstruction), upon the iliac veins (great edema), or upon the ureters (hydronephrosis). Uterine myomata usually shrink after the menopause. Pregnancy in a myomatous womb usually ends in abortion. Uterine myomata may undergo fatty, calcareous, or myxomatous change, and may be infected by septic organisms as a result of the use of a uterine sound or of infection of the pedicle after oöphorectomy. Infection of a uterine myoma causes great enlargement, elevated temperature, sweats, and exhaustion. Sarcomatous change may take place. Virchow pointed out this fact in 1863. Cullen found sarcomatous change in 17 cases out of 1400. If there be a carcinoma in some other part of the body metastatic deposit may occur in a uterine growth.

Uterine fibromyoma, if unoperated upon, often cause death. Noble claims that of cases denied operation or who refuse operation, 33 per cent. die and 28 per cent. become chronic invalids.

The symptoms of myomata of the alimentary canal are similar to or identical with the symptoms of malignant growths. Myomata of the skin are rare growths; they are encapsuled, firm or elastic, and painless.

**Treatment.**—Cutaneous myomata are removed in the same manner as fibrous tumors. A uterine tumor which causes symptoms should be operated upon. A tumor which causes no symptoms need not be operated upon if the patient can lead an easy life and can rest during the menstrual periods. Rest is an essential of the treatment. Ergot, thyroid extract, barium chlorid, and dilute sulphuric acid are recommended. In some cases the tumor shrinks, in some it disappears. If operation is required the form of operation chosen depends upon the case. In some cases the operation should be vaginal, and may be dilatation and curetment, torsion or incision of the pedicle, enucleation or hysterectomy. In other cases the operation should be by the abdominal route, and may be castration, ligation of vessels, myomectomy (cutting through the pedicle and closing with deep sutures), enucleation, shelling out growths from the wall, partial hysterectomy, or complete hysterectomy. Castration aims to create an artificial menopause, and so arrest hemorrhage and cause the shrinking of the growth. Sometimes it acts admirably, sometimes it fails, and it brings with it its own perils, for the sudden creation of the menopause is a great danger to the nervous system. For subserous myomata myomectomy is often the operation of choice. A very large tumor, a tumor which continues to enlarge after the menopause, and an infected tumor require partial or complete hysterectomy. If a myoma of the prostate causes severe obstruction, perform

a suprapubic cystotomy and remove the enlarged gland; or make both a suprapubic and a perineal opening, push the gland into the perineum and shell it out with the finger, or, if the condition is desperate, make permanent suprapubic drainage.

**Neuromata.**—A *true neuroma* springs from nerve-tissue (brain, cord, or nerve-trunks); it is composed of medullated or non-medullated nerve-fibers which form a plexus or network, and which are not continuous with the fibers of the nerve-trunk or other area from which the tumor grows. True neuromata are rare growths. They arise during middle life; are small in size, are due to injury or hereditary tendency, and may be single or multiple. There is usually around the tumor, rather than in it, severe neuralgic pain, which is greatly intensified by dampness, by blows, or by rough handling. The parts below a neuroma are cold, swollen, often anesthetic, and frequently present motor paralysis or trophic disorder. A *false neuroma* or *neurofibroma* is a fibrous tumor growing from a nerve-sheath, and is identical in structure with the sheath. False neuromata may be single, but they are often multiple; they may be as small as peas or as large as oranges; they are smooth and movable, and may cause great pain or may be painful only when pressed or struck; they may spring from roots, trunks, or branches, and they may be linked with the disease known as *molluscum fibrosum*. In *plexiform neuroma* some brances of a nerve enlarge and lengthen like an artery in a cirsoid aneurysm; the mass feels like beads or like a bag of worms; it is mobile, and no pain is felt on moving it; and it is generally congenital. In plexiform neuroma the nerve-sheath undergoes myxomatous change. *Malignant neuroma* is usually a primary sarcoma of a nerve-sheath, though any neuroma containing fibrous tissue may become sarcomatous.

*Traumatic neuromata* are false neuromata, and are occasionally well exhibited after nerve-section or amputation. After nerve-section the distal end shrinks and atrophies, the proximal end enlarges and becomes bulbous. A traumatic neuroma is composed of fibrous tissue which contains nerve-fibers. Such a growth is usually, but not always, painful on pressure or during dampness, and is most commonly seen in a stump which did not heal by first intention. In performing an amputation cut the nerves high up, and thus keep them out of the scar, permit them to remain mobile in their sheaths, and so prevent a tender stump. A tender stump may be due to anchoring of a nerve in a scar, the nerve ceasing to glide when the individual moves the extremity. The condition known as painful subcutaneous tubercle was discussed on page 355.

**Treatment.**—A false neuroma is to be removed, if possible, without destroying the nerve-trunk. If, in removing a neuroma, it is necessary to exsect a portion of a nerve-trunk, always proceed to suture the ends of the divided nerve so as to facilitate restoration of function. For multiple neuromata—at least should the number be large or should molluscum fibrosum exist—surgery can do nothing. Plexiform neuromata may often be removed, but amputation may be required. Painful neuromata in stumps should be excised.

**Gliomata.**—These tumors develop from neuroglia and more often from the white substance than from the gray. They are usually single, and arise not unusually in the brain, rarely in the cord, and very rarely in the cranial nerves. They may take origin in one of the cerebral hemispheres, in the cerebellum, in the pons, or in the medulla. Some gliomata are soft and bear a close relationship to sarcoma; others are hard and resemble fibroma.

A glioma is a circumscribed growth in contrast to a gliosis, which is a widespread and unlimited hyperplasia of the neuroglia. Syringomyelia is due to gliosis of the spinal cord.

"A glioma consists of cells containing rounded or oval nuclei with very

little protoplasm and fine protoplasmic extensions which interlace and form an intercellular reticulum" (Stengel).

A glioma passes almost insensibly into surrounding tissue, and there is no distinct edge; hence, because of the slight differentiation from brain substance, it may be overlooked during exploration. It is harder than the surrounding tissue, is vascular and of a pink or red color, and the normal shape of the part is often very little altered, although the tumor may reach the size of a lemon.

Hemorrhage may take place into a glioma, softening may occur, cavities may form, or the growth may become sarcomatous or psammomatous. The symptoms of a glioma of the brain depend on the situation.

**Treatment.**—When a glioma of the brain can be localized and is hard, removal should be attempted. No attempt should be made to remove a soft glioma.

**Angiomata or Hemangiomata.**—An angioma is a tumor composed largely of dilated blood-vessels. The older surgeons called such growths

Fig. 153.—Dr. Hansell's case of cavernous angioma of the eyelids.

Fig. 154.—Cavernous angioma of face.

*erectile tumors*. Some of the so-called angiomata are not genuine new growths, but are due to dilatation and elongation of blood-vessels.

**Simple or capillary angiomata, nevi, or "mother's marks,"** which affect the skin or subcutaneous tissue, are composed of enlarged and twisted capillaries and of anastomosing vessels surrounded by fat. These growths are congenital or appear in the first few weeks of life; they are flat and slightly raised, and are of a bright-pink color if composed chiefly of arterioles, and are bluish if composed mainly of venules; they are but little elevated; they can be almost completely emptied by pressure; they occasionally pass away spontaneously, but usually grow constantly and may become cavernous; they may ulcerate and occasion violent or fatal hemorrhage. One or several large vessels connect a nevus to adjacent blood-vessels. **Port-wine or claret stains** are pink or blue discolorations due to superficial nevi of the skin; they may be small in extent or they may involve a very large area, are not elevated, and do not usually spread. *Telangiectasis* is a form of nevus involving the skin and subcutaneous tissue in which many arterioles and venules exist. Simple angiomata are common on the forehead, the scalp, the face, the neck, the back, and the extremities. They may appear on the labia, the tongue, or the lips.

**Cavernous angiomata,** or **venous nevi** (Figs. 153 and 154), resemble in structure the corpora cavernosa of the penis; there are large endothelial lined spaces with thin walls carrying blood, and there may be distinct vessels as well. Arteries send blood into the spaces, and veins receive it from the spaces. These channels and sinuses are enormously distended capillaries. Cavernous angiomata arise in the skin and subcutaneous tissues; they are usually congenital, but may develop from simple angiomata; they are purple or blue in color; are more distinctly elevated than the capillary nevi; may be either cutaneous or subcutaneous; swell when the child cries, and are apt to pulsate; they may be emptied by pressure, and often look like cysts with very thin walls. Cavernous angiomata may arise in the breast, the tongue, the lip, the cheek, the gums, the subcutaneous tissues, or the muscles. If an angioma contains an excess of fat, the growth is called a "nevoid lipoma."

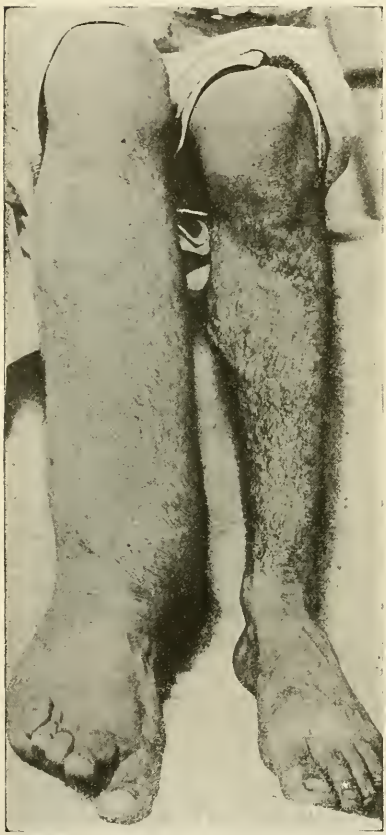


Fig. 155.—Cavernous angioma: Subcutaneous tissue of leg.

**Plexiform angiomata** are known as "cirsoid aneurysms" or aneurysms by anastomosis (see page 434).

Angiomata noticed soon after birth may disappear completely or may enlarge progressively.

**Treatment.**—A capillary nevus can often be quickly cured by touching it with fuming nitric acid. A second application of acid may be required. The growth may be destroyed by heat—"a knitting-needle at a dull-red heat or the galvanocautery" (Wharton). The application of ethylate of sodium or the employment of electrolysis will destroy the growth. Solid carbon dioxid is valuable to destroy capillary nevi. Often but one application is necessary, but in some cases two or more are required. Astringent injections are dangerous unless the base of the nevus is ligated, because they may lead to the formation of emboli.

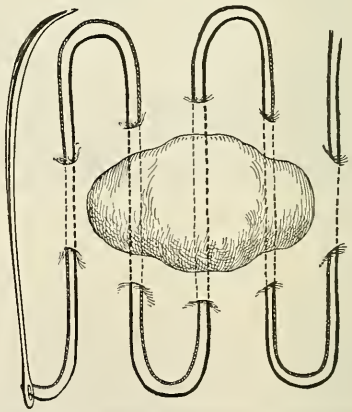


Fig. 156.—Method of applying Erichsen's suture.

Small port-wine stains may be removed by electrolysis or multiple incisions, but extensive stains are ineffaceable. Solid $CO_2$ may be used. Two or three applications are made in the same place and then another spot is selected. It

usually fails to cure. Small nevi may be ligated under harelip pins, larger nevi may be strangulated in sections by the Erichsen suture (Fig. 156), or may be completely excised. Excision is usually the best plan for the cure of angiomata. It is rapid, thorough, and leaves but a trivial scar. Excision should always be employed if we feel sure that the edges of the wound can be subsequently approximated and that there will not be a dangerous loss of blood. It is sometimes justifiable to excise an angioma even when approximation of the wound will obviously be impossible. In such a case the raw surface should be covered with Thiersch grafts.

Most superficial nevi and some cavernous angiomata can be treated by solid $CO_2$, and, if this fails, by excision. The incisions must be beyond the dilated vessels. In large angiomata involving the skin and also deeper parts, or involving a structure like the lip, which it is undesirable to remove, electrolysis should be employed. The operation should be carried out with aseptic care, and if the tumor is large an anesthetic should be given.

The positive pole produces a firm and hard clot. One or more needles

Fig. 157.—Cavernous angioma and lymphangioma.

Fig. 158.—Cavernous angioma, lymphangioma, and lymphangiectasis, also beginning cancer.

connected with the positive pole are inserted into the tumor, the needles being insulated to within about $\frac{1}{4}$ inch of their points. A flat moist pad is placed upon the skin near the tumor and is attached to the negative pole, and the pad is moved from time to time during the operation.

From 25 to 75 milliampères is the proper strength, and the current is passed for ten minutes. The current is increased for a moment before withdrawing the needles, otherwise they will stick to the tissue and cause bleeding when torn loose. After the withdrawal of the needles the nevus will be found to be hard, but the hardness will gradually disappear. It may be necessary to repeat the operation a number of times at intervals of ten days.[1]

[1] Cheyne and Burghard's "Manual of Surgical Treatment."

When solid $CO_2$ is applied it subjects the tissue to a temperature of 79° F. below zero. It is called carbonic-acid snow. It is applied to nevi with pressure. This drives out the blood. It is held against the part for thirty or forty seconds. It drives the color out of the part, but a few minutes after the cessation of the action color returns, reaction occurs, the surface becomes moist, and fibrinous inflammation takes place (E. R. Morton, "Lancet," May 7, 1910).

Necrosis seldom arises. Within an hour a vesicle forms, which should be opened aseptically. A crust forms, which drops off in about two weeks, leaving "a soft and elastic scar," the color of the normal skin (Morton, Ibid.).

**Lymphangiomata** are tumors composed of dilated lymph-vessels and are often, though not invariably, congenital (Fig. 158). A *lymphatic nevus* is a colorless or faintly pink elevation; if it is punctured with a needle, lymph flows from the puncture. One or several nevi may be present in the same individual. The dilatation is due to blocking of the lymph-channels. Local lymphangioma of the tongue is manifested by a cluster of papillary projections containing lymph. *Macroglossia* is a congenital enlargement of the anterior portion of the tongue, which enlargement grows more and more marked, until finally the tongue is forced far out of the mouth. This condition of tongue enlargement is due to lymphangioma of the mucous membrane. *Lymph scrotum* is due to a similar growth. A collection of these warty-looking dilatations is called *lymphangiectasis*. Just as cavernous angiomata constitute a variety of blood-vessel tumors, so *cavernous lymphangiomata* constitute a variety of lymph-vessel tumors, and the spaces of the latter are filled with lymph instead of with blood (Figs. 157 and 158). Areas affected with lymphangiectasis are liable to repeated attacks of erysipelas-like inflammation. Whether this inflammation is causative or secondary is not known. In tropical countries blocking of lymph-channels may be brought about by the Filaria sanguinis hominis, a parasite which lurks in the lymph-vessels during the day and is found in the blood only at night. Lymphangiectasis is often the first stage of elephantiasis.

**Treatment.**—A lymphatic nevus requires excision. In macroglossia the bulk of the mass should be removed by a V-shaped cut, the mucous membrane being sutured so as to cover the stump. In conditions due to the filaria, anilin-blue has been given internally.

**Malignant Connective-tissue Tumors, or Sarcomata.**—The sarcomata are composed of embryonic tissue-cells, the intercellular substance being very scanty, and they *resemble* a process of chronic inflammation. They develop from connective tissue, rarely have a definite stroma, and the constituent cells, as a rule, proliferate with great rapidity. If a sarcoma has a stroma of connective tissue, this stroma contains lymphatics and such a sarcoma infects adjacent glands. In most cases there is no connective-tissue stroma and no lymphatics. In a sarcoma without a definite stroma the blood-vessels are not surrounded by lymph-spaces and are quickly invaded by cells (B. H. Buxton). The rapidly growing forms are very vascular, the blood flowing in vessels whose walls are very thin or running in canals lined by endothelium and bounded by sarcomatous cells. Such a tumor may pulsate and have a bruit, and hemorrhage often takes place into its substance. A rapidly growing soft sarcoma with dusky skin above it (Fig. 161) may be mistaken for an abscess. A slow-growing sarcoma has but few vessels. Sarcoma tends strongly to infiltrate adjacent parts. The growth disseminates by means of the blood and the vessel walls, particles of the tumor being carried by the venous blood to the heart, and from this organ to the lungs, where they lodge and form secondary growths. Emboli from these secondary foci are sent out by the arterial blood to various portions of the body, as the bones, kidneys, brain, liver, etc. This process is known as *metastasis*. In some cases sarcoma

is disseminated widely throughout the body, almost all the tissues showing minute white spots of secondary sarcoma which resemble tubercles. Such widespread dissemination is called *sarcomatosis*. Sarcoma follows the vein walls for considerable distances and builds elongated masses of tumor substance inside the veins. The primary tumor may possess a capsule when it is in an early

Fig. 159.—Sarcoma of antrum.

Fig. 160.—Sarcoma of antrum.

stage, but soon loses this except in very slow-growing varieties or in mixed forms growing by central proliferation, but secondary sarcomata are often encapsuled. Sarcomata may arise at any age from birth to extreme senility, but

Fig. 161.—Small round-celled fungating sarcoma of neck.

they are commonest during youth and early middle age. They are not hereditary. They often follow traumatism and inflammation. A number of observers maintain that they are due to parasites (the question of the parasitic origin of malignant disease is discussed on page 348). A sarcoma may be primary or

may arise from malignant change in an innocent connective-tissue growth (chondrosarcoma, fibrosarcoma, etc.). A sarcoma rarely affects adjacent lymphatic glands unless it contains lymphatics, and the great majority of sarcomata do not contain them. Occasionally sarcoma cells are carried to adjacent glands by the vein walls rather than by the lymph-stream. Sarcoma of the tonsil, sarcoma of the testicle, melanotic sarcoma, and lymphosarcoma do affect the glands. The skin over the tumor may give way, a bleeding fungous mass protruding (fungus hæmatodes) (Figs. 161, 162, and 163), and suppuration may cause septic enlargement of adjacent glands. In the course of growth of a sarcoma there may be irregular episodes of elevated temperature (see page 351). After removal of a sarcoma the growth tends to recur, and the recurrent tumor may be either more or less malignant than its predecessor, the degree of malignancy being in direct ratio to the number and smallness of the cells. A sarcoma is malignant by local tissue infection and by dissemination. Sarcomata rarely cause pain when they are not ulcerated. They are commonest in the skin and connective tissue of the extremities, but they arise also

Fig. 162.—Small round-celled sarcoma of neck. Skin has given way and a bleeding mass is exposed.

Fig. 163.—Sarcoma of neck (Horwitz).

from bone, neuroglia, periosteum, the lymphatic glands, the breast, the testicle, the eyeball, the parotid, and other parts. A pigmented mole may become sarcomatous. Hemorrhages into a sarcoma often occur, with the result of suddenly increasing the size of the mass and formation of blood-cysts. Sarcomata are subject to partial fatty degeneration, to myomatous changes which produce cavities filled with fluid, to calcification, and occasionally to necrosis of large masses.

**Varieties of Sarcomata.**—The following species of sarcomata are recognized:

1. *Round-celled sarcoma* is a tumor composed of round or spherical cells and resembling a chronic inflammatory area. The intercellular substance is scanty, the mass is soft and vascular, and grows with great rapidity. It often softens, and may become cystic. The cells may be small or large. The smaller the cells, the more malignant the growth. A growth composed of small round cells is the most malignant form of sarcoma (Fig. 166). *Lymphosarcoma* is a form of round-celled sarcoma which arises from lymphatic

glands, lymphoid tissues, the thymus gland, the spleen, and some other structures. The structure of a lymphosarcoma resembles the structure of a lymph-

Fig. 164.—Dr. W. R. Bishop's case of small-celled sarcoma of the antrum.

Fig. 165.—Osteosarcoma of eighteen months' standing of right side of superior maxilla. Note bony lump on left side of lower jaw.

gland in the fact that it has a reticulum which looks like lymph-adenoid structure. *Chloroma* is a form of lymphosarcoma arising particularly from the periosteum of the bones of the cranium and face. The cells contain greenish pigment, hence the name. What is known as *glioma* of the eyeball is not a true glioma, but is really a sarcoma composed of small round cells.

Fig. 166.—Small round-celled sarcoma of the neck.

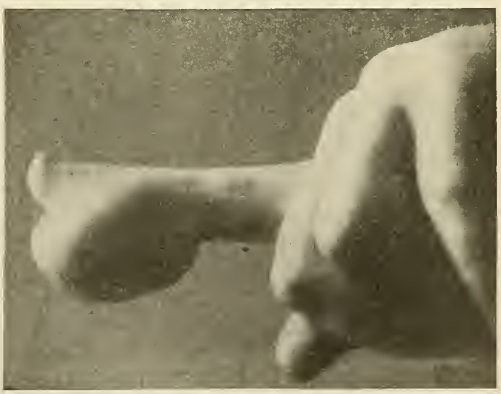


Fig. 167.—Spindle-celled sarcoma of sheath of flexor tendon of finger.

2. *Spindle-celled sarcoma* is a tumor composed of large or small spindle-shaped cells lying in a matrix, which may be homogeneous, but which may

show some attempt at fiber formation. Angular cells and stellate cells are often present. The cells may be placed in columns, which are at some places nearly parallel and which at others diverge or interlace. Often there is no orderly arrangement. Spindle-celled sarcomata are usually harder than round-celled growths, but are sometimes quite soft. Cystic changes may occur. If there is a large amount of intercellular substance the growth is known as a *fibrosarcoma.* A *rhabdomyoma* is really a spindle-cell sarcoma containing striated muscle-cells. The spindle-cell sarcomata often contain cartilage. Spindle-cell growths are by no means as malignant as round-cell tumors. Often they do not show any tendency to metastasis. The greater the amount of intercellular substance and the fewer the cells, the less the malignancy. Bloodgood points out that in one group of cases (the least malignant) the spindle cells exhibit a disposition to form fibroblasts (*fibrospindle-cell sarcoma*). In another group (the most malignant) there are no fibroblasts and round cells are distributed among the spindle cells (*mixed spindle-cell* and *round-cell sar-*

Fig. 168.—Melanotic sarcoma. Observe the pigmentation of the face.

*coma*). Spindle-cell growths constitute the majority of sarcomata met with in practice.

3. *Giant-cell, myeloid,* or *medullary sarcoma* is characterized by the presence of numerous very large cells with many nuclei, looking exactly like the myeloplaques of bone-marrow. Such a growth is maroon colored on section. It arises usually from bone, especially from the interior of a long bone, hence is often called *osteosarcoma.* It is almost invariably single, but Rehn reported a case of multiple giant-cell sarcoma of bone, and Crile and Hill reported another. It may, however, arise from other structures than bone. It is the least malignant form of sarcoma. Metastases do not occur and the growth often admits of complete extirpation and cure. Some surgeons do not class these growths with sarcomata. Bloodgood regards their malignancy as very slight. Friedländer looks upon them as benign angeiomata in which giant cells have been formed by endothelial cells budding into spaces lined with endothelium.

4. *Alveolar sarcoma* is a tumor containing both round cells and spindle cells, and characterized by the formation of acini, filled with round cells of

large size resembling epithelioid cells. The walls of the acini are formed of spindle cells and fibrous tissue, and in these trabeculæ are the blood-vessels. The collection of the cells in the alveoli makes the structure resemble that of a cancer. Such growths are often pigmented. Alveolar sarcomata arise particularly from moles of the skin, but may arise from lymphatic glands, serous membranes, the testicle, and other parts. Such growths are very malignant.

5. *Melanotic or Black Sarcoma* (Fig. 168).—The color of such a tumor is due to pigment in the cells or matrix. These growths are usually composed of round cells, but may consist of spindle cells, and they are sometimes alveolar. Melanotic sarcomata spring from parts which contain pigment (for instance, the skin and the choroid coat of the eye), pigmented moles, and pigmented nevi; they are very malignant; they implicate related lymphatic glands, undergo early metastasis, and during their existence the urine contains pigment.

Malignant growth from a congenital pigmented mole used to be regarded by most observers as melanotic sarcoma, but Bloodgood would place these growths in a group by themselves. He says that the weight of opinion is on the side of those who maintain that the cells are of epithelial origin misplaced early in embryonic life, and that the tumor is a cancer. Many surgeons still regard such a tumor as sarcoma.

Fig. 169.—Hansell's case of cystic myxosarcoma of the orbit.

6. *Hemorrhagic sarcoma* is a sarcoma containing blood-cysts which result from parenchymatous hemorrhages.

7. *Angeiosarcoma* or *hemangeiosarcoma* takes origin from the outer coat of a blood-vessel. The growth is often very vascular, and when the blood-vessels are notably dilated the tumor is called a *telangiectatic sarcoma*. The ordinary forms of angeiosarcoma are only moderately malignant, but alveolar and melanotic forms occur which are highly malignant. Angeiosarcoma may arise in the skin, in a serous membrane, in intermuscular structure, in bone, or in a salivary gland. It most frequently takes origin from a nevus.

8. *Cylindroma, or Plexiform Sarcoma.*—In this variety the sarcoma cells adjacent to vessels have undergone hyaline or myxomatous degeneration; the cells distant from vessels are unchanged. Section shows the sarcoma cells apparently contained in spaces with hyaline walls. These degenerative changes occur most often in the angiosarcomata. Cylindromata arise from the brain, salivary glands, lacrimal glands, and rarely from the subcutaneous tissue. The growths are only moderately malignant.[1]

9. *Mixed tumors* consist partly of mature and partly of embryonic tissue, the cellular elements exceeding the adult elements in amount. Among these mixed tumors are *fibrosarcoma* or the *recurrent fibroid tumor*, *myxosarcoma* (Fig. 169), *chondrosarcoma*, *gliosarcoma*, and *osteosarcoma*.

[1] Stengel, "Text-Book of Pathology."

10. *Endotheliomata* are tumors springing from endothelium, and the name is retained no matter what changes the growths ultimately undergo. Many writers include under the term "endothelioma" *psammoma*, myxosarcoma, angiosarcoma, and plexiform sarcoma. Others consider endothelioma a special and characteristic form of sarcoma. Some would not consider it with the sarcomata at all. The growth may take origin from the "endothelium of the blood-vessels and of the perivascular lymph-spaces, of the lymph-vessels, and of the great serous cavities (peritoneum, pleura, meninges)."[1] The characteristic cell is the endothelial cell, usually known as the epithelioid cell. The structure of these tumors is very variable and depends upon the origin; some tumors "recalling the original vascular network" ("American Text-Book of Pathology"), others being distinctly alveolar. Many pathologists consider a *psammoma* of the dura to be an endothelioma with a fibrous stroma. A psammoma contains calcareous particles. In appearance an endothelioma strongly resembles cancer, and such a growth is often spoken of as endothelial cancer. Such growths can arise in many different situations, but are particularly common in the peritoneum, pleural membrane, membranes of the brain, ovary, and testicle. I have removed an endothelioma of the tonsil, one of the mammary gland, two of the nasopharynx, one of the superior maxillary bone, and two of the carotid gland. The proliferating endothelial cells lie in lymph-spaces. Many endotheliomata grow rapidly, secondary growths form, and metastases are apt to pass to the serous membranes. Certain endotheliomata grow slowly, do not infiltrate adjacent structure, and do not produce secondary growths. In the brain and cord endothelioma may produce no symptoms for a long time. It is not as yet possible, clinically, to distinctly recognize endotheliomata from ordinary sarcomata.

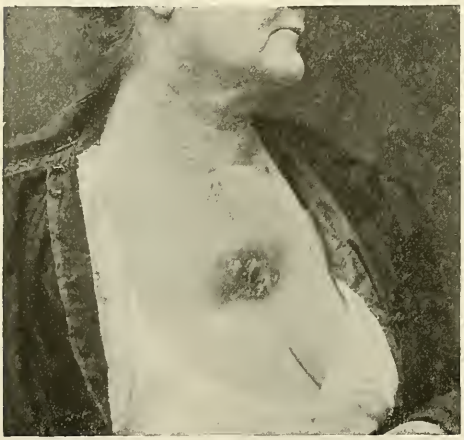


Fig. 170.—Recurrent sarcoma of the sternum.

Fig. 171.—Periosteal sarcoma of the femur.

11. *Mycosis* or *granuloma fungoides* is a disease which resembles sarcoma in many particulars and may perhaps be a form of sarcoma. It attacks the skin and subcutaneous tissues. It is preceded for months or years by an eczematous or urticarial condition. The skin becomes red and swollen; numerous pinkish or reddish nodules or flat areas of induration form; the nodules become distinct tumors, soften at their centers, usually ulcerate, and fungation

[1] "An American Text-book of Pathology," edited by Hektoen and Riesman.

occurs. When ulceration occurs the mushroom-like growths form. Microscopically, the tumor resembles a lymphadenoma. Mycosis fungoïdes is considered by some pathologists to be multiple cutaneous sarcoma. It is very chronic and lasts for months or even years. It was first described and was named by Alibert.

**Treatment of Sarcomata.**—Remove a sarcoma at once if it is in an accessible spot. Never delay removal, and always cut well clear of it. If affecting a part where amputation is impossible, the rapidly growing sarcomata will almost inevitably return, and the very malignant variety, if uninterfered with, may ter-

Fig. 172.—Central sarcoma of humerus.

minate life in six months; but even in such case operation postpones the evil day and renders it possible that death will occur from metastatic growth in an organ, and that the patient will escape the horrors of ulceration and hemorrhage from the original tumor. Slowly growing and hard tumors offer better prospects of cure. The mixed tumor (as a recurrent fibroid) may repeatedly recur, and yet the patient may be cured at last by a sixth, an eighth, or a tenth operation. In a case of spindle-cell sarcoma of the breast the younger Gross performed 22 operations in the course of four years, and eleven years later the woman was well. In a case of recurrent fibroid of the neck the younger Gross operated five times. Three years after Prof. Gross's death I

operated upon the same patient, and again two years later. Nine years after the last operation she was alive and well. In sarcoma of a long bone (though not in giant-cell sarcoma) amputation should, as a rule, be performed. Bloodgood proves that in some of these cases extensive excision is just as useful. In giant-cell sarcoma incision and curetting may be employed, or, if this is insufficient, subperiosteal excision. Bloodgood has reported the cases of giant-cell sarcoma from Halsted's clinic. The reports show that excellent results follow this plan of treatment. If the soft parts are involved, they must be removed wide of the growth. Amputation is necessary only when the removal of soft parts must be so extensive as to hopelessly mutilate the limb. In sarcoma of either jaw-bone, excision; of the eye, enucleation; of the testicle, cas-

Fig. 173.—Central sarcoma of the fibula.

tration is demanded. Sarcoma of the ovary in adults demands removal, but in children the operation is generally useless. Sarcoma of the kidney in adults calls for nephrectomy, but in children the operation is usually of little avail. In my experience, in the cases of sarcoma of the kidney which survived operation the growth always appeared in the other kidney. In melanotic sarcoma extirpate the growth widely and remove anatomically related lymph-nodes, or in some cases amputate far away from the tumor and remove lymph-nodes. In very malignant sarcoma even amputation does not often cure. Removal of a sarcoma when there is no hope of a cure is often justifiable to prolong life, to relieve the patient of a foul, offensive, bleeding mass, and to permit of an easier road to death by means of metastasis to an internal organ. In an inoperable case the ligation of the vessel of supply may do good. In sarcoma of

the tonsil Dawbarn advises the extirpation of the external carotid artery and the ligation of its branches. The operation is performed first on the side occupied by the tumor and in a week or so on the other side. I employed it in 7 cases with distinct but temporary benefit. Occasionally, though very rarely, suppuration cures a sarcoma. Wyeth, of New York, reported a case of sarcoma of the abdominal wall. It was found possible to remove only part of the growth; suppuration followed and the tumor disappeared, and ten years later had not returned. A study of statistics seems to indicate that more cases of sarcoma are cured after operation if the wound suppurates than if it remains aseptic, and it has been proposed to deliberately infect the wound with pus germs to lessen the danger of recurrence. If the wound is large, it should not be infected until it is nearly healed. If it is small, it may be infected at the time of operation or soon after. After amputating for sarcoma, Wyeth waits until the wound is nearly healed and then infects it by inserting a gauze drain saturated with cultures of pure Streptococcus pyogenes (Wyeth's "Surgery"). After removing a sarcoma from any region the patient should be given courses of injections of Coley's fluid (see below).

Fig. 174.—Inoperable sarcoma of the back.

It has been observed that an attack of erysipelas occasionally greatly benefits a sarcoma, causing large masses of the growth to soften or to slough and exposing a granulating surface. Busch noticed this in 1866, but the fact had been observed in the seventeenth century. Interest was decidedly awakened by Billroth's case of sarcoma of the pharynx which was cured by an attack of facial erysipelas. It was suggested that in inoperable cases of sarcoma erysipelas might be established artificially. Fehleisen inoculated tumors with cultures of erysipelas. Lassar in 1891 employed the toxins (cultures rendered sterile by heat and filtration). In 1892 Coley began his observations. The first plan was as follows: a bouillon culture was made of the streptococci; this culture was filtered through porcelain and an injection was given once a day into and about the sarcoma. The first dose was 10 min. and it was progressively increased. The effect was to cause a febrile reaction, and sometimes the injections were followed by softening or suppuration. Coley's present method is as follows: Make cultures of erysipelas cocci in cacao broth; after three weeks inoculate them with the Bacillus prodigiosus, and cultivate the mixed growth for four weeks. The mixed cultures are maintained at a temperature of 136° F. until they become sterile. This sterile fluid contains the toxins. The usual dose for an adult is from 1 to 8 min. Coley has given as high as 24 min. I have never given over 18 min. Most cases will show reaction at 4 to 6 min. The first dose for an infant is $\frac{1}{10}$ min. If in an adult the fluid is injected remote from the tumor the initial dose should be 1 min. If the fluid is injected into the tumor the initial dose is $\frac{1}{4}$ to $\frac{1}{2}$ min. (Coley, in "Amer. Jour. Med. Sci.," March, 1906). Some cases are treated purely by distant injections (gluteal or pectoral regions), others by alternately injecting the tumor and some distant point. The latter plan combines local action

and systemic effect. The dose should be gradually increased until a chill occurs in from one-half an hour to two hours after the injection, followed by a temperature of 101° to 104° F. In some cases there is so much depression after reaction that injections are given every other day, but if safely possible they should be given every day. The object is to obtain a reaction with each injection. The more vascular the tumor, the more severe the reaction (Coley). If an area softens during treatment Coley advises us to open and drain the softened area. If improvement is going to occur it usually begins in from one to four weeks. If there is no improvement within four weeks there will not probably be any. In most cases as injection is continued susceptibility diminishes, in some few it increases. It seems definitely proved that cases are occasionally cured by Coley's fluid. Spindle-celled sarcomata are influenced most favorably. Round-celled sarcomata are very refractory and so are cancers. The method is not entirely free from danger, but the danger is very slight if treatment is begun with the minimum dose. The toxins seem of value in postoperative cases to prevent recurrence. For this purpose the fluid is used twice a week for several months and at greater intervals for a long period of time. During the autumn of 1910 I brought before my class in the Jefferson Hospital a colored woman (Fig. 175) with an inoperable spindle-cell sarcoma of

Fig. 175.—Huge sarcoma of buttock cured by partial extirpation and Coley's fluid.

the thigh and groin. A portion of the growth was removed and the remainder completely disappeared from injections of Coley's fluid. She remains well over three years after treatment was suspended. This patient had a violent reaction every day for weeks. She was given as much as 7 min. at a dose.

How the toxins act is uncertain. They produce some change in the blood-serum, and the valuable effect is systemic. Nearly always they cause leukocytosis. Probably they cause the formation of antibodies, which are antagonistic to the sarcoma cells. The treatment, as Coley insists, is not a substitute for operation. The fluid is used in inoperable cases, a trial of it is made in sarcoma of long bones before advising operation, it is given after all operations for cancer or sarcoma to combat recurrence, and it may be tried in inoperable cancer. Coley ("Surg., Gynec., and Obstet.," August, 1911) has had 65 cases of inoperable sarcoma in which the tumor disappeared from the treatment: 7 are alive and well at the end of fifteen to eighteen years; 7 alive and well at the end of ten to fifteen years; 17 alive and well at the end of five to ten years; 10 alive and well at the end of three to five years. The others could not be traced.

Emmerich and Scholl claim good results in inoperable sarcoma from the injection of erysipelas serum. A sheep is injected with cultures of erysipelas, the blood is drawn, the serum separated, filtered to remove cocci, and injected

about the sarcoma. Results are not definite. Among other agents which have been used to inject inoperable sarcomata we may mention alcohol, chlorid of zinc, arsenic, corrosive sublimate, thiosinamin, pepsin, alkalis, etc. The injection of anilin products into the sarcoma, which once received qualified commendation from some observers, has been abandoned by most surgeons. The *x*-rays are sometimes of benefit, but are not so serviceable as in carcinoma, and possess a certain danger, for occasionally after using them dissemination rapidly occurs. Abbe and others have obtained some remarkable results by radium, but such results are exceptional and not the rule.

**Hypernephromata, or Adrenal Tumors.**—Some of the tumors bear a strong resemblance to adenomata and carcinomata. Some adrenal tumors are benign, and among such tumors we note fatty and fibrous growths and growths resembling glioma. Another benign growth imitates the structure of the cortex of the adrenal. Malignant tumors occur, and many of them are identical or almost identical with sarcoma. One form is composed of epithelioid cells and resembles endothelioma. An adrenal tumor may arise from the adrenal body proper or from "rests" in ectopic portions of adrenal within the kidney, ovary, testicle, solar plexus, renal plexus, liver, mesentery, or some other part. Some of these tumors attain a large size. Metastases are late, but tend to occur eventually even in hypernephromata which seem benign, and may occur even when the primary growth has given rise to no symptoms. The metastases are lodged particularly in the bones, the lungs, and the liver. In a case from which I removed a goiter and death resulted from reactionary hemorrhage the tumor (which I had considered to be adenoma) was found to be composed of adrenal tissue. Unfortunately, an autopsy was not permitted.

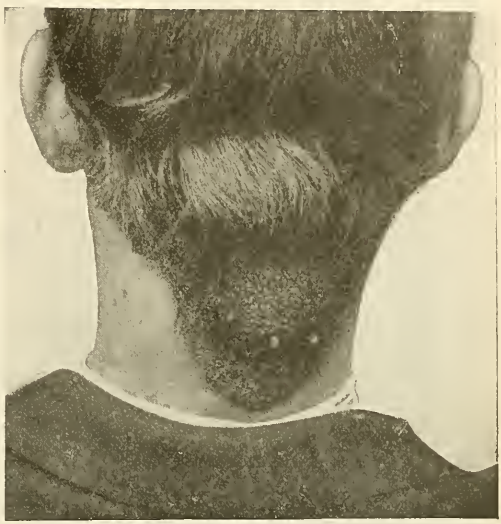


Fig. 176.—Keen's case of papilloma with angioma.

**Accessory adrenals** are common. They are known as *adrenal rests*. "They are found oftenest in the connective tissue about the main adrenals, but also in the kidneys, the right lobe of the liver, along the renal vessels and spermatic veins, in the inguinal canals, and in the broad ligaments" ("American Text-Book of Pathology").

**Innocent Epithelial Tumors.**—These growths imitate an epithelial tissue of the mature and healthy organism.

**Papillomata, or Warts** (Fig. 176).—Papillomata are formed upon the type of cutaneous and mucous papillæ. A papilloma consists of a fibrous stroma, which contains blood-vessels and lymphatics and is covered by epithelium of the variety appertaining to the diseased part. Papillomata grow from the skin and from mucous membranes; they may be single or multiple; many may form in one region or various distant parts may be affected; they may be painless or may be ulcerated or bleeding; they vary in color from light pink to deep brown or black. Papillomata of the skin are usually hard; papillomata of mucous membranes are soft. A skin-wart may be smooth and rounded,

or may look like a small cauliflower, the epidermis upon it being very rough. A papilloma of a mucous membrane looks like a small cauliflower. Papillomatous masses may gather around the anus, the vagina, or the penis during the existence of a filthy discharge (*venereal warts*, Fig. 177), and crops of warts may appear on the hands of those who work in irritant material (as petroleum). Papillomata are apt to arise in mucous membrances about carcinomata or chronic ulcerations. A large crop of warts may disappear in a single night; hence the popular belief in the efficacy of charms. Warts are particularly common on the skin of the back of the hands and fingers, the skin of the back, and the skin of the neck and scalp. A single skin-wart may reach the size of a walnut and become pigmented. The squamous epithelium covering a skin-wart may become horny (*a wart-horn*). Other cutaneous horns arise from the nails, from the scars of burns, or from ruptured sebaceous cysts.

*Villous papillomata* grow chiefly from the bladder, but they may also grow from the stomach and intestine. A papilloma of mucous membrane covered with squamous epithelium looks like a wart of the skin. Papillomata of the

Fig. 177.—Venereal warts.

larynx are formed of squamous epithelium. Villous papillomata form tufts like the villous processes of the chorion; they may be single or multiple, and may be sessile or pedunculated; they are very vascular, and are apt to bleed freely. Papillomata may arise in cysts of the paroöphoron, in cysts of the mammary gland, from the choroid plexuses of the ventricles of the brain, and from the spinal membranes. Papillomata may give rise to hemorrhage or may impair the function of a part. Any papilloma may become a cancer.

**Treatment.**—Venereal warts are treated by repeatedly washing with peroxid of hydrogen, drying with cotton, and dusting with a powder composed of borated talcum or of equal parts of calomel and subnitrate of bismuth, or of oxid of zinc and iodoform. If they do not soon dry up, cut them off with scissors and burn with the Paquelin cautery. Ordinary warts may usually be destroyed in a short time by daily applications of lactic or chromic acid. Most warts are easily destroyed by solid $CO_2$. It is held in contact with the wart for thirty or forty seconds. In a few days the warts drop off or can be easily picked off. In multiple warts of the face Kaposi applies daily for several days a portion of the following combination: sublimed sulphur, 5 dr.; glycerin,

1½ dr.; acetic acid, 2½ oz. Keeping a wart constantly moist with castor oil will usually cause it to drop off. Warts, and even extensive callosities, may be removed by painting once a day for five days with pure carbolic acid and covering with lint kept wet with boric acid. A convenient plan is to paint a wart daily with a solution containing 1 part of corrosive sublimate to 30 parts of collodion (hydrarg. chlor. corros., ½ dr.; collodion, 15 dr.). Large warts should be excised. Villous papillomata of the bladder, if not cured by fulguration, demand the performance of a suprapubic cystotomy in order to remove them. A papilloma of the larynx may be removed by the cautery or may be destroyed by sparking.

**Adenomata** are tumors corresponding in structure to normal epithelial glands. They have a framework of vascular connective tissue, and they may contain acini and ducts like racemose glands or tubes like tubular glands. The acini or tubules contain epithelium of either the cylindrical or polyhedral variety. Adenomata grow from secreting glands; either they cannot produce the secretion of the glands from which they spring, or they do secrete, but the fluid is retained and not discharged by the gland-ducts. Adenomata occur in the mammary gland, the parotid, the ovary, the thyroid gland, the liver, the sweat-glands, the sebaceous glands, the kidney, the pylorus, and the prostate; and they may spring as pedunculate growths from the mucous lining of the intestine and uterus. They are encapsuled, are usually single, but may be multiple, are of slow growth, but may attain a great size; they do not tend to recur after thorough removal, do not involve lymph-glands, and do not disseminate; they are firm to the touch; they tend to become cystic (especially in the thyroid gland), the fluid which distends the ducts being formed by mucoid liquefaction of the proliferating epithelium. If cysts form, the growth is spoken of as a *cystic adenoma*. If the framework of an adenoma contains considerable fibrous tissue, the tumor is named a *fibro-adenoma*. Adenomata are particularly liable to become carcinomatous.

In the breast a fibro-adenoma has a distinct capsule; it is elastic and movable, is usually superficial, and one occasionally exists in each gland. They are most common before the age of thirty, and are often painful, especially during menstruation. Cystic adenomata of the breast attain a large size; they are encapsuled and grow slowly, are most common after the thirtieth year, and are rarely painful. Both fibro-adenoma and cystic adenoma may arise in the male breast. Young unmarried women not unusually develop in the breast small, very tender, and painful bodies, most usually around the edge of the areola, which bodies increase in size and become more tender during menstruation; they are only cysts of the mammary tissue.

Adenomata of the thyroid gland usually begin before the fifteenth year. They may arise in the prostate if that gland be already the seat of senile hypertrophy. Adenomata of mucous glands may arise in the young or middle aged. Adenomata of mucous membranes often cause hemorrhage and interfere with function.

**Treatment.**—Adenomata should be extirpated. To let them alone exposes the patient to the danger of cancerous change. By confusing adenomata of the mammary gland with small areas of chronic mastitis an erroneous belief has arisen that the former as well as the latter may sometimes be cured by the local use of iodin, mercury, ichthyol, and the internal use of iodid of potassium. The treatment in the breast, as elsewhere, is excision.

**Malignant Epithelial Tumors, Carcinomata, or Cancers.**—Cancers are tumors taking origin from epithelial structures and composed of embryonic epithelial cells which are clustered in spaces, nests, or alveoli of fibrous tissue, and which proliferate enormously, extending beyond normal anatomical boundaries and as an invading host entering into connective tissue by way of the lymph-spaces. Certain cells "pass out of somatic coördination"

with the other body cells and become as parasites (Walker, in "Lancet," May 13, 1911). Such cells constitute cancer. The unrestrained and unlimited reproduction of epithelial cells and the assumption by them of parasitic properties are the characteristics of cancer. The healthy epithelium has a strictly limited power of reproduction, as is illustrated by a skin-graft. Cancerous epithelium has an unlimited power of reproduction. The alveoli of cancer are distended lymph-spaces filled with proliferating cells. The cells of a cluster are not separated by any stroma, and the walls of the alveoli carry blood-vessels and lymphatics. The growth may have been cancerous from the start, or may have begun as an innocent epithelial tumor which became cancerous. Cancers are always derived from epithelium (of glands, of skin, of mucous membrane, etc.), and if found in a non-epithelial tissue must be secondary, or must have arisen from a depot of embryonal epithelial cells of prenatal origin or from a dermoid cyst lying in the midst of a non-epithelial tissue, or epithelial cells must have been displaced by inflammation or injury so as to be among mesoblastic elements. For instance, the bone does not normally contain epithelial cells. If osteomyelitis arises operation is performed, and a lot of skin may be buried in the bone cavity or an epithelial graft may adhere. Such an epithelial area may become cancerous. Carcinomata have no capsules, rapidly infiltrate surrounding tissues, and are firmly anchored and immovable. In the beginning a cancer is a local lesion, but it soon attacks adjacent tissue and related lymph-glands, and by means of the lymph-cells are carried to other structures, producing secondary tumors and diseases and enlargement of more distant lymph-glands. Finally, lymph containing cancer-cells reaches the blood by the lymph-vessels and passes to distant parts, and secondary tumors or metastatic deposits form. When lymphatic vessels are obstructed, lymph filled with cancer-cells may flow in a direction the reverse of that pursued in health. Widespread or general dissemination may be due to carcinomatous thrombosis of a vein, or perforation of the wall of a vein, multiple emboli forming. Strange to say, emboli composed of cancer-cells may be surrounded with blood-corpuscles and move against the blood-current. A secondary growth (Fig. 178) consists of cells identical in character with and similar in arrangement to those of the parent growth. It may be clinically more or less malignant than the parent growth. The cells of the secondary growth were transported from the primary growth and multiply in their new situation. For instance, the cells of a primary carcinoma of the liver may secrete bile, and the cells of a metatastic area may do the same. Fütterer has reported a case of carcinoma of the thyroid the pulmonary metastases of which secreted colloid. Stewart reported a case of cancer of the lungs and liver secondary to cancer of the pancreas. The secondary growths were of a structure similar to the pancreas and contained trypsin. Metastases from a columnar-celled rectal cancer are composed of columnar cells. Metastases from a squamous-celled epithelioma are composed of squamous cells. In rare cases metastasis of carcinoma of the stomach has occurred in the rectum. Schnitzler reported 11 cases ("Mitth. a. d. Grenzgeb. der Med. u. Chir.," 1908, xix, No. 2).

Fig. 178.—Secondary carcinoma of the submental and submaxillary lymphatic glands following carcinoma of the lip (Senn).

Such a condition is probably due to implantation. Contact cancer has already been referred to (see page 349). We often speak of lymph-nodes enlarging when affected with cancer. The enlargement certainly occurs, but is not due to growth of the cells of the lymph-node. It results from multiplication of the carcinoma cells deposited in the gland. As Henry Morris says ("The Bradshaw Lecture," "Lancet," Dec. 12, 1903), the parenchyma of the involved part does not undergo transition into cancer. After the growth of epithelium has lasted for a length of time the patient becomes poisoned by materials absorbed from the seat of disease, and finally dies from cachexia and exhaustion or some complication. These materials are probably enzymes. During the progress of cancer irregular fever may arise from time to time (see page 351). Cancer is rare before the age of forty, although occasionally it is met with in younger persons. Cancer of the rectum is sometimes met with as early as the twenty-fourth year. I have operated on a woman of twenty-six for cancer of the breast, on a man of twenty-four for cancer of the stomach, and on a man of twenty for epithelioma arising in the old scar of a burn (see Fig. 82). Karsner ("Proc. Path. Soc. Phila.," Feb., 1910) reports 10 cases of carcinoma occurring before the age of twenty-four: 1 was seven; 1, ten; 1, eleven; 1, fourteen; 1, nineteen, 1, twenty-one; 1, twenty-two, and 3, twenty-three. In De la Camp's collection of 9906 cases of cancer only 19 were under twenty years of age. *X*-ray cancer usually occurs in young people. When xeroderma pigmentosum exists in children cancer may arise in areas of the disease. If cancer appears in a young person, growth is apt to be extremely rapid and early recurrence is common. A carcinoma is often the seat of pricking pain; the growth tends strongly to recur after removal; is prone to ulcerate, causing pain, hemorrhage, and cachexia; makes rapid progress, and is often fatal in from one to two and a half years. It is more common in women than in men, and rarely exists in association with tubercle. After a cancer has existed for a time in an important structure, or after a superficial cancer has ulcerated and become hemorrhagic, there are noted in the individual evidences of illness and exhaustion. We speak of this condition as the *cancerous cachexia*, and in it the muscles are wasted, the body-weight is constantly diminishing, the complexion is sallow, the face is sunken, pearly white conjunctivæ contrast strongly with the yellow skin, the pulse is weak and rapid, and night-sweats occur. The above condition is due to the absorption of toxic products from the diseased tissues, which products damage the blood-corpuscles, and also to pain, loss of sleep, deprivation of exercise, malassimilation of food, and perhaps bleeding. It is held that cancer cells contain an enzyme which disintegrates the body cells; hence the wasting. From the disintegrating body cells poisons also come, hence some of the toxemia. Perhaps the materials from disintegrating body cells favor the proliferation of cancer cells. Mental depression is not believed by many surgeons a cause of recurrence. As J. D. Bryant says, it is simply expressive of a condition of nutritive failure which may favor recurrence. We must remember, however, that the great name of Paget is associated with the belief that not uncommon antecedents of the disease are "deep anxiety, deferred hope, or disappointment" ("Lectures on Surgical Pathology," 1863). Victims of rheumatoid arthritis are particularly liable to cancer. Recurrence after operation is due to the growth of cells which were not removed at the operation. Cancer may kill by obstructing a canal, by destroying the functions of a viscus or organ, by hemorrhage, by anemia, by sepsis, or by exhaustion. The duration of life varies in different forms of cancer and in different situations of the growth. After the first symptoms appear, cancer of the gall-bladder, as a rule, causes death in about four months; cancer of the stomach, in less than a year; cancer of the face, in from three to three and one-half years. Billroth's case of carcinoma mastitoides killed the patient in six weeks.

**Serum Reaction in Cancer.**—Kelling in 1906 pointed out that the blood-serum of a cancer patient has a hemolytic action on the red corpuscles of the lower animals. It has been known for some time that the serum of persons affected with certain diseases is able to destroy the red blood-corpuscles of normal individuals; in other words, such sera are hemolytic to the red blood-corpuscles of healthy human beings. The agents in a serum which hemolize the red corpuscles in other sera are called isohemolysins. Isohemolysins are contained in the sera of syphilis, tuberculosis, and cancer. Freund and Kaminer pointed out that normal serum dissolves carcinoma cells, but the serum of a victim of carcinoma does not, and suggested that this fact might be employed diagnostically. In about two-thirds of cancer cases the reaction is positive, but in one-third it is negative. The test is not conclusive and is unreliable. Cobra poison contains hemolysins and has been used as a test for carcinoma (Weil's reaction). The results are positive in about 80 per cent. of cancer cases. Weil pointed out that the serum of a dog suffering from advanced lymphosarcoma destroys the red corpuscles of normal dogs, but is resisted by the red corpuscles of animals that are victims of the same disease. Attempts are being made to utilize the serum reaction for diagnostic purposes. Different observers employ different technic and differ widely in their conclusions. Crile ("Jour. Am. Med. Assoc.," Dec. 12, 1908) is disposed to think highly of the diagnostic value of the serum reaction. Janeway ("Annals of Surgery," May, 1909) obtained positive results in 48.5 per cent. of the 35 cancer cases examined. Janeway's conclusions are wisely cautious. He states that a negative reaction is not proof positive that cancer is absent. A positive reaction makes the existence of malignant disease probable, especially if advanced tuberculosis and syphilis are absent.

The fact that the serum of a cancer patient contains agents destructive to red corpuscles explains the anemia and cachexia of cancer. Janeway points out that the serum of sufferers from benign tumors never exhibits the reaction.

**Some Theories as to Cause of Carcinoma.**—Heredity is discussed on page 347.

1. *Contusion and Irritation.*—As Dennis says, clinical evidence points strongly to the view that inflammatory changes following irritation are responsible for cancer. Individuals with phimosis are particularly prone to cancer of the penis. Those who smoke a short-stemmed clay pipe, which grows hot when in use, are most liable to cancer of the lower lip. In the old days chimney-sweeps often developed cancer of the scrotum, which was always irritated by soot in the cutaneous folds. Cancer of the gall-bladder may arise if gall-stones exist. Cancer of the skin of the hands may arise in *x*-ray workers. Cancer of the skin may be induced by the influence of light (James Nevins Hyde, in "Amer. Jour. Med. Sciences," Jan., 1906). Aniline workers are rather liable to cancer of the urinary bladder. An ulcer may be the irritating focus which leads to the development of cancer at its edge (see Marjolin's Ulcer, page 159). So may a scar. As is well known, certain innocent tumors may become cancerous. The believers in the parasitic theory maintain that irritation and inflammation simply open the gates to the real cause, which they assert is a parasite.

In certain regions of the body, notably the tongue and lip, we regard prolonged chronic inflammation as very apt to eventuate in cancer, and if an ulcerated area is not soon cured by ordinary means we advise operation. A condition persisting in spite of ordinary treatment, prone to eventuate in cancer, but not as yet demonstrably cancerous, is called the *precancerous* stage of cancer. It probably is already cancer, although so early as to lack the positive signs.

A wart is the result of inflammation and a wart may become a cancer.

The edge of a gastric ulcer may become cancerous. Cancer may arise from a scar (see Fig. 85) or the edge of an old ulcer of the skin, the lip, the cheek, or the tongue.

Certain benign tumors tend to become cancerous, especially if irritated by injuries, caustic applications, or inefficient attempts to remove them surgically. Any papilloma and any adenoma may become cancerous. A benign epithelial tumor is always a menace and is to be regarded as a possible or potential carcinoma.

Whereas chronic inflammation or irritation of epithelial structures is not infrequently followed by carcinoma, a single traumatism, as a blow, seldom is. Nevertheless well-established cases are on record of cancer due to a single trauma (Coley, in "Annals of Surgery," April and May, 1911). A woman with cancer of the breast is apt to lay the blame upon a blow, but very seldom can the surgeon regard the blow as causal. In many cases cancer was present when the injury was received, and the injury drew attention to the tumor.

2. *The Inclusion Theory of Cohnheim.*—This theory was set forth on page 346.

3. *The Thiersch Hypothesis.*—This maintains that normal, healthy connective tissue has a restraining influence on the growth of adjacent epithelium; when connective tissue degenerates (as in advancing years or after prolonged irritation) its control over epithelium is weakened, and the epithelium grows more rapidly than it does normally, and from the moment it invades the connective tissue cancer exists. This theory assumes that the connective tissue is a police force and the epithelial cells the criminal class. When the first is weakened or corrupted, the second becomes active and uncontrolled.

Fig. 179.—Epithelioma of right temporal region. Paralysis of right side of face. Papule noticed by patient two years prior to admission.

4. *The Parasitic Theory.*—Various agents have been described as causes, viz., bacteria, protozoa, and yeast fungi.

This theory was discussed on page 348. We do not regard it as proved, and even Plimmer, warm advocate as he is of the theory of contagion, admits that as yet there is no clearly demonstrated case of contagion of cancer from one man to another. I can find no authenticated case on record of a surgeon having been infected by cancer during an operation. Transplantation has been carried out from one animal to another of the same species, although attempts to do so usually fail. Tyzzer, of Harvard, succeeded in nearly 46 per cent. of his inoculation experiments with the Jensen tumor and he has kept up the tumor formation for ten generations. (See "Fourth Report from the Harvard Medical School of the Caroline Brewer Croft Fund Cancer Commission.") It is a serious question, however, if mouse cancer is really cancer at all. Mouse cancer is far more strongly hereditary than human cancer; spontaneous cure is by no means uncommon; metastasis is rare;

the disease may occur as an epidemic in a laboratory. It has been asserted that mouse cancer may revert to sarcoma (Apolant, in "Münch. med. Wochenschr.," 1907, liv), and it may revert to adenoma (Ibid.). These tendencies separate mouse cancer very positively from human cancer. It is said that epidemics of fish cancer may occur in a hatchery. Cancer has not been transplanted from an animal of one species to an animal of another species. If a portion of human cancer is implanted in the tissues of an animal, the cells of the growth retain their vitality for a very few days and then perish. If a piece of mouse cancer is transplanted into a rat the same thing happens. In any case, even a successful transplantation of cells is a very different thing from contagion. The late Prof. Nicholas Senn deliberately implanted a piece of cancer in the tissues of his own forearm without result ("Jour. Am. Med. Assoc.," April 28, 1906). Recently advocates of the contagion theory claim that mouse cancer can be reproduced after transplantation even when the cells in the inoculated matter have been first killed by exposure to the intense cold induced by liquid air (Salim, Moore, and Walker, in "Lancet," Jan. 25, 1908). If these observations should be sustained, they would indicate that the element responsible for growth of a graft is not cellular and might be microbic. Alibert carried out similar experiments and claims to have obtained like results. Most observers believe that transplantation cancer is due to cell transplantation.

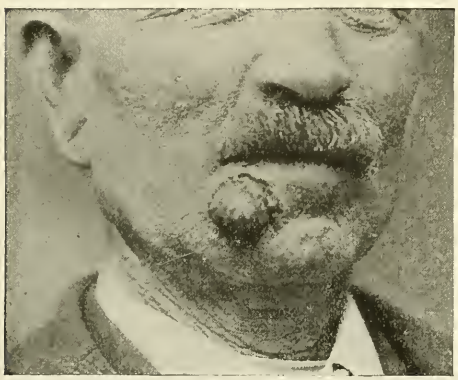


Fig. 180.—Carcinomatous horn.

5. *The Biological Theory.*—In a unicellular organism the function of reproduction is, of course, possessed by the cell. In a multicellular organism certain cells are set apart for the performance of the function of reproduction, but all the cells possess the potentiality for reproduction, but fail to exercise it. If cells undergo atavistic reversion they may again assume the reproductive function. If they do, unrestrained growth will result, and such unrestrained growth is cancer.

N. F. MacHardy ("Lancet," Oct. 24, 1903) states that if a unicellular organism has not sufficient reproductive energy it fuses with another cell and is thus stimulated to produce numerous daughter-cells. In multicellular organisms cells may also fuse, take on active reproductive power, and produce hosts of new cells. When cells are persistently irritated, MacHardy affirms that they become worn out by making repeated attempts at repair, undergo atavistic reversion, and actively assume the power of reproduction. According to this theory cancer is expressive of atavistic reversion of epithelial cells.

**The Prevalence and the Alleged Increase of Carcinoma.**—Crile estimates that at the present time there are probably 80,000 cases of cancer in the United States, and states that in hospital autopsies cancer is found in 1 case out of 12 ("Med. Record," June 6, 1908). In the United States cancer causes 5 per cent. of the annual deaths. Kellogg ("N. Y. Med. Jour.," Sept. 2, 1911) claims that there are 300,000 cancerous people in the United States, that 75,000 die of it each year, and that in 1909, of women who died between the ages of forty-five and fifty-five, 1 in 6 died of cancer. In England in 1909 there were over 34,000 deaths from cancer. In France in 1908 there were over 30,000 deaths from cancer. It has been stated that of persons living

above the age of thirty-five, that 1 woman in 8, and 1 man in 12, will die of cancer (Copeman, quoted by Brand, in "Lancet," Jan. 11, 1908). Is cancer increasing? Of course, the number of cases increases with the increase of population. The apparent death-rate from cancer increases year by year. It is pointed out by W. Roger Williams that in England and Wales the mortality from cancer has increased from 1 to 5646 in 1840 to 1 to 1306 in 1896, and the proportion to deaths from other causes has risen from 1 to 129 in 1840 to 1 to 22 in 1896 ("Lancet," Aug. 20, 1898). Roswell Park comments on the increasing number of deaths from cancer in New York State, and says if it continues for the next ten years the disease will kill more persons annually than phthisis, small-pox, and typhoid combined. Kellogg ("N. Y. Med. Jour.," Sept. 2, 1911) believes that the increase is enormous, and claims that in the United States the disease has increased 500 per cent. in sixty years. Bertillon, of the Statistical Department of Paris, believes that cancer is increasing in all countries. The increase is greater among men than women. Such statements are truly alarming, and yet the reality of all of this apparent increase is doubtful. A part of the apparent increase is due to the greater frequency of exploratory operations for diagnostic purposes, to the greater frequency of postmortem examinations, to more correct diagnoses of obscure internal conditions, and to greater accuracy than was once either usual or expected in filling up death certificates. Neusholme says that just as deaths certified as due to old age grow apparently fewer every year, so other non-specific certifications grow fewer, and cancer gains as they lose. The diminution in infant mortality also causes a relative rise in the apparent cancer mortality. Further, more people than formerly live to reach the cancerous age, and people in general live longer than formerly, and in the later years of life cancer is common. The above facts certainly account for a portion of the alleged increase, but we must also remember that we are curing many more cases by operation than we used to be able to, and hence that the death-rate from cancer is not the real and final measure of the incidence of cancer. The experience of most practical surgeons is that there is a real increase in cancer, but the extent of the increase cannot be ascertained with any accuracy.

**Hereditary Influence.**—This was referred to on page 347. It can be at most only tissue predisposition or a diminution of tissue resistance to the real cause of cancer, whatever that may be. Some previously quoted cases are too impressive to be regarded as coincidences. Williams ("Brit. Med. Jour.," May 9, 1908) points out that 24.2 per cent. of women with cancer of the breast have or had relatives with a history of cancer. Williams states that Butlin's estimate is 37 per cent.; Leaf's, 23 per cent.; and Nunn's, 29.3 per cent.

**Immunity.**—This was referred to on page 347.

It is known that mouse tumors which follow transplantation in some cases retrogress and undergo spontaneous cure, and that animals in which this has occurred are found to have become immune to a re-inoculation of a like tumor. Crile and Beebe present some studies on this interesting subject in the "Journal of Medical Research," June, 1908. Gaylord and Clowes found that the serum of an animal thus rendered immune tends to destroy tumor cells, and experimented with the transfusion of the blood of immune animals into animals with active tumors; 7 animals out of 10 were cured. The blood of an animal naturally immune to tumor inoculation is said to act similarly to that of an animal which has acquired immunity, as shown by retrogression of a tumor.

**Sex.**—Cancer is more common in women than in men. If we leave out of consideration cancer of the uterus and breast, men suffer from cancer more often than women. Men are most apt to get cancer of the lip, tongue, and digestive canal.

**Distribution of Cancer.**—It occurs in all climates and probably all races, although it has been asserted that Eskimos are not liable to it. It is much more common among civilized than barbarous people. It is rarer among the black and yellow races than the white race. The American Indians seldom suffer from it. It occurs in the lower animals far less often than in man, is more common in domestic than in wild animals, and in captive wild animals than in those free and at large. It can even occur in cold-blooded animals. Cancer is most common in the temperate zone. It is usually asserted that the disease is rare in the tropics, but Dudley denies that this statement is true of the Philippine Islands ("Jour. Am. Med. Assoc.," May 23, 1908). Cancer is certainly less common in India than in England, and it is very rare in Greenland. It is almost unknown among the natives of Algeria. It is usually believed that cancer is most prevalent in low and marshy districts. It is less common at high altitudes and among the dwellers on soils of chalk and lime.

**Cancer Regions and "Cancer Houses."**—Some regions show a remarkable frequency of cancer. In Bookfield, New York, during five years nearly 10 per cent. of the deaths were due to cancer.

Tynes (Ibid., March 21, 1908) reports that in Fisherville, Pennsylvania, in 265 families there were 105 deaths, and 18 of them were due to cancer. It is maintained by Haviland and others that certain houses become infected and that cancer appears in such houses again and again among successive families inhabiting them. Such houses are called "cancer houses," and many remarkable facts have been collected relating to them, facts which to some observers seem to prove contagion, but which to others merely serve as interesting examples of coincidence.

Leeson ("Practitioner," Feb., 1909) is of the latter opinion and shows that there was not a cancer house in his district. He studied 248 cases of cancer and all but 4 of them were in different houses. As this author says: "If we are to accept such evidence as that on which the belief in 'cancer houses' is founded, we must believe in 'apoplexy houses,' 'liver houses,' etc."

**Influence of Diet.**—Some blame meat, some tomatoes, etc., for the development of cancer. Vernueil and Reclus, commenting on the fact that carnivora are much more prone to cancer than herbivora, suggested that the increase of cancer during recent years might be due to the increased consumption of meat by the poorer clasess. There is no proof of the truth of this suggestion. In fact, Prof. Senn points out that the Eskimos seem immune to tumor formation, yet they live on an exclusive animal diet and, furthermore, are the healthiest people in the world.

**Arsenic Cancer.**—Sir Jonathan Hutchinson pointed out in 1887 that the administration of arsenic may lead to cancer. Dubreuilh, of Bordeaux, has collected 19 cases ("Annales de Dermatoses," Feb., 1910). It will be highly important to find out if salvarsan can ever be responsible for malignant growth.

**X-ray Cancer.**—A number of *x*-ray operators who worked with the rays soon after Röntgen's discovery developed cancer. An *x*-ray operator in Philadelphia died of carcinoma of the hand. It led to axillary and mediastinal growths. I operated on him twice in vain. Another Philadelphia operator submitted to amputation of the arm and subsequently died. The cells are repeatedly injured by the rays and, finally, normal repair becomes impossible.

**Recurrence After Operative Removal.**—This is usually due to the fact that all of the cells were not removed. It may be due to a new growth. Recurrence may be due to cutting across lymph-tracts and flooding the wound with carcinoma cells, which lodge and grow. The growth of cancerous nodules in the abdominal scar resulting from an exploratory operation for cancer of the stomach is observed every now and then. It is probably due to contact invasion of the scar area, which area has lessened vital resistance to cancer cells.

Murphy thinks that the same explanation holds when the stitch cicatrices become cancerous ("General Surgery," in "Practical Medicine Series for 1909").

**Extension of Cancer.**—It spreads by the lymphatics and rapidly involves the anatomically associated lymph-nodes. In the nodes the migrating cancer cells are imprisoned for a time, and in this incarceration lies the hope of surgery. The adjacent glands are involved much more rapidly than we used to think. They are usually involved within a few weeks of the start of the growth, except in superficial epithelioma, in which cases they are not involved at all.

In a structure devoid of capsule (as the tongue, the mammary gland, etc.) Lockwood points out that involvement of related lymph-nodes is practically immediate.

Lymphatic involvement may result in the formation of a mass much larger than the parent growth. The ducts between the primary cancer and the involved glands are filled with carcinoma cells and their walls become infiltrated. Hence in an operation the ducts should not be cut across or the wound would be flooded with fluid rich in cancer cells. The ducts should be extirpated as well as the glands. To flood the wound with fluid containing embryonic cells is very dangerous, for some of them may adhere, multiply, and reproduce the disease. After a time the capsules of cancerous nodes rupture and periglandular tissue becomes involved. The cells are held in the first glandular stopping-place (the anatomically related glands) for a time, but sooner or later other and more distant glands become involved. In certain abdominal cancers (stomach, rectum, and uterus) the thoracic duct may become obstructed by cancer cells. Large glands may cause much trouble by pressure. When they soften and break down the skin becomes involved and dreadful sores form, oozing foul matter and blood. Death may be due to hemorrhage from a large vessel which has become infiltrated.

Several times I have been consulted by patients on account of glandular enlargements of the neck, the patients never having noticed a small primary lesion in the mouth, and yet the entire glandular disease was secondary to the limited oral trouble.

**Dissemination or Metastasis.**—These terms mean the formation of secondary growths. These growths are formed by small fragments of cancer being broken off and carried to lodgment in distant structures. Such small fragments are called *cancer emboli*. Cancer emboli may be carried by lymph or blood. When cancer emboli lodge in a region favorable to their growth their constituent cells multiply and produce secondary growths. A secondary growth is the histologic counterpart of the parent growth, and an examination of a secondary growth gives us accurate information as to the nature of the primary growth. In cancer of the rectum there may be secondary deposits in the liver or in bone containing structure like rectal glands. In cancer of the stomach secondary nodules in the skin may contain structure resembling the gastric glands. Secondary deposits are by no means as common in cases of squamous-celled cancer as in glandular cancer.

Another method of dissemination is observed in the abdomen. When cancer of a viscus breaks through the peritoneal coat the cells are spread widely by peristaltic movements and peritoneal fluid, and the peritoneum becomes extensively involved. This involvement is a form of contact cancer. Any structure may be the seat of a secondary growth. The lung is frequently affected, so is the liver, so are the bones. Any organ or tissue may become the host for secondary deposits of carcinoma.

**Spontaneous Disappearance of Cancer.**—This is an excessively rare event in human beings, but it does occasionally occur. Gaylord has collected 11 cases which he considers authentic, viz., 2 epitheliomata (1 of the tongue and

1 of the lip), 1 scirrhous cancer of the breast, 1 malignant adenoma of the rectum, and 7 cases of chorion carcinoma ("Seventh Annual Report of the Cancer Laboratory of New York State Department of Health"). The same author also notes the spontaneous disappearance of two sarcomata. Spontaneous disappearance of Jensen tumors successfully inoculated in mice is quite common. It occurred in 23 per cent. of Gaylord's animals. Whereas it is common in mouse tumors resulting from inoculation, it is rare in spontaneous mouse tumors. Bushford finds spontaneous healing in less than 1 per cent. of the latter group. Spontaneous disappearance is not due to the fatty degeneration and necrosis so often found about the center of a carcinoma, but to deprivation of the epithelial cell of some or all of its vitality by an utterly unknown process. Some observers think spontaneous cure is brought about by the stimulation of an immunizing force. When spontaneous cure occurs cancer cells are gradually replaced by scar tissue, and the resulting scar may contain cancer cells immeshed in it. Hence, after apparent retrogression, growth may begin anew.

Fig. 181.—Carcinoma of the auricle.

Besides the apparently positively authenticated cases reported by Gaylord, there are numerous cases on record in which it is highly probable cancer disappeared spontaneously. These cases are collected in the appendix to the previously cited report of Gaylord.

**Blood Changes in Cancer Cases.**—In early cases there is no notable change in either erythrocytes or hemoglobin. In more advanced cases as cachexia begins secondary anemia develops, fall of hemoglobin antedating diminution in erythrocytes.

The anemia may become so profound that it resembles pernicious anemia; in fact, some observers have asserted that pernicious anemia may arise. The anemia of cancer is not benefited by medical treatment.

In gastric cancer, because of vomiting and diarrhea, blood concentration may occur, the red corpuscles being 6,000,000 or even 7,000,000 per cmm.

The leukocytes may be normal, but are often increased. It has been claimed by Macalister and Ross ("Lancet," Jan. 16, 1909) that the blood of a patient with cancer contains a material in its plasma which is an excitant for the leukocytes of healthy persons. As previously stated (see page 384) the serum of the blood of a person with cancer contains agents destructive to the red corpuscles of healthy blood.

**Classification of Carcinomata.**—Carcinomata are classified as follows: (1) Epithelioma; (2) rodent ulcer, or Jacob's ulcer; (3) spheroidal-celled cancer; (*a*) schirrous; (*b*) encephaloid; (*c*) colloid, and (4) cylindrical-celled cancer. Clinically, we speak of *cuirass cancer*, a condition sometimes arising when the mammary gland is cancerous and due to the infiltration of the cutaneous lymphatics with cancer cells; *chimney-sweeps' cancer* and *paraffin worker's cancer*, if either of these occupations seems to have been causative; *cancer à deux*, a phrase used in France to signify that carcinoma has occurred in two persons of a household who are not blood relations, but have been in close contact; *contact cancer*, when cancer appears in an area which was in close

contact with a cancerous area in the same or in another individual—for instance, when a cancer of the upper lip follows a malignant growth of the lower lip; when a carcinoma of the face follows a like growth of the hand; when a cancer appears on the penis of a husband whose wife has cancer of cervix uteri or vagina. A *melanotic carcinoma* is a form of encephaloid in which the cells contain melanin. Scirrhous cancer contains much fibrous tissue and is densely hard. An encephaloid is very soft or brain-like. *Marjolin's ulcer* is an epithelioma which arises from the epithelial edge of a chronic ulcer, a scar, or a sinus (see page 159). Figures 82, 83, 84, and 85 show a Marjolin ulcer arising in the scar of a burn.

*Epitheliomata.*—An epithelioma arises from surface epithelium, and may arise from squamous cells or cylindrical cells, according to the location.

*Squamous-celled epithelioma* (see Fig. 179) takes origin from the skin or from a mucous membrane covered with pavement-epithelium. It is especially apt to appear at the junction of skin and mucous membrane (as the lips) or the point of juxtaposition of different kinds of epithelium. Such a growth may arise in the anus or vagina; on the penis, scrotum, lips, or tongue; in the mouth or nose; on the skin, and other situations. There is an ingrowth of surface epithelium into the subepithelial connective tissue, colonies of cells growing inward and forming epithelial nests. It may arise without discoverable cause, it may follow prolonged irritation, or it may arise in a wart or fissure. In the nipple it is not very unusually, and in the scrotum and nose it is occasionally, preceded by a persistent dermatitis due possibly to psorosperms, and known as *Paget's disease.* Paget's disease is not true eczema, but is rather malignant dermatitis. A crust gathers on the part, and beneath this crust is a raw, red, and moist surface, the edge of which is slightly elevated and somewhat indurated. In the beginning there is a strong resemblance to eczema. The nipple is apt to retract. The parts are the seat of a constant itching and scalding sensation. The area may become cancerous in a few weeks, but may not for years. I have seen two cases of Paget's disease of the glans penis. Squamous epithelioma generally begins as a warty protuberance which soon ulcerates. A malignant or *true cancerous ulcer* (see Fig. 179) has a hard, irregular base, uneven edges, a foul, fungus-like bottom, and gives off a sanious or ichorous discharge. This ulcer is the seat of sharp, pricking pain, sometimes bleeds, and extends over a considerable area, embracing and destroying every structure. Epithelioma usually affects lymphatic glands early, but such infection may be long delayed. Epitheliomatous glands break down in ulceration, making frightful gaps and often causing fatal hemorrhage. Dissemination is not nearly so common as in other forms of cancer, but it does sometimes occur.

*Cylindrical-celled Epithelioma.*—This form of growth takes origin from structures covered with or containing cylindrical epithelium, and it contains cylindrical or columnar cells. It is composed of a stroma of fibers between which lie tubular glands lined with columnar epithelium and containing masses of epithelial cells. Such tumors are found in the uterus and gastro-intestinal tract, and may begin from the surface epithelium or from the cells of tubular glands. In these tumors there is an acinus-like structure and the spaces are filled with proliferating epithelium. Cylindrical-celled cancers may also arise from the mammary gland, liver, and kidney. One of the most common seats of cylindrical-celled cancer is the rectum. Cancer of the rectum may occur at an earlier age than cancer elsewhere, being not uncommon between the ages of twenty-eight and forty. Cylindrical-celled epitheliomata are at first covered with mucous membrane, but they soon ulcerate and involve the submucous and muscular coats in the growth. They may grow rather slowly, usually but not always cause lymphatic involvement, and finally disseminate widely. They require in some regions from five to six years to cause

death. In the rectum, however, growth is much more rapid and few victims of cylindrical-celled carcinoma of the rectum, if unoperated upon, live beyond two years, and many of them die long before this period.

A *rodent* or *Jacob's ulcer*, *epithelioma exedens* or *cancroid* (Fig. 182), was called by the older surgeons *noli me tangere*, because they found that surgical interference (incomplete removal as we now know) was sometimes followed by very active growth. A rodent ulcer is scarcely ever met with except upon the face, though Jonathan Hutchinson saw one upon the forearm, and James Berry met with one upon the arm. It is especially common upon the nose and forehead. It begins after the age of forty as a little warty prominence which ulcerates in the center, the ulceration progressing at a rate equal to the new growth. The ulcer becomes deep; it is not crusted; its edges are irregular, hard, and everted; the floor is smooth and of a grayish color; the discharge is thin and acrid; and the parts about the sore contain numbers of visible vessels. Jacob's ulcer grows slowly, may last for years, does not involve the lymphatics, produces no constitutional cachexia, and is rarely fatal. In some cases, although growth is very slow, destruction eventually becomes very great because of ulceration, there is great loss of tissue and horrible deformity. A rodent ulcer is usually considered to be a malignant epithelial growth which springs from a sweat-gland, a sebaceous gland, or a hair-follicle, but Kanthack asserts that before ulceration the rete and the sweat-glands are normal, but the sebaceous glands are destroyed. The base and edges of the ulcer are hard, which differentiates it from lupus; and, further, the bacilli of tubercle may perhaps be cultivated from the discharge of an area of lupus (see page 247). Rodent ulcer begins below the skin, ordinary epithelioma begins in the skin, and a rodent ulcer contains no cell-nests. A rodent ulcer very rarely undergoes cicatrization, a fact which differentiates it from lupus. Occasionally, but very rarely, a small portion of the growth sloughs out and a temporary scar forms at this point.

Fig. 182.—Rodent ulcer. Case in the author's wards in Philadelphia Hospital.

*Adenocarcinoma or Glandular Carcinoma.*—Glandular carcinomata in structure resemble racemose glands. They consist of a stroma of connective tissue and alveoli filled with proliferating epithelial cells. If the proportion between the fibrous stroma and the cellular elements is about the same as in a normal gland, the growth is called simple. When the cellular element is in excess the growth is soft (medullary), and when the fibrous stroma is in excess the growth is hard (scirrhous).

1. *Scirrhous carcinoma* is a white and fibrous mass which has no capsule, which infiltrates tissues, and which, by the contraction of its outlying fibrous processes, draws in toward it adjacent soft parts, thus producing dimpling, or, as in the breast, retraction of the nipple. It is composed of spheroidal cells in alveoli formed of connective-tissue bands. The commonest seat of

scirrhus is the female breast. It occurs also in the skin, vagina, rectum, prostate, uterus, stomach, and esophagus. It is most frequent in women after forty. It begins as a hard lump which is at first painless, but which after a time becomes the seat of an acute, localized, pricking pain. This lump grows and becomes irregular and adherent, causing puckering of the soft parts. After the skin or mucous membrane above it has become infiltrated ulceration takes place and a fungous mass protrudes which bleeds and suppurates. The adjacent lymphatic glands usually become cancerous in from six to ten weeks, and constitutional involvement is rapid and certain.

2. *Medullary* or *encephaloid carcinoma* is a soft gray or brain-like mass. It is a rare growth, it has no capsule, and it may appear in the kidney, liver, ovary, testicle, mammary gland, stomach, bladder, and maxillary antrum. An encephaloid cancer often contains cavities filled with blood, and this variety is known as a "hematoid" or a "telangiectatic" carcinoma. These growths are soft and semifluctuating, they infiltrate rapidly and soon fungate, and they terminate life in from a year to a year and a half. If the cells of encephaloid become filled with melanin, the condition is called "melanosis" or "melanotic cancer."

3. *Colloid cancer* is extremely rare. It arises from either a scirrhus or an encephaloid, when the cells or the stroma of such a growth undergo colloidal degeneration. On section there will be seen in the center of the growth a series of cavities filled with a material resembling honey or jelly; the periphery is frequently an ordinary scirrhus or encephaloid cancer. Colloid degeneration is most prone to attack carcinomata of the stomach, mammary gland, and intestine. The name colloid cancer is often given to glistening, gelatinous, malignant growths springing from the ovary, testicle, mammary gland, or gastro-intestinal tract. The condition is due to mucous degeneration of the connective tissue or of the epithelial tissue of a carcinoma. Only a portion of the tumor may degenerate or the entire mass may become gelatinous.

*Syncytioma Malignum.*—By this name is meant a malignant epithelial growth arising from the site of the placenta during pregnancy or the puerperal state. It resembles placenta in appearance and rapidly causes metastases by way of the blood-vessels. It is quickly fatal.

**Treatment.**—Cancer is so prevalent, is so dreadful in its nature and inexorable in its progress, tends so strongly to cause death in from two to five years, people are so afraid of it, and so many physicians are hopeless of curing it that multitudes seek relief from the obsessed Christian scientists or from the vulgar criminal quacks. It cannot be too strongly insisted that in the beginning cancer is a local disease curable by early and radical operation, that early diagnosis should be made, and that prompt operation is imperative. Delay is not only disastrous, it is usually fatal. Certainly at least 50 per cent. of the cases of cancer I see are beyond operation when they are first brought to the hospital, they having sacrificed the golden moments during which cure was possible. Carcinomata demand early and wide excision, with removal of implicated glands. Anatomically related lymph-nodes must be removed even if they show no evidence of involvement. If operation is early and thorough, and if certain regions are involved, a considerable proportion of cases can be cured. Carcinomata of the lip, the skin, and the mammary gland can often be cured. The operation must be radical. That the tumor is small and recent is no justification for minimizing the extent of operation. That is the sort of case which may be cured by radical removal. Anything short of radical removal is bound to fail. Recurrence almost certainly means that cancer cells have been left behind. Unless a wide area is removed cancer cells are sure to be left. During removal the parts should be handled as little as possible so as not to squeeze malignant cells into the wound. Cancer cells in a wound soon become

fixed and multiply. For the same reason tumor and glands are removed in one piece. The surgeon avoids cutting across lymph-vessels and planting cancer cells in the wound. After operation the *x*-rays should be used in hope of destroying cancer cells which may have remained in the tissues. To use the rays lessens the danger of recurrence. Czerny ("Deutsche Med. Wochen.," Nov. 2, 1912) is so impressed with the necessity of special effort to prevent recurrence that he leaves the operation wound open for two or three months, uses radium, the *x*-rays, or fulguration in the wound, and then, if everything seems to be going on well, he closes the wound by a plastic operation. A recurrent growth may be removed as a palliative measure to lessen pain and to relieve the patient from ulceration and hemorrhage, but such an operation is rarely curative. If a growth does not recur within five years after removal, a cure has very probably been attained; in fact, if there is no recurrence within three years, the case is probably cured. The three-year limit has been usually accepted since Volkmann's paper on the subject. A rodent ulcer should be excised or else be curetted and cauterized with the hot iron or the Paquelin cautery. In cancer of the lower *lip*, remove the growth by Grant's operation (*q. v.*), or, if necessary, cut away the entire lip. In every case remove the glands beneath the jaw. In cancer of the *tongue*, excise this organ and also the lymph-nodes from beneath the jaw and in the anterior carotid triangles. In cancer of the *breast*, remove the breast, the pectoral fascia and the pectoral muscles, and take away the fat and glands of the axilla. In cancer of the *rectum*, if near the surface, excise the rectum from below; if above 5 inches from the anus, do the sacral resection of Kraske and then remove the growth. In cancer of the *esophagus*, perform gastrostomy; in cancer of the *pylorus*, perform pylorectomy or gastroenterostomy; in cancer of the *bowel*, do resection, side-track the diseased area by an anastomosis, or make an artificial anus; in cancer of the *penis*, amputate the organ and remove the glands of the groin.

**Treatment of Inoperable Cancer.**—Erysipelas toxins are seldom of any service in carcinoma. In very rare cases they do good. It is justifiable to try them. Claims have been made that cancer can be benefited by the injection of material expressed from carcinomatous tumors. There are suggestions of the value of such treatment. The late Dr. Hodenpyl had charge of a case in which a cancer of the liver was undergoing spontaneous retrogression. When a cancerous individual was injected with ascitic fluid from this case a local reaction was observed in the tumor (swelling, redness, diminution in size, necrosis). It was assumed that the fluid contained a specific material which might cure cancer. The results of the study of such fluid have been negative (Richard Weil, "Jour. Med. Research," August, 1910). Serum from animals suffering from cancer is without therapeutic value. Pyoktanin, thiosinamin, trypsin, and many other materials have come upon the scene as remedies. They were like plausible confidence men, but each was soon exposed. There is no drug and no serum at present known to be capable of curing cancer. Honest investigators have at times been lead into error by forgetting that at times the rate of growth of a cancer may temporarily lessen or that growth may for a time actually cease. (See Lewis S. Pilcher's address before the Surgical Society of Brooklyn in Feb., 1909.)

Fulguration has of late excited much interest. By this term we mean bombarding a part from a metal electrode with sparks flashed from a high-tension current. It was introduced as a treatment for cancer by De Keating-Hart in 1907. The sparks have no specific action on cancer cells. All the surface cells of an ulcerated growth are converted into an eschar and the connective tissue under and around the sore is stimulated to cicatrize. Sometimes after fulguration healing occurs over cancerous nodules. Fulguration cannot act through the unbroken skin. It is not a real cure, though it may retard the

progress of a case or aid in preventing recurrence after extirpation of a growth. Application of sparks may be followed by grave or fatal sepsis. The early enthusiasm for fulguration in cancer has largely waned. No operable growth should be treated by it, as, at best, it has only a local effect and cannot act upon the involved lymph-nodes. The *x*-rays are of distinct value in certain cases of carcinoma. Surface growths may be apparently cured, although, unfortunately, they are apt to return even after total disappearance. Deeper growths are seldom lessened in size and practically never cured, but the rate of growth may be diminished and pain be abated or abolished. The knife is to be preferred to the *x*-rays, except in very superficial skin cancer about the eyelid or nostril, and in inoperable cancer. The real curative power of radium is as yet undetermined. The *x*-rays and radium have a decided influence in lessening the horrible pain of recurrent or inoperable cancer. In lymphatic recurrence after operation thyroid extract may perhaps be of some value. In some cases of recurrent cancer ligation of the artery of supply or extirpation of the artery, as suggested by Dawbarn, notably retards growth. I have been able to confirm this statement. In cancer of the breast, oöphorectomy occasionally produces great benefit (Beatson's operation). In inoperable cases palliative operations may be justifiable to relieve some urgent discomfort or get rid of a foul or bleeding mass. Gastro-enterostomy, gastrostomy, and colostomy are palliative operations. In a malignant growth of the nasopharynx tracheotomy may be required, and in a malignant growth of the bladder it may be advisable to perform suprapubic cystostomy. In an inoperable case it may be necessary to relieve the pain by opium, giving as much as may be required to secure ease. Opium so used seems not only to relieve pain, but to retard the growth of the tumor and to favor the development of fibrous tissue in the stroma.

**Chemotherapy of Cancer.**—Wassermann has been able to cause mouse cancer to disappear by injecting into the veins of the animal negrosin, and also by injecting eosin and selenium. This proves that a chemical, as well as a parasite, may have an affinity for certain cells. The chemicals used by Wassermann attacked the cells of mouse cancer, and did not attack or only slightly influenced the other cells of the body. The world is seeking for a chemical agent to destroy cancer.

Wassermann desired to introduce selenium into the cancer cells. He found eosin (an aniline dye) to be the helping hand to put selenium into the cells. When the pink solution of eosin-selenium is thrown into the blood of a mouse the skin at once becomes pink. The pink color of the skin disappears in a few hours; the tumor remains deeply stained. Numerous injections will kill the mouse or cause the tumor to disappear, and the action is just as positive in spontaneous as in transplanted tumor.

This material is too dangerous to use in man, but the investigation suggests splendid possibilities for the future (E. F. Bashford, in the "Lancet," Jan. 13, 1912).

**Malignant Growth from Congenital Pigmented Mole** (Fig. 183).—As previously stated, the embryonic origin of the pigment-producing cells is uncertain. Some regard a malignant growth of a congenital pigmented mole as epithelioma, others as alveolar pigmented sarcoma. Bloodgood thinks it is probably carcinomatous.

Malignant change seldom occurs before the fiftieth year, the growth rarely attains a large size, metastasis takes place very rapidly by the blood and lymphatics, and the patient seldom lives more than a year after malignant change begins. (See Bloodgood, in "Progressive Medicine," Dec., 1907.)

Because of the danger of subsequent malignant change it is wise to remove large pigmented moles. Every pigmented mole which is irritated or begins to

enlarge must be removed, and the associated lymph-nodes should also be removed. Bloodgood knows of no definite cure on record of a malignant tumor arising in a pigmented mole. Prevention is easily secured by extirpation before the onset of malignancy.

**Cystomata.**—A cystoma is a benign cystic tumor in which the cells of the cyst wall constitute the new growth. The cyst contents are derived from the cells of the wall. The tumor is the cyst wall; the cells of this wall are derived from the epiblast, the hypoblast, or the mesoblast, and are either epithelial or endothelial. The cells of the cyst wall adhere to connective tissue which seems to constitute a part of the wall. A thick wall contains much connective tissue, a thin wall very little. The nature of the contents is dependent on the character of the cells which constitute the tumor. Cysts lined by endothelium contain serous fluid; a cyst of the thyroid gland usually contains colloid material; a cyst lined by flat epithelial cells contains matter resulting from fatty degeneration, etc.

Fig. 183.—Melanotic growth, secondary to pigmented mole.

Cystomata may be congenital or acquired, and an acquired cystoma may arise after injury or follow inflammation. The cyst may increase in size progressively or its growth may be halted. The wall may become calcareous or even bony. When a cyst has one cavity, we call it monolocular; when there are several or many cavities it is called multilocular.

**Varieties of Cystomata.**—The chief varieties are: Traumatic epithelial; atheromatous; mucous; mesoblastic.

**Traumatic Epithelial Cystomata.**—These growths have been called traumatic dermoids. Such a growth may arise after an injury which carries and deposits epithelial cells or a bit of skin deep into the connective tissue. For instance, a punctured wound of the hand may be followed by an epithelial cystoma (Fig. 185). It may arise after a scalp wound or in the scar of a burn. The cyst grows only to a certain size and then remains stationary. It is lined by pavement-epithelium and it contains products of the fatty degeneration of epithelial cells.

*Treatment.*—Extirpation of the wall of the cystoma.

**Atheromatous Cystomata.**—These growths, according to Senn, are met with particularly in the ovaries, in the orbital region, and at the base of the tongue, but they can arise almost anywhere. They may remain small or may attain a great size. Such a cystoma contains columnar epithelial cells which have undergone fatty degeneration and sometimes contains oil. An atheromatous cystoma is deep seated and is not connected with the skin, in contrast to a sebaceous cyst, which is superficial and is a part of the skin. An atheromatous cystoma is lined with epithelium, but not with skin. A dermoid cyst is lined with skin or other definite structure. An atheroma is due to the displacement of a mass of epithelial cells, which mass was the matrix of the cystoma. "The displacement of the matrix of an atheroma occurred at a time prior to the differentiation of the epiblastic cells into the organs representing the appendages of the skin, while the matrix of a dermoid cyst points to

a later displacement of the matrix" ("Pathology and Surgical Treatment of Tumors," by Nicholas Senn). Atheromatous cystomata may be congenital, but may not appear until puberty or even much later.

*Treatment.*—Extirpation of the wall of the cystoma.

**Mucous Cystomata.**—A mucous cystoma, like an atheromatous cystoma, is due to the displacement of epithelium, but in the former condition it is pavement-epithelium and in the latter it is columnar epithelium. The latter is filled with a mucoid material and the former with a fatty débris. Such a mucous cystoma must not be confused with a retention cyst of a mucous membrane. Mucous cystomata are found particularly about the lips, mouth, and pharynx. They rarely attain any considerable size. Cystomata lined with ciliated epithelium may arise in the testicle, the liver, and the brain.

*Treatment.*—Incise, cauterize, and drain. The wall is so delicate that excision is rarely possible.

**Mesoblastic Cystomata.**—They are lined with endothelial cells. They contain serous fluid, often grow to a large size, and sometimes disappear spontaneously. Mesoblastic cystomata are probably distended lymph-spaces. They are congenital and are most common in the neck, axilla, and perineum.

Fig. 184.—Hydrocele of neck in boy nine weeks of age.

In one case seen by the author such a cystoma of the neck appeared late in life, but it is probable that it had existed in childhood, and after disappearing for a long time had reappeared. The most common form of mesoblastic cyst is known as *hydrocele of the neck* (Fig. 184).

*Treatment.*—Excision is very difficult. In 1 case in which I assisted Professor Keen it was successfully accomplished. The usual treatment is to tap frequently, after each tapping washing out with carbolic acid (2 to 5 per cent.) and applying pressure.

Cystomata of bone, of the thyroid gland, of the mammary gland, etc., are considered in the sections on Regional Surgery.

**Teratomata.**—The teratomata contain tissues or higher structures derived from two or all of the blastodermic layers. The tumors we previously considered are derived from only one of these layers. The elder Senn, in his work on "Tumors," thus defines a teratoma: "A teratoma is a tumor composed of various tissues, organs, or systems of organs which do not normally exist at the place where the tumor grows. The highest type of a teratoma is a fetus in fetu. In the simpler varieties the tumor is composed of heterotopic tissue, such as bone, teeth, skin, mucous membrane, etc. All teratoid tumors are congenital; that is, the tumor either exists at the time of birth or

the patient is born with the essential tumor matrix. A teratoma never springs from a matrix of postnatal origin." Any human structure may be found in a teratoma. Various fetal malformations belong to this group, as do also double monsters, in which one of the embryos is rudimentary. Teratomata are divided into *external* and *internal*. To the external teratomata belong the parasitic fetus and the suppressed fetus. A *parasitic fetus* is the result of fusion of two embryos, one having gone on to complete development, and the other developing partially, and obtaining nutrition from the fully developed embryo to which it is attached. A *suppressed fetus* is an irregular mass attached to the posterior surface of the sacrum, to the chest, or to the abdomen. It contains a conglomeration of tissues and fragments of organs, for instance, bone, cartilage, lung tissue, kidney tissue, a piece of intestine, or a portion of liver. In a case pictured by Sir J. Bland-Sutton a leg projects from the sacral region.

An *internal teratoma* may be found within the cranium, chest, abdomen, or pelvis. The internal teratoma consists of a conglomeration of the tissues and visceral fragments of a suppressed fetus, but, unlike the external teratoma, it is surrounded by a cyst wall. The members of this group most often seen by the surgeon are *dermoid cysts*.

**Dermoid Cysts.**—These cysts were first studied and described by Lebert. The name "dermoid" implies that the cyst contains skin, and it does contain skin or mucous membrane, the chief mass of the tumor being derived from proliferation of the cells of a portion of displaced epiblast or hypoblast, but it also contains mesoblastic derivatives. There are two varieties of true dermoid: *sequestration dermoid* and the *tubulodermoid*. In this section we speak of the first form. The second form is considered on page 401. A superficial dermoid is formed by the inclusion in mesoblastic tissues of a portion of the epidermis or mucous membrane. Superficial non-traumatic dermoids are situated in regions where the blastodermic layers were in contact. A deep dermoid is formed from a collection of epithelial cells completely separated from the epiblastic tissue from which they originated. When a cyst originates from epiblastic cells so immature that the skin appendages have not as yet been formed, it will contain only atheromatous material like that found in a sebaceous cyst. When a cyst arises from epiblastic cells after they have so matured that the appendages of the skin have been formed, it will contain atheromatous matter, sweat, sebaceous matter, and hair. The first form is known as an *atheromatous cystoma;* the second, as a dermoid. A deep-seated dermoid may contain also such structures as prove it must have taken origin from "a displaced matrix representing different tissues and organs" (Senn). Such a dermoid may contain portions of organs, bone, cartilage, and teeth. A dermoid cyst may be defined as a heterotopic cyst, the wall of which is composed of connective tissue lined with epithelium and containing material formed by the proliferation of epithelium and often hair, teeth, or even bone. An injury may displace a bit of epithelium and lodge it in connective tissue and from this a traumatic dermoid may arise (Fig. 185). Traumatic dermoids are not true dermoids. Garrè called them *traumatic epithelial cysts*. They are most often encountered in the palmar surface of the hand or fingers. The skin above such a cyst is not adherent to it and often a scar is visible. The cyst wall is composed from without inward of connective tissue and epithelial cells, the stratum corneum being the inner layer (Leo Buerger, in "Annals of Surgery," August, 1907). The cyst contains desquamated epithelium and often cholesterin. The causal injury is usually a puncture, but may be a laceration, a contused wound, or a bite. Sometimes a cyst arises in the track of a healed sinus. Pietzner collected reports of 73 cases ("Ueber Traumatische Epithelcysten. Dissert. Rostock.," 1905).

True dermoid cysts are most commonly found in the ovary and in regions where, during bodily development, the blastodermic layers come in contact; for instance, in the neck, the eyelids, the orbital angles, the lumbosacral region, the root of the nose, and the floor of the mouth. Such cysts are also found in the ovary, testicle, brain, eye, mediastinum, lung, omentum, mesentery, and carotid sheath.

A dermoid of the lumbosacral region may be mistaken for a spina bifida. Sarcoma may form from the connective-tissue elements of the wall of a dermoid cyst. A dermoid cyst may become cancerous, or innocent epithelial tumors may originate from the cyst lining. The epithelial cells may become fatty and an oil-cyst may actually form. If the cyst epithelium was derived from mucous membrane, mucus may gather in the sac. A dermoid cyst may inflame or even suppurate. It is free from pain unless it suppurates, inflames, or develops into a malignant tumor; it grows slowly and rarely attains any considerable size unless it arises in the ovary. A subcutaneous dermoid may or may not fluctuate. It is not in the skin as is a sebaceous cyst, but the skin can be moved over it. A sebaceous cyst moves with the skin. Subcutaneous dermoids about the orbit are adherent to the under-

Fig. 185.—Traumatic dermoid cyst.

lying periosteum. The matrix of a true dermoid is congenital, but the cyst often does not appear until puberty or later. Teratoids and dermoids connected with the rectum require special consideration (see page 1165).

*Treatment.*—Complete extirpation. If any of the epithelium of the cyst-wall is left, the cyst will re-form. A superficial dermoid should be removed in the same manner as a sebaceous cyst, and if it is adherent to underlying periosteum the portion of this membrane to which it adheres should also be removed. A deep dermoid ought to be removed as a tumor would be, if operation is feasible.

**Branchial Cysts and Fistulæ.**—When a branchial cleft fails to become completely obliterated, a branchial cyst may form. The branchial clefts are the analogues of the gill-slits of a fish. There are four of these clefts on each side of the neck. They are called clefts, but they are really grooves, and each groove on the skin has its counterpart in the mucous membrane of the pharynx. Each pharyngeal groove is covered with hypoblastic epithelium; each cutaneous groove is covered with epiblastic epithelium, and the two grooves are separated by mesoblastic structures. When the sides of a cleft do not unite and an opening forms in the mucous membrane, a *complete branchial fistula*

(*complete congenital cervical fistula*) results. When the sides of a cleft fail to unite and, although the mucous membrane is not perforated, the skin does not cover the cleft, a *branchial sinus* or an *incomplete branchial fistula* (*incomplete congenital lateral cervical fistula*) results. When the sides of a cleft toward the pharynx fail to coalesce, a *pharyngeal diverticulum* is produced. When the pharyngeal surface and the cutaneous surface both close, but the deeper part of a cleft remains open and epithelial cells are caught in mesoblastic elements, a *branchial cyst* is formed. Sinuses are more common than complete fistulæ or cysts.

The essential cellular element of a branchial cyst is epithelium, derived either from the skin or pharynx; hence the branchial cyst is not a true dermoid, because its histologic elements are derived from only one of the blastodermic layers. Branchial cysts are most common in the triangle of election of the left side. They are round, smooth, often fluctuating, and are very deeply situated, being in close relation with the great vessels. Some cysts contain mucus, others serous fluid, others fatty débris. An abscess may form. The origin of a cyst or fistula is usually from the second branchial cleft, but I have operated on two cysts of the first branchial cleft, and my colleague, Dr. Nassau, also operated on one of like origin. There may be one, two, or three openings of a fistula. Not unusually the openings are bilateral. Hereditary tendency is often manifest. The cutaneous openings are always along the anterior margin of the sternocleidomastoid muscle. At the external orifice there is often an irregularly shaped bit of skin and cartilage, which is called a *cervical auricle*.

*Treatment*.—In old children and in adults it may be possible to extirpate a cyst or a fistula, although this is very difficult and often impossible. Other methods employed are incision, cauterization with the Paquelin cautery, and packing with gauze; frequent tapping and injection with iodin; incision and drainage, every antiseptic care being observed. In all young children and in some older persons with deep cysts the latter plan is the only one advised, and it will often fail, but may sometimes produce a cure.

**Cysts.**—A cyst is a cavity, abnormal or pathological in character, lined by a membrane and containing material usually fluid or semifluid. It is necessary to bear in mind the distinction between a cystoma and a cyst. Hektoen and Riesman, in the "American Text-Book of Pathology," insist on this distinction. They say: "A *cystoma* is a true tumor, arising from active proliferation of a matrix destined to form cystic spaces; whereas a cyst is a secondary formation not primarily due to tissue proliferation." Cysts are divided into the following classes: Retention-cysts; cysts from softening; tubulocysts; and parasitic cysts (Ibid.).

**Retention-cysts.**—A retention-cyst is formed by blocking of the duct of a gland or by failure in the absorption of the proper amount of the secretion of a ductless gland. A few characteristic forms of retention-cysts will be described.

**Sebaceous Cysts (Wens).**—A sebaceous cyst arises when the excretory duct of a sebaceous gland is blocked by dirt or occluded by inflammation. The orifice of the duct is often visible as a black speck over the center of the cyst. They are very common in the scalp, being known as *wens*, and upon the face, neck, shoulders, and back. Arising in the skin, and not under it, the skin cannot be freely moved over a sebaceous cyst. A sebaceous cyst is lined by epithelium and is filled by foul-smelling sebaceous material. A sebaceous cyst may suppurate. When a cyst ruptures and the contents become hard, a *horn* is formed. Another form of horn has been previously alluded to as due to horny transformation of a wart.

*Treatment*.—To treat a sebaceous cyst, incise the portion of skin above it and dissect the sac entirely away by scissors or a dissector, trying to not

rupture the delicate wall. If even a small particle of the wall is left, the cyst will re-form. If it ruptures during removal and it is feared that some portion may remain, cauterize the interior of the wound by pure carbolic acid. If acid is not used, close without drainage; but if acid is used, drain for twenty-four hours. If an abscess forms in a sebaceous cyst, open it, grasp the edges of the cyst lining with forceps, dissect out this lining by scissors curved on the flat, cauterize by pure carbolic acid, and drain for twenty-four hours.

**Mucous Cysts.**—A mucous cyst is due to the blocking of a mucous gland or a mucous crypt. Mucous cysts occur particularly in the mucous membrane of the mouth and genito-urinary organs, and are filled by thick, adhesive mucus containing numerous epithelial cells. Such a cyst is of spherical outline, and the epithelial membrane which lines it is strongly adherent to tissues beyond.

*Treatment.*—Incision, curetment, cauterization by pure carbolic acid, and packing or extirpation of a considerable part of the cyst, and curetment and cauterization of the part remaining.

**Oil Cysts.**—An oil cyst is due to fatty degeneration of the epithelial lining of a sebaceous cyst, or a milk cyst of the breast. As previously noted, a dermoid may become an oil cyst.

*Treatment.*—Extirpation, as for sebaceous cysts.

**Salivary Cysts.**—A retention-cyst of a salivary gland is known as a *ranula* (*q. v.*). These cysts are most common in the submaxillary or sublingual gland.

Fig. 186.—Dr. Weatherby's case of multiple sebaceous tumors of the scrotum.

**Lacteal or Milk Cysts (Galactoceles).**—Such a cyst occasionally arises in the mammary gland during lactation, and is the result of blocking of a lactiferous duct (see Cysts of Mammary Gland).

Among other forms of retention-cysts, most of which are discussed in special sections of this book, we mention *hydrosalpinx*, a cyst due to blocking of a Fallopian tube; cysts due to obstruction of the bile-ducts (the most common form is known as *hydrops*, which is a dilated gall-bladder the result of obstruction); cyst of the thyroid gland; cyst of the pancreas; and *hydronephrosis*, a condition produced by obstruction of the ureter.

**Cysts from Softening.**—These cysts are formed by the disintegration of degenerated tissues. For instance, after a hemorrhage into the brain, softening may follow and a cyst arise. Cystic changes of this sort are frequently observed in sarcomata and carcinomata. A cyst from softening has a wall of connective tissue, but there is no endothelial or epithelial layer.

**Tubulocysts.**—This name was given by Sir J. Bland-Sutton to cysts formed in certain remains of embryonal ducts, which vestiges ought to have been destroyed in the developed body. A small cavity is left unobliterated, and in this space fluid gathers. The source of the fluid is usually the lining cells of the cavity. Branchial cysts are frequently considered under this heading. Among the commoner tubulocysts are cysts of the vitello-intestinal

duct, cysts of the urachus, and thyroglossal cysts. Thyroglossal cysts and sinuses are considered on page 926.

Mesenteric cysts, not hydatid and not due to carcinoma, are embryonic developments from remains in the mesentery of the vitelline duct, the Wolffian ducts, the Wolffian bodies, and the Müllerian ducts (Moynihan).

What are called "chyle cysts" of the mesentery are embryonal cysts placed in such close adjacency to lacteals that chyle enters into them (E. P. Baumann, in "Lancet," May 7, 1904).

**Cysts of the Vitello-intestinal Duct.**—Such a cyst presents itself as a small, bright red, globular mass, which appears to arise from the umbilicus of a baby or a young child, and which usually has a distinct pedicle, but may be sessile. A cyst of this character forms when the vitello-intestinal duct atrophies from the gut toward the umbilicus, but a remnant at the umbilicus escapes obliteration, and from this remnant a cyst forms. The wall of such a cyst contains unstriped muscular fiber and is lined by mucous membrane. Occasionally the duct in the process of involution is not destroyed—its caliber is simply lessened—and the duct remains open in the navel and feces come from it. If the duct fails of obliteration at the intestinal end, a diverticulum remains at this point (*Meckel's diverticulum*).

*Treatment.*—A pedunculated cyst at the navel is treated by ligating its base and dividing the stalk beyond the ligature. A cyst with a thick base is dissected out. The surgeon must be careful to avoid confounding an umbilical hernia with a cyst of the navel.

**Urachal Cysts.**—The urachus is the obliterated allantois, and is a cord running from the summit of the bladder to the umbilicus. This structure is in the middle line of the abdomen and in front of the peritoneum. A portion of the allantois may not be obliterated at birth, and in consequence of this failure a cyst forms. It grows to a considerable size, may push the peritoneum away and reach the pelvis, may communicate with the bladder, may break through the umbilicus, or grow backward toward the spine.

*Treatment.*—Extirpation of the lining membrane, partial closure of the cavity by suture, and packing the unobliterated part. Complete extirpation of the cyst is seldom attempted. W. R. Weiser ("Annals of Surgery," Oct., 1906) collected 86 cases of cyst of the urachus. In 8 of these complete extirpation was performed, and Macdonald has since reported a successful complete extirpation (Ibid., August, 1907).

**Parasitic cysts** are due to the development of certain parasites in the tissues. The form most often encountered is known as hydatid cysts.

**Hydatid cysts** are especially common in Iceland, and are frequent in Australia and South America, but are very rare in the United States. In the United States 91 per cent. of cases occur in foreigners (Lyon). Hydatid cysts are due to echinococci. The adult echinococcus is the tapeworm of the dog (Tænia echinococcus), and its ova or larvæ gain access to man's body by accompanying the food he eats and passing into the alimentary canal, from which situation they are transported to various organs by the blood. Osler says the embryo (which has six hooklets) burrows through the wall of the bowel and enters the peritoneal cavity or muscles; it may enter the portal vessels and reach the liver, or may enter the systemic circulation and pass to distant parts. The danger depends on two factors: "the situation and the liability of the cyst to suppurate" (Sidney Coupland). The organs most usually attacked are the liver and lung. In 60 per cent. of cases the liver suffers, and in 12 per cent. the lung (Thomas). Lyon estimates that the liver is the seat of disease in 73 per cent. of cases. Cysts sometimes arise in the intestine, genito-urinary passages, brain, or spinal canal. When the embryo lodges, the hooklets disappear and a cyst is formed. This cyst is composed

of two layers, an outer capsule (cuticular membrane) and an inner layer (endocyst). The cyst contains clear saline fluid. As the cysts grow, daughter-cysts bud out from the wall of the mother-cysts, the structure of the daughter-cysts being identical with that of the mother-cysts. From the lining membrane of all the cysts, after a time, growths arise known as scolices, which represent the head of the echinococcus and exhibit four sucking disks and a row of hooklets (Osler).

The fluid is not albuminous, is occasionally saccharine, is thin and clear, and may contain scolices or hooklets.

A hydatid cyst may calcify, may rupture, or may suppurate. These cysts are very firm, but usually fluctuate. Palpation with one hand while percussion is practised with the other gives a persistent tremor (*hydatid fremitus*). If the cyst can be safely reached, some fluid should be drawn and examined for diagnostic purposes. When a cyst suppurates, positive constitutional and local symptoms arise. Hydatid cysts of the brain and cord tend to produce death in the same manner as do tumors. A cyst of the liver may rupture into the pleural sac, into the belly cavity, into the stomach, or into the bowel, producing shock, hemorrhage, and probably death. In rare cases hydatid cysts rupture into the pericardium or into a great abdominal blood-vessel, or externally. Rupture into the bile-passages is usually followed by suppuration of the cyst. Suppuration of a cyst may follow uncleanly tapping. It has been pointed out that eosinophilia is noted in most persons suffering from hydatid disease.

*Treatment.*—An unruptured hydatid cyst of a superficial structure should be incised and the sac wall should be dissected out. Hydatids of the brain have been successfully removed in Australia. A cyst of the kidney is removed through a lumbar incision. Omental cysts should be radically removed if possible; if this is not possible, open the abdomen, surround the cyst with gauze, evacuate through a trocar, stitch the cyst wall to the wound, incise, irrigate, and drain by gauze. Bond advocated evacuating the cyst, closing it by sutures, and dropping it back in the abdomen. Gardner says tapping is dangerous, as it may cause rupture of the cyst. In a hydatid of the liver the abdomen should be opened, the cyst should be surrounded by gauze pads, and tapped by a trocar and cannula. When the cyst is emptied of fluid it is grasped by forceps and pulled to the incision in the abdominal wall; it is sutured to this incision, the trocar opening is enlarged, and the endocyst is removed by irrigation.[1] This operation is called *marsupialization.* If the cyst is on the summit of the liver, it may be reached by a transpleural hepatotomy. If aspiration is performed to settle a diagnosis, operate at once after doing it, because of fear that the cyst may leak and disseminate the disease throughout the peritoneal cavity. If hydatid fluid is disseminated throughout the peritoneal cavity, it may or may not lead to the development of new cysts, but it is almost certain to cause a febrile condition accompanied by urticaria and known as *hydatid toxemia.* Brewer ("Annals of Surgery," April, 1908), in operating on a case of hydatid cyst of the liver, wounded the portal vein and was obliged to tie it. The patient recovered. That there was no failure of nutrition Brewer attributes to the fact that the vein had been long pressed upon and the collaterals were dilated when ligation was performed.

[1] John O'Conor, of Buenos Ayres, in "Annals of Surgery," May, 1897.

## XIX. DISEASES AND INJURIES OF THE HEART AND VESSELS

**Rupture, Wounds, and Injuries.—Rupture.**—The heart may rupture and cause instant death, but rupture may not be instantly fatal. Curtin reported a case in which death did not occur for over twenty-four hours. Elsner reported a case of rupture in which life was prolonged for ten days. One case lived eleven days. In cases in which death does not occur rapidly the rupture must be so small that very little blood escapes. Rupture occurs in a damaged heart, a heart in which the muscular fiber is fatty, is fibroid, or is necrotic from suppuration. It may be traumatic, resulting from a fall or a blow upon the chest, or non-traumatic, following a great effort or strain. If death does not at once take place the pulse becomes very rapid, there is precordial pain, dyspnea, cyanosis, feeble heart-sounds, rapid respiration, great restlessness, collapse and syncope, and the development of a triangular area of dulness on percussion. Positive diagnosis is impossible. Meyer collected 36 cases of rupture of the heart reported since 1870. Death occurs from accumulation of blood in the pericardium. This acute compression of the heart due to blood escaping from the heart is called **heart tamponade.** It is held by Franke, of Berlin, and others that in heart tamponade the pressure within the pericardium comes to exceed the pressure within the auricles, that the pressure within the pericardium causes the symptoms of the injury, and finally death. Some would treat heart tamponade by puncture or aspiration. It would seem that either must be useless, as fresh blood is bound to replace what is withdrawn. Suturing must fail in non-traumatic cases because of the badly diseased myocardium. In traumatic cases it may possibly succeed.

**Wounds of the Pericardium and Heart.**—Severe wounds usually, though not always, produce death, but slight wounds may not prove fatal. It is a popular impression that the expression "stabbed to the heart" is another way of saying that instant death has occurred. This view was accepted even by surgeons during many centuries. During the sixteenth century sportsmen found now and then bullets and arrow-tips healed in the heart walls of animals they had slain. At this time the famous case of a duelist was published by Paré: a man received a sword thrust in the heart, but was able to run after his opponent many hundred feet before falling down in death. (See "An Experimental Investigation of the Treatment of Wounds of the Heart," by Charles A. Elsberg, in "The Journal of Experimental Medicine," Sept. and Nov., 1899.) From Paré's time until our own it has been recognized by surgeons that a wound of the heart does not of necessity produce immediate death, and may even be recovered from.

In 1867 G. Fisher published a study of 452 cases of wound of the heart, and pointed out the surprising fact that from 7 to 10 per cent. of such cases recover. In more recent years Rosenthal, Block, Del Vechio, and others proved by animal experimentation not only that cardiac wounds are not of necessity instantly fatal, and that in some cases they may be recovered from, but that the suturing of such wounds is possible and greatly enhances the chance of recovery. L. L. Hill ("Med. Record," Nov. 29, 1902) shows that although 90 per cent. of heart-wounds are penetrating, only 19 per cent. are immediately fatal. Sudden death occurs when Kronecker's coördination center is damaged. Several times during postmortem examinations on human beings healed scars have been found upon the heart. The heart has been punctured a number of times accidentally or intentionally, and death has not ensued. John B. Roberts,[1] of Philadelphia, suggested in 1881 that it would be proper to try to suture wounds of the heart.

[1] The author, in "Progressive Medicine," vol. i, 1899.

**Symptoms.**—A wound of the heart causes hemorrhage, which is usually copious, but owing to the interlocking of muscular fibers the hemorrhage may be slight. Bleeding may take place into the pericardial sac in some cases where the pericardium has been injured and the heart has escaped. Such an injury is occasionally inflicted by the sharp end of a fractured rib. The wound is rarely at or near the apex of the sac. In most heart wounds the pleural cavity is also opened and severe hemothorax occurs. The lung may or may not be injured. A wound of the pericardium or heart causes profound shock, irregular or very weak pulse, sighing respiration, dyspnea, and, it may be, the signs of hemopericardium, pneumopericardium, or hemothorax. In hemopericardium splashing sounds are heard with the heart-beats and the heart-sounds are very feeble. In pneumopericardium there is a tympanitic percussion-note in the area which should exhibit the cardiac dulness. There may or may not be serious external bleeding. Fatal concealed hemorrhage may occur. Pain is constant, and attacks of syncope are the rule. The position of the wound and the evidences of hemorrhage may aid in making the diagnosis. Death is apt to occur suddenly from shock, hemorrhage, inability of the heart to contract because of the severed fibers, or inability of the heart to dilate because of the pressure of blood in the pericardial sac (heart tamponade). If a wound of the pericardium or heart does not cause death during the first day or two, inflammation follows (traumatic pericarditis or carditis) and the patient may die of suppurative pericarditis or of empyema.

**Treatment.**—Wounds of the pericardium and heart should be sutured. We should explore if, from the location of the wound and the symptoms, we suspect a cardiac wound. I agree with Vaughan, that if there is a wound in the cardiac region and if the symptoms threaten life, exploration should be performed at once. In a doubtful case exploration should be made by enlarging the wound, and this may be done under local anesthesia. In operating for a heart-wound the cutaneous surface should be rapidly disinfected, and every effort be made to antagonize shock during the operation. The patient should be wrapped in hot blankets and surrounded with hot bottles or hot-water bags, or should be placed upon a table composed of pipes in which hot water circulates. The foot of the bed should be raised. Hot saline fluid containing adrenalin chlorid should be infused into a vein, or, in desperate cases, into an artery. The extremities, except the one selected to infuse salt solution in, should be bandaged (autotransfusion), an enema of hot coffee and whisky should be given, and atropin should be injected hypodermatically. It is wiser, in most cases, to give a general anesthetic than not to give it. Local anesthesia is slow and unsatisfactory. Without an anesthetic the patient will probably struggle, and struggling is very dangerous, as it loosens clots and permits hemorrhage to begin again (L. L. Hill, in "Med. Record," Sept. 19, 1908). Chloroform is the anesthetic used. If the patient is unconscious and the corneal reflex is abolished, no anesthetic should be given. The heart is exposed by resecting several ribs. In a knife-wound of the right pleural cavity and right side of the pericardium, Barth, of Danzig, removed 1 inch from each of three right costal cartilages (fifth, sixth, and seventh), close to the side of the sternum, and removed also the ensiform cartilage and 1 inch of the sternum. The same surgeon, in the case of a man stabbed in the fourth left intercostal space, removed the fourth and fifth left costal cartilages and part of the sternum ("Deutsche Zeitschrift für Chirurgie," Bd. lxix, No. 1). Schwerin, of Berlin, in a stab-wound of the chest exposed the heart by resecting the fourth and a portion of the fifth left ribs ("Proceedings of German Surgical Congress," 1903). Wilms ("Centralblatt f. Chirurgie," Leipzic, vol. xxxiii, No. 22), in a case of gunshot-wound, obtained access to the anterior and posterior surfaces of the heart by a simple intercostal incision. Parrozzani makes a trap-door in the

chest, the hinges of the door being the rib cartilages. In exposing the heart Giordono enters along the wound, removing any obstacles that intervene. It is needless to try to avoid opening the pleura if a flap with an internal hinge is used; it has usually been opened by the accident, and in any case can very seldom be avoided. Matas advises Spangaro's intercostal incision. The mammary vessels are tied and the width of the intercostal space is greatly increased by strongly retracting the ribs and cartilages. If more space is needed, the incision is carried upward at the junction of the cartilages and sternum. The heart is exposed, clots are removed from the pericardial sac, and the sac is irrigated with hot saline fluid. The bleeding may be furious. A non-penetrating wound of the ventricle may bleed so profusely during systole as to resemble a penetrating wound (Sherman). A penetrating wound may bleed most during diastole. The motions of the chest make manipulation difficult. It is wise to insert two traction sutures in order to lift the heart toward the operator. A wound in the heart is sutured with interrupted sutures of catgut, which are passed by means of a round, curved needle, and if a cavity of the heart is open each suture includes the whole thickness of the heart wall except the endocardium. It has been said that the sutures should be tied during diastole, otherwise they are apt to cut out, but Profs. Gibbon, Stewart, and Nassau tell me that in their cases such a procedure was impossible because of the very rapid action of the heart. As few stitches are used as will efficiently close the wound. Numerous stitches cause extensive degeneration of muscular fiber and stitch-holes may permit leaking. The pericardium is sutured with silk or, as was done in 1 successful case (Rehn), the sac is packed with iodoform gauze. It is not absolutely necessary to drain the pericardial sac. Clots are removed from the pleural sac by irrigation with hot saline solution, pulmonary bleeding is arrested by the suture or by packing, and a wound in the lung, especially if it communicates with the air-passages, is sutured if the patient's condition justifies prolonging the operation.[1]

After such an operation the patient is in great danger and every effort should be made to save him from shock. In performing operations upon the heart the pleura is almost always opened, and if it is open there is always pneumothorax and grave danger of pulmonary collapse and overwhelming shock. It is a great advantage in such cases to have at hand an apparatus which will prevent or amend pulmonary collapse (see page 896).

Frazier ("Progressive Medicine," March, 1913) collects 218 cases of operation for heart-wounds. The mortality in the series was 55.5 per cent. This estimated mortality is probably much too low. Many operators have reported a single successful case each. It is reasonable to believe that many unsuccessful cases have not been reported. It is eminently desirable to have the reports of a number of consecutive cases. We have here such a record. Hesse (Bruns's "Beiträge," 1911, lxxv) reports 21 cases with a mortality of 71 per cent. We believe this represents about the average mortality of heart-wounds subjected to operation. In 1881 Dr. John B. Roberts, of Philadelphia, suggested that heart-wounds should be sutured. In 1887 Dr. Harvey Reed sutured a wounded pericardium and the patient recovered. In 1891 Dalton, of St. Louis, obtained recovery by a similar operation. The first operation on a wounded human heart was performed in 1896 by Farina, of Rome. The patient had been stabbed in the right ventricle. The wound was sutured, but he died of pneumonia on the sixth day. Rehn, of Frankfort, in 1896 sutured a wound of the heart and packed the pericardium with gauze, and the patient recovered. Among others reporting cases are Cappelan, Peyrot, Williams, Barth (in this case the internal mammary artery was also injured), Wilms, Hill, Sherman, Harte, Gibbon, Stewart, Guinard, Sultan, Cumming and Beattie, Wolff,

[1] The author, on "Suture of the Heart," in "Progressive Medicine," 1899, vol. i.

Picqué, Lenormand, and Parrozzani. There have been 12 cases of heart suture in Philadelphia and 7 recoveries. Stewart had 5 cases and 3 recoveries; Gibbon, 2 cases and 1 recovery; Nassau, 1 case, which recovered. I have never operated for a wound of the heart. According to Hill, the right ventricle is most often, the left auricle least often, injured; wound of the auricle is generally considered to be more dangerous than wound of the ventricle; and wound of the apex is less dangerous than either. Peck points out, however, that there are 11 reported cases of auricle wounds with 4 deaths, a mortality of 36.3 per cent., while the general mortality of heart-wounds is about 64 per cent. ("Annals of Surgery," July, 1909). Wounds of the left ventricle give a better prognosis than of the right ventricle; wounds of the right auricle a better prognosis than of the left auricle. A needle puncture rarely causes serious bleeding from a ventricle, but is very apt to cause severe bleeding from an auricle. A wound received during diastole is less dangerous than one received during systole. Wounds of the right heart bleed more than wounds of the left heart. Wolff points out that ligation of one coronary artery can be done and recovery follow; wounds of the left ventricle give the best prognosis because the wound is closed by thick edges of muscle. In 37 operations for heart-wounds the left pleura was opened, in 3 the right pleura, and in 2 the pleura was uninjured. In bullet-wounds death usually occurs before operation can be done (Wolff, "Deutsche Zeitschrift für Chirurgie," Bd. lxix, No. 1). Bircher reports a case of gunshot-wound of the heart in which there was no operation, yet the patient recovered. He believes that a gunshot-wound by a small bullet should not be operated upon, but he would operate for wounds by large bullets and for stab-wounds ("Arch. klin. Chir.," 1912, xcvii). Manteuffel reported 7 cases, occurring during the war in Manchuria, in which recovery followed gunshot-wounds of the heart which were treated expectantly. Without operation the mortality of heart-wounds will be at least 90 per cent.; with operation it will be about 60 to 70 per cent.

Matas ("Southern Med. Jour.," August, 1908) discussed 160 cases of heart-wounds with 43.83 per cent. of recoveries. In 134 cases the wound was sutured, with 49 recoveries. In 11 cases it was exposed, but was not sutured, and 5 recovered. In 5 cases foreign bodies were removed with success.

In Peck's table ("Annals of Surgery," July, 1909) there are 69 wounds of the right ventricle with 48 deaths (69.6 per cent.); 74 of the left ventricle with 45 deaths (60.8 per cent.); 5 of the left auricle with 2 deaths (40 per cent.); 6 of the right auricle with 2 deaths (33.3 per cent.); and 7 miscellaneous cases with 5 deaths (71.5 per cent.), a total of 160 cases with 58 recoveries and 102 deaths, a mortality of 63.7 per cent.

The immediate dangers of the operation are hemorrhage, shock, and the entrance of air. The late dangers are pericarditis, empyema, and pneumonia (Vaughan). Traumatic carditis or pericarditis is treated in the same way as idiopathic cases. Pus in the pericardial sac should be evacuated by resection of the fourth left costal cartilage and incision of the pericardium (von Eiselsberg's case). Pool reviewed the subject of heart-wounds in the "Annals of Surgery," April, 1912.

**Pericarditis** is an infectious condition that may be traumatic or non-traumatic. If pericarditis follows an open wound, it is obvious how the infection must have entered; if it follows a bruise or a contusion, the injury has rendered the pericardium a point of least resistance. In some few cases, which are known as primary pericarditis, it is impossible to determine how the micro-organisms gained entrance. The ordinary form appears as a complication of certain infectious diseases, such as septicemia, pneumonia, rheumatism, and tuberculosis. It may be secondary to some adjacent infection, such as an empyema. A tuberculous abscess may break into the pericardium,

and an abscess, even from a considerable distance, may burrow into it. It may arise secondary to a distant infection, as a suppurating wound, osteomyelitis, middle-ear suppuration, abscess of the mastoid, tonsillitis, abscesses anywhere, peritonitis, and gastric ulcer. It sometimes follows gastro-enterostomy and may arise in an individual with Bright's disease. In a recently born child infection of the stump of the umbilical cord may cause pericarditis. A pericardial effusion in a newborn child is invariably purulent and in a young child it is usually purulent. In children the condition is usually associated with pulmonary disease (Poynton, in "Brit. Med. Jour.," August 15, 1908). A great variety of bacteria may be responsible for pericarditis. The exudation may be serofibrinous; this is an evidence of its being a mild infection, and such an exudate may undergo absorption. On the other hand, the exudate may become purulent, and in such a case cure will never be obtained by absorption of the pus. In pericarditis there is usually some pain in the region of the heart, and this pain is apt to extend into the left arm. Epigastric pain is a common symptom. The heart is overacting, the heart-sounds are indistinct, the pulse is strong and very rapid, there is an increased area of cardiac dulness, and the patient complains of dyspnea. The temperature is elevated and a double friction-sound may be made out upon auscultation.

**Treatment.**—Ordinary pericarditis, without pus-formation or extensive effusion, is managed by the physician; but when there is extensive effusion with symptoms of dangerous compression it is advisable to open the pericardium, and if there is purulent effusion the pericardium must be opened. The procedure usually practised in the past to relieve pericarditis with marked effusion was puncture or aspiration. This, however, is dangerous. The heart is not, as was formerly taught, pushed back and up by the pericardial effusion, but is lifted upward and forward, and may be pushed to the right or left if there are adhesions between the pericardium and heart; and it is impossible to select any place for aspiration that assures us that there will be no danger of puncturing the heart. A coronary vein may be injured, the pleural cavity may be entered, and a dry tap is usual from blocking of the needle. Brentano has shown that tapping cannot completely empty the sac. Many surgeons, however, do not fear puncture, and explore by inserting a fine needle in the fourth or fifth space of the left side close to the sternum. In cases of extensive pericardial effusion, and also in cases of suppuration within the pericardium, I believe that *pericardiotomy* should be performed. An inch or more of the cartilage of the fourth rib of the left side should be removed or 2 inches of the fourth rib itself, and the pericardial sac should be exposed and, after exploratory puncture, formally incised. In this operation it may be necessary to tie the internal mammary artery. In pyopericardium the pleural cavity is very seldom invaded, because the pleural space in front of the pericardium has usually been obliterated by the spread of the inflammation. The pericardial sac is opened as directed above, is cleared of purulent material and fibrinous masses by irrigation, the edges of the pericardial wound are sutured to the edges of the superficial wound, and gauze drainage is introduced. Incision is safer and more certainly curative than aspiration; for whereas aspiration might be curative in pericardial effusion, it cannot be so if the effusion is purulent. In 41 cases of purulent pericarditis (Roberts's table of 35 cases and Ljunggren's 6 cases) operated upon, 16 recovered. Local anesthesia is safer than general anesthesia.

**Phlebitis, or Inflammation of a Vein.—Acute Phlebitis.**—Phlebitis may be *plastic* or it may be *infective.* Plastic phlebitis, while occasionally due to rheumatism, to gout, to advanced phthisis, to a febrile malady, or to some other constitutional condition, usually takes its origin from a wound or other injury, from the extension to the vein of a perivascular inflam-

mation, or, in the portal region, from an embolus. Varicose veins are particularly liable to phlebitis. When phlebitis begins a thrombus usually forms (see Thrombosis, page 184) because of the destruction of the endothelial coat of the vessel, and this clot may give rise to emboli, may be absorbed, or may be organized. An aseptic clot organizes and the vein becomes permanently narrowed or blocked. A septic clot is apt to soften and break up. In the lower extremities *paraphlebitis* is common with slight involvement of coats, and a clot may not form. Clot-formation causes edema. Infective phlebitis is a suppurative inflammation of a vein arising by infection, perhaps from suppurating perivascular tissues (*infective thrombophlebitis*), perhaps from the blood-stream or in the portal system, perhaps from infective embolism. It is not unusually met with in cellulitis or phlegmonous erysipelas, may arise in the lateral sinus as a result of mastoid suppuration, or in the liver from appendicitis or from phlebitis of the rectal veins. Sometimes as the convalescence from pneumonia begins, phlebitis due to pneumococci arises. If a septic thrombus forms, the vein wall suppurates, is softened and in part destroyed, and the infected clot softens and gives rise to emboli. No bleeding occurs when the vein ruptures or is opened, as a barrier of clot keeps back the blood-stream. The clot of suppurative phlebitis cannot be absorbed and cannot organize. Septic phlebitis causes pyemia, and the infected clots are disseminated.

*Postoperative phlebitis* of the iliac, femoral, or saphenous veins is not uncommonly the result of a mild or attenuated infection, toxins in the blood probably attacking the vein. As a rule, the toxins are non-pyogenic. It may follow an abdominal operation when there is no evidence of infection. According to Cordier, it occurs in 2 per cent. of abdominal operations. It is called, as is the like puerperal condition, *milk-leg*, or *phlegmasia alba dolens*. Nearly always the femoral vein is the one which suffers. Strange to say, it is most apt to attack the left iliac, femoral, or saphenous veins; it matters not upon which side the operation was performed. In over 90 per cent. of cases the left femoral or left saphenous veins are attacked (Cordier, in "Jour. Am. Med. Assoc.," Dec. 9, 1905). One theory regards the pressure of the right common iliac artery upon the left common iliac vein as a predisposing cause. Another theory attaches importance to the pressure of a loaded sigmoid. It is most common in anemic subjects, especially when anemia results from blood loss. It may be due to toxins damaging the inner coat of the vein, but feeble circulation is a powerful factor in its production. I believe, with Clark, that powerful traction on the sides of an abdominal wound may be responsible for it (see Thrombosis after Abdominal Operations, page 187). Vanderveer reported 4 cases in which sepsis was positively absent ("American Medicine," July 13, 1901). It occurred in the left iliac vein of a woman on whom I had operated for carcinoma of the left breast six days before. There was no obvious infection of the wound. I have seen it occur in the left iliac vein after an interval operation for appendicitis. Phlebitis may arise in the vein of one extremity, a clot may form, and this may be absorbed or may organize. The other extremity may be involved afterward or simultaneously (horseshoe form). It may come on seven or eight days after operation, many days, or several weeks. It usually is ushered in by chilly sensations or slight chills and elevated temperature. There is always pain in the limb. The pain may be dull or acute; it is made worse by motion and by pressure over the involved vein. The entire extremity swells from edema. It is probable that marked edema signifies associated lymphangitis or an extensive clot running into many venous branches. The edema is seldom characterized by very distinct pitting on pressure. The skin is white and looks stretched and shiny. The inguinal glands are usually enlarged. Sensation is impaired except over the vein. Paresthesia is common. The involved veins come to feel like cords to the touch.

In mild cases the symptoms disappear in a few days. Severe cases continue for several or many weeks or even months. Involvement of the deep veins causes prolonged swelling. There is seldom any disposition to gangrene.

In some cases there is extensive muscular atrophy, in some there are trophic disturbances, in some muscular contractures. Postoperative phlebitis is sometimes responsible for embolic pneumonia and cerebral embolism. Many postoperative pneumonias are due to this cause.

The *symptoms* of plastic phlebitis are pain, tenderness in and around a vein, discoloration over it, and edema below the seat of the disease. Suppurative phlebitis, besides these conditions, causes the constitutional symptoms of pyemia (see page 197). Any thrombus, if it loosens, forms emboli. It is said that the clot resulting from pneumococcic phlebitis forms so rapidly that it adheres slightly and is peculiarly apt to loosen and give rise to emboli (Pierre, in "Gazette des Hôpitaux," Sept. 3, 1904). Septic thrombi are apt to cause septic pneumonia.

Fig. 187.—Varicose veins.

The *treatment* of plastic phlebitis of an extremity comprises rest in bed for from four to six weeks, slight elevation of the part, the use of cold for the first twenty-four hours, and then the application of external heat and a flannel bandage. If the patient is gouty or rheumatic, appropriate remedies should be given. A clot does not always form in an inflamed vein, but if one forms there is danger of embolism; hence massage and both active and passive movement are dangerous until the clot becomes firm. When a vein is involved in a suppurative process and septic thrombophlebitis exists, we should operate when the situation makes operation possible. Ligate the vein (compress a sinus by packing) above and below the clot, open the vessel, and wash out the infected clot, or, if dealing with an accessible vein, extirpate the involved portion. This plan of treatment is always to be applied in infective thrombophlebitis of the lateral sinus and of the internal saphenous vein. The constitutional treatment is that of pyemia.

**Chronic Phlebitis.**—This rare condition is known as *phlebosclerosis*, and it is a chronic inflammation of the wall of a vein, producing a fibrous change in the vascular coats. It may arise in a part the seat of chronic venous engorgement, but its most frequent cause is syphilis. It is often associated with arteriosclerosis.

**Varicose Veins; Phlebectasis, Phlebectasia, or Varix** (Figs. 187 and 188).—**Definition and Causes.**—Varicose veins are unnatural, irregular, and permanently dilated veins which are elongated and pursue a tortuous course. This condition is very common, and 20 per cent. of adults exhibit it in some degree in one region or another. Some facts indicate hereditary predisposition. In over 80 per cent. of cases the trouble begins before the age of twenty-five. The causes of varicose veins are said to be obstruction to venous return and weakness of cardiac action, which lessens the propulsion of the blood-stream. A. Pearce Gould says obstruction is not a cause, because in pregnancy varicose veins may be seen early, before the

womb is much enlarged. The real cause is probably a predisposition to the growth of vein-tissue, which leads to valve failure and a regurgitation of blood from the deep veins into the superficial venous channels (A. Pearce Gould, in "Lancet," March 1 and 15 and June 7, 1902). As Billroth said over thirty years ago, sudden obstruction causes edema and gradual obstruction a free collateral circulation. Neither sudden nor gradual obstruction causes true varicosity unless the veins are predisposed by a tendency hereditary or acquired.

Varicose veins may occur in any portion of the body, but are chiefly met with on the inner side of the lower extremity, in the spermatic cord, and in the rectum. Varix in the leg is met with most commonly during and after pregnancy and in persons who stand upon their feet for long periods. It is especially common in the long saphenous vein, which, being subcutaneous, has no muscular aid in supporting the blood-column and in urging it on.. The deep as well as the superficial veins may become varicose. Verneuil maintained that varix of the superficial veins is almost always secondary to varix of the deep veins, a radical view which seems improbable. It is certain, however, that after contusions of the leg it is not unusual for the deep veins to become filled with clot and for the superficial veins to dilate notably. By the term "*caput medusæ*" is meant dilated veins radiating from the umbilicus. The veins of the esophagus may become varicose, and this malady is commonly unrecognized clinically. Varicose veins are in rare instances congenital, but they are most often seen in the aged, and usually are first observed between the ages of twenty and forty. They are more common in women than in men, owing, it is believed, to the influence of pregnancy.

Fig. 188.—Varicose veins.

Varix of the spermatic cord is known as *varicocele.* It is apt to appear about the time of puberty, and most adult men have at least a slight varicocele. Varix is more likely to appear in the left spermatic vein than in the vein of the right side, because the left spermatic vein has no valves (see page 1400).

Varicose tumors of the rectum constitute *hemorrhoids* or *piles.* Piles are caused by obstruction to the upward flow in the hemorrhoidal veins, either by obstructive liver disease, enlargement of the uterus or prostate, or the presence in the rectum of fecal masses in a person habitually constipated (see page 1177).

A vein under pressure may dilate more at one spot than at another, the distention being greatest back of a valve or near the mouth of a tributary.

The valves become incompetent and the dilatation becomes still greater. Callender has pointed out that varix of the lower extremity is apt to begin where the deep vessels join the superficial veins. At this point Treves says three forces meet: the blood column above, the valve below, and the force of the blood-current. At the spot where the pressure is greatest the vein wall dilates, and from this dilatation the blood-current is deflected and causes another dilatation higher up and on the opposite side of the vessel. The blood is again deflected and causes another dilatation, and so on. The vein wall may become fibrous, but usually it is thin and sometimes it ruptures. The veins not only dilate, but they also become longer, and hence do not remain straight, but twist and assume a characteristic form. It seems probable that the first step in the process is a growth of new venous tissue (A. Pearce Gould), and then follow lengthening, tortuosity, incompetence of the valves, and dilatation of the vessel.

Delbet[1] points out that varicose veins of the leg, which begin in the thigh, result from valvular incompetence; varicose ulcers arise from variations of venous pressure due to valvular incompetence. The incompetence of the valves does harm by allowing the intravenous pressure to equal the pressure in the arterioles, a condition which arrests capillary circulation, causes congestion, and greatly lowers tissue resistance. Incompetent valves also favor ulceration by developing a *vicious venous circle*, first described by Trendelenburg. Blood passing through this circle loses nutritive elements. Trendelenburg has described the vicious circle as follows: Blood in the saphenous vein flows toward the periphery instead of toward the center, because of incompetent valves—it passes into the veins which connect the superficial veins with the deep veins and then enters the tibial and peroneal veins. It passes from the tibial and peroneal into the popliteal and femoral veins, and some blood leaves the femoral vein and again enters the saphenous.

The skin over varicose veins in the leg is often discolored by pigmentation due to red blood-cells having escaped from the vessel and broken up. The tissues around a varicose vein become atrophied from pressure, and it is not unusual to meet with a very large vein whose thin walls are in close contact with skin. In this condition rupture and hemorrhage are probable. When the vein wall forms a pouch-like dilatation the condition is spoken of as a *vein cyst*. Varicose veins are apt to inflame, and thrombosis frequently occurs. When a thrombus forms, emboli may be broken off and carried into the circulation, especially if the patient walks about. The formation of emboli is not nearly so common as a result of thrombosis in a varicose vein as in thrombosis in an undistended and unelongated vessel. In varicose veins of the thigh, however, the chance of embolism following thrombosis is much greater than when the veins of the leg alone are involved. In some elderly people thrombus actually effects spontaneous cure. When a thrombus organizes, more or less calcification is apt to ensue, and a *vein-stone* or *phlebolith* is formed. After middle life many varicosities remain stationary or cease to give trouble. The chief complications of varicose veins of an extremity are thrombosis, edema, violent hemorrhage from rupture, phlebitis, eczema, and chronic ulceration.

**Treatment of Varix of the Lower Extremitiy.**—The treatment of varix of the leg may be *palliative* or *curative*, but whichever plan is followed, the surgeon should endeavor first of all to remove the exciting cause. An essential part of palliative treatment is to attend to the general health, to keep up the force and activity of the circulation, and to prevent constipation. Massage is useful, especially alcohol frictions, if eczema is absent. *Cold* baths are always forbidden (Bennett). The patient should exercise regularly in the

[1] Delbet, "Sem. méd.," Oct. 13, 1897.

open air and should lie down for a time, if possible, every afternoon. Instead of lying down for a time during each day, he may sit down and elevate the legs, resting them on a table, and thus assuming a position supposed to be peculiarly American. If there is no pain, distinct discomfort, or edematous swelling, a support is unnecessary, but if these conditions exist it is needed. If a support is required in varix of the leg, use a flannel roller or a perforated rubber bandage applied over a long stocking. Such a bandage supports the veins and drives the blood into the deeper vessels, which have muscular support. The use of a rubber pad filled with glycerin and applied over the saphenous vein, so as to support the blood column and act as a valve, has been recommended. A purely local varix should be excised, because there is always danger of injury, and consequently of hemorrhage or thrombosis. If the superficial veins have dilated because of thrombosis of the deep veins and edema exists, ligation or excision is contra-indicated, as its performance might lead to permanent edema. In such a case it would perhaps be proper to incise the deep fascia the length of the thigh, place the vein beneath it, and suture the fascia. The vein would then be supported, but not blocked. If the disease involves the leg only, operative treatment is rarely required and may even do harm. Such cases are operated upon if there are cyst-like dilatations, if thrombi form, and, as Bennett points out, if a thin-walled vein crosses the tibia, and is thus exposed to the danger of injury and thrombosis.[1]

If the leg is involved in the process, and the saphena in the thigh is also varicose, operation should be performed.

If a thrombus forms in a superficial varicose vein, tie the vein above and below the clot, divide the vessel in two places, and remove the vein and the clot within it. Thrombosis of a varicose vein is not so apt to lead to emboli as thrombosis in a non-varicose vein, but it may do so, and the condition has some elements of danger.

If edema is marked, and increases in spite of properly applied bandages, etc., it probably signifies clot-formation, and the patient should remain in bed until this question is determined. Hemorrhage from a ruptured varicose vein of an extremity is usually readily arrested by compression and elevation.

The radical treatment of varix of the leg often does good, often relieves some annoying condition, but rarely absolutely cures (W. H. Bennett). There are several methods of operation: ligation with excision of part of the vein, exposure and ligation of the vein below the saphenous opening, or circular incision around the leg. (See Operations Upon Vessels.)

**Nevus.**—(See Tumors.)

**Arteritis,** or inflammation of an artery, is *acute* or *chronic.*

**Acute Arteritis.**—Slight inflammation is by no means unusual, but severe arteritis is decidedly rare. It may follow direct injury or arise secondarily to a perivascular inflammation. An artery is very resistant to the spread of inflammation, but we sometimes encounter suppurative arteritis in a suppurating area. Arteritis may arise in the course of an infective malady, being produced by bacteria, but it is also found in intoxications, and is then due purely to toxins. It may occur in the eruptive fevers, in influenza, typhoid fever, acute rheumatism, gout, syphilis, diphtheria, septicemia, and septic intoxication. Ford points out that acute arteritis developing during acute or chronic infections is particularly apt to arise in the lower extremities (Ford, "Thèse de Paris," 1901). Toxins or bacteria usually reach the artery in the main blood-stream, but may be lodged in the vessel wall by the lymph or the flow in the vasa vasorum. The inner coat of a portion of an artery becomes lined with inflammatory exudate and the coats are infiltrated with small cells. Often parietal thrombi form. Sometimes, though rarely, the vessel is com-

[1] W. H. Bennett, "Lancet," Oct. 15, 1898.

pletely blocked by thrombosis. In acute suppurative arteritis pus accumulates in the arterial wall, a clot forms in the lumen, and the coats of the vessel undergo necrosis and give way. Violent hemorrhage may thus arise, but often, in thrombo-arteritis as in thrombophlebitis, rupture does not cause hemorrhage. Acute arteritis, if non-bacterial in origin, is usually recovered from with slight structural change. Infective arteritis is recovered from if the causative germ is not very virulent or if the toxin is not present in excessive quantity. Acute arteritis may terminate in arterial obstruction with or without gangrene, permanent dilatation, arterial rupture, or chronic arteritis.

The *symptoms* may be merged with those of an acute or chronic intoxication or infection, or with those of a local perivascular inflammation. In arteritis arising during infections the symptoms appear abruptly and the onset is marked by great pain. Ford studied 18 cases in influenza. He says it attacks particularly persons over thirty years of age, occurs in one leg or both, arises most commonly during convalescence, but may not begin until the individual is apparently well. There is pain and tenderness over the vessels, low surface temperature, paresthesia, and mottled skin (Ford, "Thèse de Paris," 1901). The artery may be obstructed, and if a large vessel is blocked, the pulse below the clot is lost. The block may be temporary or persistent. Gangrene may follow. Ford points out that if the artery only is blocked, the gangrene is dry; but if the vein also is occluded it may be moist. I have seen 2 cases of dry gangrene following influenza.

*Treatment.*—Secure rest in bed; elevate the extremity slightly, relax it, smear the skin over the inflamed vessel with ichthyol ointment or mercurial ointment, or follow Ford's advice and use methyl salicylate or an ointment of salicylic acid, turpentine, and belladonna. Wrap the part in cotton and surround it with bottles or bags filled with warm water. If a patient is very restless, a splint must be used. It may be necessary to give morphin for pain, and any infection or toxemia must be combated with appropriate remedies.

If gout, rheumatism, or syphilis is regarded as causative, proper remedies must be given. It is most important to maintain the secretion of the kidneys. If abscesses form in a septic case, they must be opened and drained. If a large artery of one of the lower extremities becomes occluded, raise the foot about 2 inches from the bed, wrap the foot and leg in cotton-wool, apply a flannel bandage from the toes up, and surround the limb with bags of warm —not hot—water. Hot water would take more blood to the region of the block than could be distributed. If gangrene occurs, amputation is necessary.

**Chronic Endarteritis (Arteriosclerosis, Atheroma, Arteriocapillary Fibrosis, Cardiovascular Degeneration).**—By these terms we mean thickening of the walls of the arteries, limited in area or widespread, due to inflammation or degeneration of the middle coat, the media undergoing hypertrophy and the intima fibrous hyperplasia (Wm. Russell, "Brit. Med. Jour.," June 4, 1904). Atheroma is used to designate the disease when it attacks the large vessels and is characterized by advanced degeneration. Chronic endarteritis is due to increase of blood-pressure. Hypertension precedes sclerosis and causes it. Hypertension is detected and measured by the sphygmomanometer. Increase of blood-pressure means increase of arterial tension, because the lumen of the vessels is lessened and the heart works more strongly to urge the blood along, and finally hypertrophy of the middle coat occurs. The persistence of arterial contraction which causes increase of blood-pressure may be brought about by kidney disease, hard work, violent strains, heart disease, care and anxiety, worry and mental strain, alcoholic or venereal excesses, habitual gluttony, syphilis, gout, rheumatism, lead-poisoning, diabetes, and acute infections like typhoid fever and influenza. It may arise in an old man who has not suffered particularly from any of the above-named causes, or may occur prematurely

from toxemia or heredity. It is a true saying of Cazalis that "A man is as old as his arteries," and a young man debilitated by syphilitic disease or alcohol may have diseased arteries, and hence be really older than a healthy man of sixty. Heredity may be commonly traced in heart disease due to diseased coronary arteries, and cerebral hemorrhage due to disease of the middle cerebral arteries. The aorta, of all vessels, is most prone to suffer. The large vessels are more apt to be diseased than the small, but even the arterioles can be involved. The arteries of the stomach, liver, and mesentery are rarely sclerotic. In arteriosclerosis connective tissue is substituted for the normal elements of the vascular wall, and this tissue undergoes hyperplasia and subsequent contraction and induration. If the mass of proliferating fibroblasts undergoes fatty degeneration, *atheroma* is said to exist, and an atheromatous vessel may be calcified by deposition of lime-salts. When fatty degeneration occurs the endothelium is destroyed, the vessel wall is damaged, and the blood may obtain access to the deeper coats. Atheroma is a frequent cause of thrombosis, aneurysm, senile gangrene, and apoplexy.

A sclerosed artery is rigid, non-contractile, and inelastic, and the parts it supplies are cold, congested and ill nourished, and often edematous. When the caliber of arteries remains narrowed because of persistent contraction or of arteriosclerosis there is marked accentuation of the second aortic sound. The valve or door which opened during systole is slammed shut during diastole by the peripheral resistance. The heart is obliged to overwork and in consequence undergoes hypertrophy. The hypertrophied heart finally dilates. If a hypertrophied heart exists with diseased arteries, apoplexy or aneurysm is apt to occur (Nammack, "Med. Record," Oct. 26, 1901). Syphilitic arteritis is characterized by an enormous growth of granulation tissue from the inner coats of arteries of small size (*obliterative endarteritis*). Calcification of an artery may be secondary to fatty change, or may occur primarily from deposit of lime-salts in the middle coat. *Periarteritis* is inflammation of the sheath and outer coat. An acute arteritis is always local, but a chronic arteritis may be general. If obliterative endarteritis exists in a limb, the veins are almost certain to be involved as well as the arteries. For this condition of veins and arteries, Buerger suggests the term "thrombo-angiitis obliterans" (see page 165).

*Treatment.*—In treating chronic arteritis, endeavor to antagonize the dangers to which the patient is obviously liable. Forbid alcohol as a beverage, though a little whisky may be taken at meals. Maintain the activity of the skin by daily baths, and of the kidneys by diuretic waters. A daily bowel movement should be secured. The diet is to be plain and is to contain a minimum of nitrogen. If syphilis has existed, occasional courses of iodid of potassium are to be given. If the arterial tension at any time becomes inordinately high, administer nitroglycerin. One danger to which the patient is liable is apoplexy, hence excitement and violent exercise are to be avoided. Another danger is senile gangrene; hence the patient should wear woolen stockings, put a bottle or bag of warm water to his feet at night, and be careful to avoid injuring his toes or feet, especially when cutting his corns. A bag of *very* warm water is dangerous and may actually excite gangrene. When a patient with atheroma has dyspnea and is of a livid color, or when the arterial tension is very high, a moderate blood-letting (16 to 18 oz.) does good, and may prevent or arrest edema of the lungs. Still another danger is aneurysm, which may appear suddenly from rupture or gradually from progressive distention.

It has been suggested that endarteritis threatening gangrene of the foot should be treated by an anastomosis between the common femoral artery and the femoral vein, in order that the blood may be directed from blocked

to open channels, and hence may still nourish the extremity. The operation is only to be advised when pulsation is absent in the tibials, when the femoral high up seems normal, and when the deep veins are patent (see page 183).

Buerger suggests the following test of the patency of the deep veins ("Jour. Am. Med. Assoc.," April 24, 1909): "I allow the limb to hang, watch for the advent of the erythema, and wait until a fair degree of cyanosis has become established. This may take considerable time—five to ten minutes. The veins are then obliterated above the knee by means of a Martin bandage properly applied. The limb is then raised high and the bandage loosened just enough so as to remove pressure from the deep, but not from the superficial, veins. If the cyanosis is slow in disappearing or fails to disappear, it may be concluded that the function of the deep veins is impaired."

An **aneurysm** is a pulsating sac containing blood and communicating with the cavity of an artery, and formed partly or entirely by the arterial walls or is a fusiform dilatation of an artery. Some restrict the term "true aneurysm" to a condition of dilatation involving *all* the coats of the vessel. We shall consider, with Heath, a *true* aneurysm to be one in which the blood is included in one or more of the arterial coats, and a *false* aneurysm to be a condition in which the vessel has been wounded, has ruptured or has atrophied and the aneurysmal wall is formed by a condensation of the perivascular tissues.

**Forms of Aneurysm.**—The following forms of aneurysm are recognized:

1. *True aneurysm*—one whose sac is formed of one or more arterial coats.

2. *False aneurysm*—one whose sac is formed of condensed perivascular tissues and contains no arterial coat.

3. *Traumatic diffused aneurysm*—a false aneurysm due to a wound or traumatic rupture of a blood-vessel. At first the blood is widely diffused and unlimited by any sac or capsule, later a limitation or encapsulation may occur by the condensation of tissue, any wound being healed. A traumatic diffuse aneurysm may follow a puncture or an incised wound of an artery, the injury causing the aneurysm directly. It may follow an effort or a strain, the injury indirectly causing the aneurysm by acting on a diseased vessel. As Barwell says, the term "traumatic diffused aneurysm" is an extremely bad one, as the term "aneurysm" conveys the idea of some sort of a sac. In this condition there is no true sac and blood is either unlimited or limited only by condensed tissue.

4. *Diffused aneurysm*—a term used to mean a ruptured aneurysm, the blood being diffused in the tissues and either unlimited or limited only by condensed tissues. The term should be limited to conditions in which the effusion of blood is slow and trivial. If the effusion is large and rapid, the term *ruptured aneurysm* is preferable.

5. *Consecutive aneurysm* results from the rapid growth of a sacculated aneurysm. At a certain portion of the sac of a true aneurysm the arterial coats give way completely, and at this point blood is limited only by clot and by condensed perivascular tissue. The blood is not diffused, but is encapsuled, partly by the old sac, partly by condensed tissues, aided it may be by bone and fascia.

6. *Fusiform* or *tubulated aneurysm*—a variety of true aneurysm, the sac being spindle-shaped and formed, as Matas states, "at the expense of the artery," the artery dilates, the continuity of the parent artery is interrupted for a variable length, and is lost in the sac, to be restored once more as a normal vessel at the outlet of the aneurysm ("Transactions of Am. Surg. Assoc.," 1905). Such an aneurysm has, of course, two openings. This form, according to Matas, comprises 66.6 per cent. of all aneurysms.

7. *Sacculated aneurysm*—a common form of aneurysm, in which the dilatation is like a pouch, arising from a part of the arterial circumference

and adjoining the lumen of the vessel by a single aperture. As Matas points out, the parent artery is involved in but a portion of its circumference, the continuity of the vessel is not lost, the arterial caliber is maintained at a nearly normal diameter, and "the sac is simply grafted or attached to the artery by a narrow neck, forming a sort of diverticulum of variable shape and dimensions" ("Proceedings of Am. Surg. Assoc.," 1905). Such a sac has but one orifice. The opening from the artery into the sac is called the *mouth;* around and just above the mouth is the *neck;* the balance of the sac is much larger than the neck and is called the *body*. A sacculated aneurysm may arise from an artery of normal size, from a dilated artery, or from a fusiform aneurysm. A sacculated aneurysm of unknown cause is called a *spontaneous aneurysm;* one which is due to injury is called a *traumatic aneurysm*. The first step in the formation of a sacculated aneurysm is stretching or giving way of an area of the middle coat (media), followed by a gradually advancing stretching and dilatation of corresponding areas of the outer coat (adventitia) and the inner coat (intima).

8. *Dissecting aneurysm* (*Shekelton's aneurysm*)—a pouch-like dilatation of an artery due to the blood-stream, which has gained access to the middle coat through an atheromatous ulcer or a minute rupture of the inner coat. It used to be taught that the blood flows between the media and adventitia; we now know that it flows between the layers of the middle coat. The outer wall of the aneurysm consists of adventitia and a portion of the middle coat. The unnatural channel may or may not join the lumen of the artery at another point by a fresh aperture in the intima. Dissecting aneurysm is practically only met with in the aorta. It is most common in the thoracic aorta. About 80 cases have been reported.[1]

9. *Arteriovenous aneurysm,* which is divided into aneurysmal varix, or Pott's aneurysm, when there is direct communication between a vein and an artery; and varicose aneurysm, when there is communication between an artery and a vein by means of an interposed sac.

10. *Acute aneurysm*—a cavity in the walls of the heart, which cavity communicates with the interior of this organ, and which is due to suppuration in the course of acute endocarditis or myocarditis.

11. *Aneurysm by Anastomosis.*—(See Angiomata.)

12. *Aneurysm of bone*—an inaccurate clinical term used to designate a pulsatile tumor of bone.

13. *Circumscribed aneurysm*—when the blood is circumscribed by distinct walls.

14. *Cirsoid aneurysm*—a mass of dilated and elongated arteries shaped like varicose veins and pulsating with each heart-beat.

15. *Cylindrical aneurysm*—a dilatation which maintains the same dimensions for a considerable space.

16. *Embolic* or *capillary aneurysm*—dilatation of terminal arteries due to emboli.

17. *Spontaneous aneurysm*—non-traumatic in origin.

18. *Miliary aneurysm*—a minute dilatation of an arteriole.

19. *Secondary aneurysm*—one which, after apparent cure, again pulsates, the blood entering by means of the anastomotic circulation.

20. *Verminous aneurysm*—one containing a parasite. This form of aneurysm is met with in the mesenteric artery of the horse.

The sac of a sacculated aneurysm is at first composed of at least two of the arterial coats, reinforced by the sheath and perivascular tissues. After a time the blood-pressure distends the sac, and the inner and middle coats either stretch with interstitial growth or—what is more common—are worn away

[1] Coleman, in "Dublin Jour. Med. Sciences," Aug., 1898.

and lost. When all the coats are lost and the blood is sustained only by the sheath and surrounding tissue, a true aneurysm becomes a false or consecutive aneurysm, the limiting tissues and sheath being condensed, thickened, and glued together. This limiting process is deficient in the brain, hence cerebral aneurysms break soon after their formation. When all the arterial coats are lost, the blood-pressure, acting on the tissues, finds some spots less resistant than others, the blood follows the lines of least resistance, the aneurysm grows with great rapidity, and soon ruptures externally or into a cavity.

An aneurysm may rupture into a cavity (pleural, pericardial, or peritoneal), into the perivascular tissues, or through the skin. Rupture into the tissues may produce pressure-gangrene. When rupture occurs through the skin the hemorrhage is not often instantly fatal, but during several days recurs again and again in larger and larger amounts. The pressure of an aneurysm causes atrophy of tissues, hard and soft, bones and cartilages being as easily destroyed as muscles and fat. Sometimes the perivascular tissues inflame and suppurate, and the sac is opened rapidly by sloughing. An aneurysm usually progresses toward rupture, the slowest in this progression being the fusiform dilatation, which may exist for many years, but which finally is converted into the sacculated variety.

In some rare instances there takes place spontaneous cure, which may result from laminated fibrin being deposited upon the walls of the sac as the blood circulates through it. The laminated fibrin is known as an "active clot," and eventually fills the sac. The weaker and slower the blood-stream, the greater is the tendency to the formation of an active clot, hence any agent impeding, but not abolishing, the circulation aids in the deposition. The weakening and slowing of circulation may be brought about by great activity of the collateral circulation diverting most of the blood from the area of disease. Sometimes a clot breaks off from the sac wall and plugs the artery beyond the aneurysm, and the anastomotic vessels, enlarging, divert the blood-stream. A large aneurysm, falling over by its own weight upon the vessel above the mouth of the sac, may, in very unusual cases, diminish the blood-stream. The development of another aneurysm upon the same vessel nearer to the heart weakens the circulation in and may cure the older one. Inflammation occasionally forms a clot. The tissues about an aneurysm tend to contract when arterial force is lessened, hence tissue-pressure may more than counteract blood-pressure when the circulation is feeble. Clotting of the blood contained within a sac, circulation through the aneurysm having ceased, causes a "passive clot." A passive clot, which occasionally induces cure, may arise from a twist of the neck of the sac preventing the passage of blood, from the lodgment of a clot in the mouth of the sac, and from inflammation. Spontaneous cure is, unfortunately, very rare.

**Causes of Aneurysm.**—Gradual distention of arterial coats which are in a condition of arterial sclerosis, or of coats whose resisting power is lowered because of atheroma, may cause aneurysm. Hence, the causes of sclerosis and atheroma are also causes of aneurysm. The principal cause of aneurysm is increased blood-pressure. This increase may be brought about by severe labor; by sudden strains, as in lifting; by violent efforts, as in rowing in a boat-race; by chronic interstitial nephritis; by hypertrophy of the heart; by alcoholic excess, and by syphilis. Arterial disease is commonest in the larger vessels, and in the aged, but it may occur in youth. When an aneurysm follows a strain, it may be due to laceration of the media and loss of resistance at a narrow point. The intima may lacerate, permitting the blood to come in contact with the media or causing blood to diffuse between the coats (dissecting aneurysm). When an embolus lodges in an artery the vessels may

become aneurysmal on the proximal side of the clot. The embolus, if infective, causes softening, and if calcareous causes laceration (Osler). Colonies of micrococci may cause aneurysm.[1] The parasite *Strongylus armatus* causes aneurysm of the mesenteric arteries in horses. Suppuration around a vessel weakens its coats and tends to aneurysm by inducing acute arteritis and softening. Sometimes an individual develops multiple aneurysms the origins of which are absolutely unknown. A bruise of a vessel may be followed by aneurysm. A cut or puncture of a healthy artery may lead, after the surface wound heals, to the development of an aneurysm. Such an aneurysm does not differ in symptoms or treatment from the other form.

*The constituent parts of an aneurysm* are: (1) the wall of the sac; (2) the cavity; (3) the mouth, and (4) the contents.

**Symptoms of Aneurysm.**—The formation of an aneurysm, when sudden, is occasionally, though rarely, appreciated by the patient, and is described by him as a feeling of something having given way. In most instances the feeling of beating and the discovery of the lump are the first intimations that anything is wrong. An oval or globular, soft, elastic, and pulsatile protrusion develops in the line of an artery. It is usually quite evident to the touch that the sac contains fluid, but sometimes in old aneurysms the sac feels firm or even hard, because of the deposit of fibrin upon its inner surface. In a partially consolidated aneurysm pulsation may be slight or even inappreciable. The protrusion instantly ceases to pulsate and almost disappears on making firm pressure on the artery above. On relaxing the pressure the pulsatile enlargement at once reappears. Direct pressure upon the tumor may cause it to almost disappear. Pressure upon the artery below causes the tumor to enlarge. The pulsation is expansile—that is, the sac expands in all directions during every cardiac contraction—and if an index-finger be laid on each side of the tumor so that the points nearly touch, each pulsation not only lifts the fingers, but it also separates them. It is important to remember that a *large* intrathoracic aneurysm which is in contact with the chest may not exhibit expansile pulsation, but simply transmit pulsation from the blood-stream (Sidney Lange, in "N. Y. Med. Jour.," Nov. 21, 1908). On placing a stethoscope over the aneurysm or over the vessel below the aneurysm there is imparted to the ear a distinct bruit, which travels in the direction of the blood-stream, is systolic in time, and is usually blowing in character. In some cases bruit is absent (when a sacculated aneurysm has a very small mouth, when the circulation is tranquil, or when the sac is full of blood and clot). When bruit is absent, it may sometimes be developed by muscular exercise or raising the affected limb (Halloway). In rare cases there may be a double bruit. Occasionally, in fusiform aortic aneurysm linked with aortic regurgitation, a diastolic bruit exists. A bruit is arrested by pressing upon the artery between the aneurysm and the heart. A patient who has an aneurysm of an extremity complains of a sensation of beating, of weakness or stiffness of the limb, frequently of pain in a nerve, a feeling of fatigue in the muscles, and edema and dilated veins are apt to develop because of pressure upon large veins and loss of *vis a tergo* in the circulation. The skin over an aneurysm may be normal, may be discolored, may ulcerate, or even slough. The pulse below an aneurysm is weaker than the pulse of a corresponding part of the opposite limb. This is well shown by sphygmographic tracings (Fig. 189). The tracings taken below an aneurysm are rounded without a sudden rise or an abrupt fall. In internal aneurysms pressure symptoms are marked. Thoracic aneurysm causes intercostal pain; iliac aneurysm causes pain in the thigh. Abdominal aneurysm is very rare. It is most common near the diaphragm. It is more apt to be sacculated than fusiform. As a rule, it distends forward; if it distends back-

[1] See Osler on "Malignant Endocarditis."

ward it may destroy the vertebræ and press upon the cord. Pain practically always occurs, usually in the back, sometimes in the abdomen. Expansile pulsation and bruit make the diagnosis clear. The *x*-rays may be valuable in establishing the diagnosis. Aneurysm of the thoracic aorta pressing upon the pneumogastric nerve causes spasmodic dyspnea, and upon the recurrent

Fig. 189.—Radial pulse-tracings in aneurysm of right brachial artery: 1, Left radial pulse; 2, right radial pulse (after Mahomed).

laryngeal causes hoarseness, which may be associated with loss of voice, cough, and laryngeal spasm, and is due to unilateral abductor paralysis. Pressure upon a bronchus or the trachea causes dyspnea from obstruction, dysphagia, and cough from laryngeal spasm. Pressure upon the cervical sympathetic

Fig. 190.—*X*-ray of aneurysm of thoracic aorta.

first causes dilatation and later contraction of the pupil of the same side. A thoracic aneurysm may erode the ribs, sternum, or vertebræ. The *x*-rays are of great value in diagnosticating thoracic aneurysm (Fig. 190). An aneurysm in the neck may interfere with the cerebral circulation and produce vertigo and even attacks of unconsciousness. The evidences of rupture of an aneurysm of

an extremity into the tissues are loss of distinctness of outline and increase in area of the swelling, weakening or disappearance of both bruit and pulsation, absence of pulse below the aneurysm, severe pain, edema and coldness of the surface, shock, and possibly syncope. External hemorrhage may arise; the tissues may become extensively infiltrated with blood; sloughing or gangrene may ensue. Death is frequent, and only in very rare cases does spontaneous cure take place. Rupture of a large aneurysm into a cavity causes intense pallor, advancing weakness, syncope, and death.

**Diagnosis.**—A cyst or abscess over a vessel may show transmitted pulsation which is not expansile, and the swelling does not disappear when pressure is made upon the vessel above it. The pulsation ceases when the growth is lifted off the vessel, or when the position is changed so as to permit it to fall away from the vessel. There is no true bruit, and the history is widely different. A growth under a vessel may lift the vessel and simulate an aneurysm, but the pulsation is not noted in the entire growth, the growth does not disappear on proximal pressure, and there is only a false, and never a true, bruit. The larger the growth under a vessel the less is the pulsation, because of pressure narrowing the caliber of the vessel. A sarcoma, especially a soft sarcoma attached to the bone, and also a nevoid mass, pulsate and often have a bruit; the tumor never disappears from proximal pressure, though it may slowly diminish in size, to gradually enlarge again when pressure is withdrawn. These growths do not feel fluid, and are rarely circumscribed. An aneurysm may cease to pulsate from consolidation leading to cure or from rupture. Rupture of a large aneurysm into a cavity induces deadly pallor, syncope, and rapid death. Rupture of an aneurysm of an extremity into the tissues is made manifest by a sensation of something breaking, by pain, by sudden increase in size, by diminution or absence of bruit and pulsation, by absence of pulse below the aneurysm, by swelling and coldness of the limb, and by shock. The *x*-rays are valuable in diagnosticating thoracic aneurysm and abdominal aneurysm.

**Treatment.**—(For the history of the evolution of the treatment of aneurysm, see "Studies in Aneurysm," by James G. Mumford, "Cleveland Med. Jour.," Feb., 1908.) In inoperable aneurysms, *general*, *medical*, and *dietetic* treatment must be tried. A chief element in treatment is rest in bed to diminish the rapidity and force of the circulation and favor fibrinous deposit. Valsalva long ago suggested rest, occasional bleeding, and a diet just above the point of starvation. Tuffnell's plan is to reduce the heart-beats by rest and mental quiet, and to rigidly restrict the diet so as to diminish the total amount of blood and render it more fibrinous. Liquids are restricted in amount, and the patient lives through each twenty-four hours upon 4 oz. of bread, a very little butter, 8 oz. of milk, and 3 oz. of meat. This plan is pursued for several months if possible, or it is employed for several weeks, intermitted for a short period, the rigid diet again returned to, and so on, over and over again. There can be no doubt that Tuffnell's treatment sometimes cures aneurysm by decidedly lowering the blood-pressure. Many who suffer from aneurysm may be permitted to go about, taking their time about everything and avoiding work, worry, and excitement. The diet should be low and non-stimulating, and the bowels must be maintained in a loose condition.

Even in an operable case diet and rest are of importance. Often a patient is kept in bed for a number of days before operation, the daily diet consisting of 10 or 12 oz. of solid food with a pint of milk. If the circulation is very active, use aconite and allay pain by morphin.

Iodid of potassium in doses of 20 gr. undoubtedly does good in aneurysm and not only in syphilitic cases. It seems to lower the blood-pressure. Balfour taught that it thickened the walls of the sac. Osler says it relieves the

pain. Iron, acetate of lead, and ergotin are prescribed by some. Digitalis is contra-indicated, as it raises the blood-pressure. S. Solis-Cohen has used with some success the hydrated chlorid of calcium. Morphin and bromid of potassium are occasionally useful to tranquilize the circulation, allay pain, or secure sleep. Aconite and veratrum viride have long been employed.

Lancereaux and others claim that hypodermatic injections of gelatin at some indifferent point may cure aortic and subclavian aneurysm. In 1896 Dastres and Floresco proved that gelatin injected into the blood increases coagulability. Later, Lancereaux and Paulesco showed that injections into the subcutaneous tissue act similarly. Carnot pointed out that gelatin applied to a wound may arrest bleeding. How gelatin acts is uncertain, but that it does increase blood coagulability seems proved. The value of injections of gelatin for aneurysm is in dispute. Lancereaux warmly advocates its use for sacculated aneurysm, and says that after the first dose the aneurysm is seen to shrink and the pulsation is observed to lessen. He injects it slowly and with aseptic care into the subcutaneous tissue of the thigh, using normal salt solution containing from 5 to 10 per cent. gelatin. He never injects less than 5 gm. He gives an injection every tenth to fifteenth day and administers from ten to twenty injections. But the treatment is not free from danger; several deaths have taken place, and several persons have died from tetanus. Care must be taken not to inject gelatin into a vessel, and it must never be thrown about the aneurysmal sac. It irritates the kidneys and its use is contra-indicated in renal disease. The injections cause much pain, and it is very doubtful if they do any real good in aneurysm. If used it should be given at the temperature of the body, and not over 3 gm. should be administered at one dose. A 10 per cent. solution is the proper strength and from 10 to 20 c.c. the correct dose. Gelatin can be given by the mouth. When thus given it is not so powerful, but its coagulating property is not destroyed by digestion. Gelatin in normal salt solution is known as Carnot's solution. *Carnot's solution* is best prepared by Sailer's formula, as follows (Joseph Sailer, in "Therapeutic Gazette," August, 1901): Take 5 gm. of common salt, 1 liter of distilled water, and 100 gm. of gelatin. Bring the water to a temperature of 80° C. and slowly stir in the gelatin until it is all in solution. Remove the solution from the stove, cool it to 40° C., add to it the white of one egg, and stir for several minutes, and then put the flask on the stove and boil the fluid. The white of egg coagulates and clears the solution. Filter through gauze and then through paper. Place the fluid in test-tubes, each of which will contain 10 c.c., and insert a cotton plug in the mouth of each tube. Sterilize by putting the tubes in a steam sterilizer for fifteen minutes on three successive days. When wishing to use a tube, place it in a cup of hot water until the gelatin liquefies, pour the gelatin into a sterile glass, and draw it up into a sterile syringe. When kept several weeks the gelatin dries out.

Other expedients sometimes used in the treatment of aneurysm are: the kneading of the sac to release a clot, in the hope that it will plug the mouth of the sac or the artery beyond it—this is dangerous; electricity; electrolysis; the injection of an astringent liquid; the insertion of a fine aspirating needle and the pushing through it into the sac of a large quantity of silver wire, in the hope that it will aid in whipping out fibrin. Some physicians have inserted needles and others horsehair.

*Treatment by Pressure.—Instrumental pressure* is made by applying two Signorini tourniquets or some specially devised apparatus to limit the flow of blood through an aneurysm without entirely stopping it, the aneurysmal sac being felt to still slightly pulsate. In some situations Lister's abdominal tourniquet is applied; in other regions we may use Tuffnell's compress, which

is like a spring truss and is strapped in place. A heavy body suspended over the artery and resting part of its weight upon the vessel has occasionally brought about cure. Compressing instruments can be worn for from twelve to sixteen hours at a time; usually they are removed to permit sleep and are reapplied the next day, and so on for several days. Before applying the compress be sure the sac is full of blood, and render this certain by applying for a few minutes distal compression. This method may cure, but it is very painful. It cannot be used successfully in treating aneurysm of the axillary, subclavian, or carotid artery. It aids in the formation of an active clot.

*Digital pressure*, made with the thumb aided by a weight, and maintained for many hours by a relay of assistants, has cured many cases. This method may be used alone or may be used as an accessory to instrumental pressure. Its chief field is in the treatment of aneurysm for which other methods are inapplicable (orbit and some aneurysms at the root of neck). It entirely cuts off the blood and promotes the formation of a passive clot. If cure does not take place in three days, abandon pressure. It must often be abandoned far earlier because of pain.

*Direct pressure* upon the sac has been used in aneurysm of the popliteal artery, the pressure being obtained by flexing the leg; and in aneurysm of the brachial artery pressure has been applied at the bend of the elbow by flexing the elbow. The pressure of a hollow rubber ball has been used in aneurysm of the subclavian.

*Rapid pressure* completely arrests the passage of blood through the sac for a limited time, and is applied while the patient is under the influence of an anesthetic. Take, for example, a case of popliteal aneurysm: the patient is placed under the influence of ether; two Esmarch bandages are used, one being applied to the limb from the toes up to the lower limit of the aneurysm, and the other from the groin down to the upper limit of the sac, and the Esmarch band is fastened above the upper bandage. This prodecure stagnates the blood both in the veins and in the arteries, and the sac remains full of blood. Pressure is thus maintained for three or four hours, and on removing the Esmarch apparatus a tourniquet is put on the artery above the aneurysm and partly tightened in order to limit the amount of blood passing through and thus prevent the washing away of clot. This method of rapid pressure sometimes cures by forming a passive clot, but it sometimes results in gangrene. It was devised by John Reid.

*Operative Treatment: By the Ligature and by Sutures.*—Ligation of the main artery was the operation employed by most surgeons until the Matas operation was introduced. The methods of ligation are: (1) the method of Antyllus; (2) extirpation of the sac; (3) the method of Anel; (4) the method of Hunter; (5) the method of Wardrop, and (6) the method of Brasdor.

*Aneurysmotomy*, the *method of Antyllus* (Fig. 191), a Roman successor to Galen, who lived in the third century, A. D., is usually described as a method involving a direct attack upon the sac itself. The artery is ligated immediately above and below the sac, the sac is opened and its contents turned out, or the sac is extirpated. As a matter of fact, Antyllus advocated applying a ligature on each side of the sac and opening the sac in order to evacuate its contents, but he distinctly opposed extirpation because of its danger. All we know of Antyllus is found in the writings of Oribasius, who lived in the fourth century. Syme maintained many years ago that incision of the sac is the proper operation for aneurysm of the gluteal, iliac, carotid, and axillary arteries, but Syme's method is productive of fearful hemorrhage, and the plan of Antyllus is vastly better. Syme opened the sac, inserted his finger, and plugged the artery toward the heart until a ligature was applied and tied. He then packed the sac with lint.

*Aneurysmectomy* (*extirpation of the sac*), if practised, should be carried out after applying a ligature on each side after the method of Antyllus. It was originally practised by Philagrius and was reintroduced by Purmann in 1699 (Moynihan, in "Annals of Surgery," July, 1898).

Extirpation finds warm advocates in Delbet, Littlewood, and Moynihan. Moynihan claims that, as compared with distal ligature, there is a greater chance of recovery, no chance of recurrence, less risk of gangrene, and complete recovery from troubles due to nerve interference (Ibid.). Extirpation is regarded by some surgeons as the best operation for traumatic aneurysm, but if the vessel is seriously diseased near the sac some other method should certainly be employed. In aneurysm of the common carotid after extirpation (as after ligation) there is grave risk of cerebral embolism, and it might be wise to attempt a re-establishment of the circulation by circular suture of the two ends, or, as Lexer did in the axillary artery, autoplasty with a piece of the internal saphenous vein. In the extremities there is less danger of gangrene after Matas's operation than after extirpation. Monod and Vanverts ("Rev de. Chir.," 1910, xli and xlii) collected 205 cases of excision. Of these, 90 per cent. were cured. Relapse occurred in 1½ per cent. Direct operative mortality was 3 per cent. Gangrene occurred in 4 per cent. Extirpation shows a higher percentage of cures, a lower rate of mortality, fewer cases of gangrene, and fewer relapses than any operation except that of Matas. Monod and Vanverts collected 138 cases of ligation by different methods. The mortality was 7 per cent. There were 12 per cent. of relapses and 6½ per cent. of gangrene. The cures numbered 74 per cent.

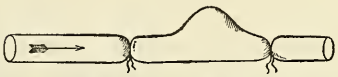


Fig. 191.—Old operation of Antyllus for aneurysm ("Am. Text-Book of Surgery").

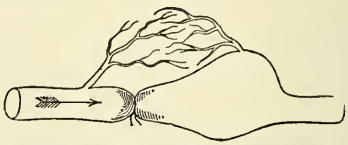


Fig. 192.—Anel's operation for aneurysm ("Am. Text-Book of Surgery").

*The Method of Anel.*—Anel, of Turin, devised and performed this operation in 1710. In Anel's method the artery is ligated above the sac, and so close to it that there are no anastomotic branches between the sac and the ligature (Fig. 192). It is used only for traumatic aneurysms, and is never employed when the vessel is diseased beyond the aneurysm. Either extirpation or Matas's operation is preferable to Anel's operation.

*The Method of Hunter.*—This operation, which is the modern method of ligation, was devised by the illustrious John Hunter, and was first employed by him in January, 1786. He is said by Sir Everard Home to have recognized the fact that the vessel adjacent to an aneurysm was apt to be diseased, and he discovered the anastomotic circulation. Putting together these two facts, he devised the operation which goes by his name. It consists in applying a ligature between the heart and the aneurysm, but so far above the sac that collateral branches are given off between it and the point of ligation (Fig. 193). This operation, which is done upon a healthy portion of the artery, does not permanently cut off all blood, but so diminishes the force and frequency of the circulation that an active clot forms within the sac. Thus are lessened the dangers of secondary hemorrhage and gangrene. According to Stimson ("New York Med. Jour.," July, 1884), Hunter really builded better than he knew, for he sought only to tie the artery without opening the sac and at a healthy point, but said not a word about the necessity of having branches between the sac and the ligature or about the desirability of diminishing the flow of blood instead of cutting it off completely (Moynihan, in "Annals

of Surgery," July, 1898). Hunter tied the artery in the region now known as Hunter's canal. Scarpa introduced the custom, which we still follow, of tying it in Scarpa's triangle. The Hunterian method was for many years regarded by most surgeons as the proper operation for aneurysm in the majority of cases. In some cases pulsation does not return in the aneurysm after tightening the ligature; in most cases, however, it reappears for a time after about thirty-six hours, but is weak from the start, constantly diminishes, and finally disappears permanently. Previous prolonged compression by enlarging the collateral branches permits strong pulsation to recur soon after ligation, and thus militates against cure; hence, it is a bad plan to use pressure in cases admitting of ligation and in which the success of pressure is very doubtful. Occasionally after Hunter's operation the sac suppurates, producing symptoms like those of abscess. Suppuration may occur between the first and the thirty-second week after ligation.[1] When pus forms, open freely, as we would open an abscess, and if no blood flows, treat as an abscess, but have a tourniquet loosely applied for several days ready to screw up at the first sign of danger. If hemorrhage occurs, tie the vessel above and below the aneurysm, open the sac, and pack with iodoform gauze. If bleeding recurs, there is no use reapplying the ligature and there is little use tying higher up.

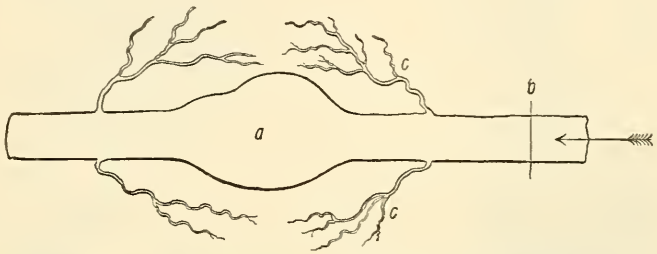



Fig. 193.—Hunter's method of ligating for aneurysm: *a*, The aneurysm; *b*, point of ligation; *c*, the branches between the aneurysm and the ligature. The arrow shows the direction of the blood-current.

If dealing with the upper extremity, try the application of a ligature higher up; if dealing with the lower extremity, amputate at once.

*Distal Ligation.*—When an aneurysm is so near the trunk that Hunter's operation is impracticable, or when the artery on the cardiac side of the tumor is greatly diseased, distal ligation may be employed. Distal ligation forms a barrier to the onflow of blood, collateral branches above the aneurysm enlarge, the blood-current is gradually diverted, and a clot may form within the aneurysm. Distal ligation is used in some aneurysms of the aorta, iliac, innominate, carotid, and subclavian. It occasionally causes rupture of the sac of the aneurysm. I have obtained two notably successful results in aneurysms of the innominate artery by ligation of the common carotid and subclavian of the right side. In each of these cases I tied both vessels at one séance, tying the carotid first. In 1 case I tied the third part of the subclavian and in the other the first part. The first patient returned to his work as a blacksmith and died over a year and a half later from rupture of a secondary aneurysm of the carotid at the point where the ligature had been applied. The second case is living and apparently well over five years after the operation (the author, in "Surg., Gyn., and Obst.," June, 1910).

The *operation of Brasdor* consists in tying the main trunk some little distance below the aneurysm (Fig. 194). It completely arrests circulation in the sac. The operation was introduced in 1760 by the French surgeon Brasdor.

[1] See the case described by Sir Astley Cooper.

The *operation of Wardrop* consists in tying one of the branches of the artery beyond the aneurysm. Wardrop originally advocated ligation at a point where there is no intervening branch between the sac and the ligature. Later, he advocated ligation at a point where there is an intervening branch. Since then it is the custom to consider Wardrop's operation to be the ligation of one branch beyond the aneurysm, as shown in Fig. 195. The circulation is but partially arrested by Wardrop's operation. The operation was introduced in 1825. An *x*-ray picture should be taken in every case of aortic aneurysm. Such a picture may aid us in coming to a conclusion as to which vessel or vessels to tie.

*Matas's Operation* (*Aneurysmorrhaphy*).—This procedure is the greatest advance in the surgery of the arterial system since the observations of John Hunter. It was first practised by Matas in 1889 on a negro suffering from traumatic aneurysm of the brachial artery in the middle of the arm. The operation was a complete success. In 1900 he began again to use this method, and in 1902 described it to the profession ("Trans. of Am. Surg. Assoc.," 1902; "Annals of Surg.," Feb., 1903; "Trans. of Am. Surg. Assoc.," 1905).

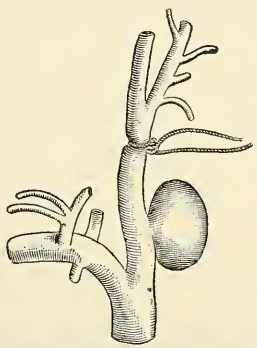


Fig. 194.—Brasdor's operation (Holmes).

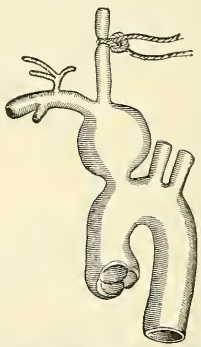


Fig. 195.—Wardrop's operation (Holmes).

One procedure, applicable to ordinary fusiform aneurysms, is called *obliterative endo-aneurysmorrhaphy without arterioplasty* (Fig. 196). "No attempt is made to reconstruct the parent artery (arterioplasty), and the arterial orifices are simply obliterated by suture." By sutures applied within the incised sac the sac is cut off from the circulation without disturbing adjacent collaterals and without interfering with the nutrition of the sac walls. After this operation there is very seldom secondary hemorrhage, gangrene, or relapse.

A modification of the above operation applied to sacculated aneurysms in which there is one orifice of communication with the artery is called *endo-aneurysmorrhaphy with partial arterioplasty* (Fig. 196). The sac is opened, clots are washed away, the opening of the aneurysm into the artery is closed by a continuous suture passing through all the coats of the sac at the edge of the opening into the artery. Blood is thus excluded from the sac, the lumen of the artery is not, however, obliterated, and the blood-supply of parts beyond is not interfered with. After closing the cut in the arterial wall the sac is obliterated by rows of sutures inserted in its walls. Matas reports 4 cases operated upon successfully by this plan. In a fusiform aneurysm with a firm and resisting sac wall, and in which there are two openings near together on the floor of the sac, *endo-aneurysmorrhaphy* with complete *arterioplasty* may be performed (Fig. 196). This operation restores arterial continuity, a new channel being made out of the sac walls "by simply holding these over a rubber guide (tube or catheter) and suturing them firmly together so as to restore

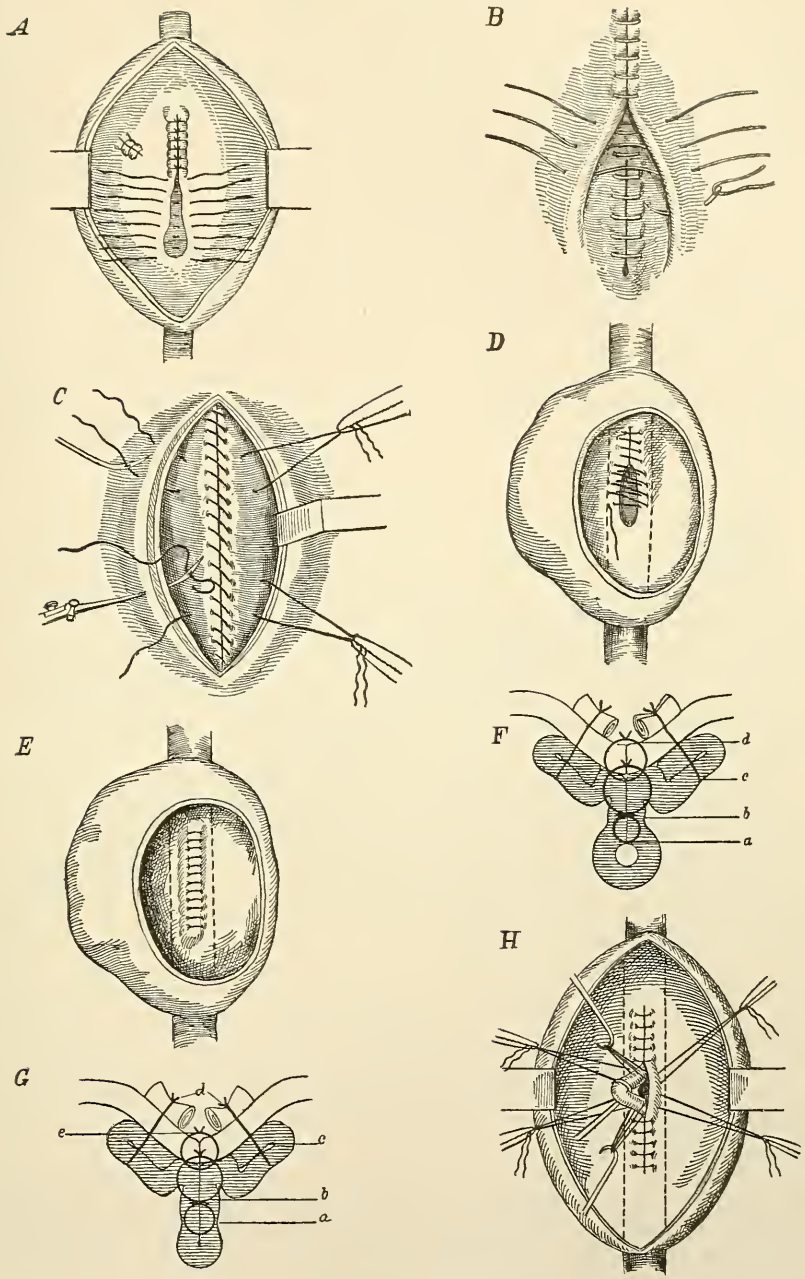



Fig. 196.—The radical cure of aneurysm based upon arteriorrhaphy (Matas): *A*, First tier of sutures in a fusiform aneurysm; *B*, second tier of sutures, some of which are tied; *C*, sutures to approximate the walls of the aneurysm; *D*, suturing the opening in a sacculated aneurysm—the main artery is not obliterated; *E*, opening completely closed: *F*, diagram of cross-section of parts after complete obliteration of sac, but with restoration of blood-channel; *G*, diagram of cross-section of parts after complete obliteration of sac and blood-vessel; *H*, operation for fusiform aneurysm when we wish to restore the blood-channel—sutures applied over a rubber tube, most of the sutures tied, tube withdrawn, and remaining sutures tied.

the continuity of the artery lost in the sac." The catheter is withdrawn before the final sutures are tied. This operation has been performed successfully by Morris and also by Craig. Some surgeons are fearful that such an operation will be followed by relapse, and one of the reported cases did relapse. Matas says that preservation of the arterial lumen is "only indicated positively in the sacciform aneurysms with a single opening where the parent artery already exists as a formed vessel, and in which the closure of the fistulous opening can be accomplished with the greatest facility and simplicity" (address delivered at the Medical Assoc. of Alabama, April 22, 1906). It is not probable that the artery remains patent long, because the seat of aneurysm is a diseased vessel and vascular disease will probably cause clotting, but even a temporary restoration of circulation if followed by *gradual* abolition prevents gangrene. The Matas operation differs notably from the Antyllus operation in the fact that it saves certain collaterals which the Antyllus method destroys, and the retention of these collaterals may prevent gangrene in the limb. It differs from it further in the fact that it occludes certain small vessels which after the Antyllus method continue to convey blood into the sac. It is superior to extirpation because it does not destroy the vascular walls of the sac, the blood-vessels of which, if unblocked, aid in preventing gangrene of the limb.

Matas points out that suture of an aneurysm is indicated only when certain essentials exist.

1. The situation of the aneurysm must admit of the control of the circulation temporarily on the proximal side of the sac. In most aneurysms of the extremities this is done by the elastic band of Esmarch. In the neck and abdomen both the cardiac and peripheral sides of the main vessels must be secured by traction loops and compression.

2. The sac must be freely opened in a longitudinal direction. Its wall must not be dissected and must be separated as little as possible from surrounding tissue.

3. Every orifice opening into the sac must be thoroughly exposed so that they can be closed by sutures. The suture material is chromic gut, the number being 1, 2, or 3, according to the size of the aneurysm.

Fig. 196, *A* to *H*, shows Matas's various operations. For a full description of them see the previously quoted articles of the author. I believe that the Matas operation is a very notable advance in surgery, that it is safer than older methods, and much less apt to be followed by gangrene. The idea seems to be general that Matas always seeks to restore arterial lumen. This is not the case. He only seeks to do this in exceptional cases. The essence of his method is to cure the aneurysm by sutures within the sac and by obliteration of the sac. I have performed the Matas operation (obliterative endo-aneurysmorrhaphy) successfully on a case of ruptured fusiform popliteal aneurysm and on a case of ruptured sacculated popliteal aneurysm. In the latter case there was profuse hemorrhage during the operation from vessels opening into the sac.

Matas, in "Annals of Surgery," July, 1910, collected 149 cases with 133 cures. In Monod and Vanvert's 103 cases there were 89 cures. Relapse occurred in 1.5 per cent. Gangrene occurred in from 3 to 5 per cent. of the cases. Direct operative mortality was 3 per cent. ("Rev. de Chir.," 1910, xli and xlii).

Less than 2 per cent. of cases relapse. The direct mortality is 3 per cent. Gangrene occurs in 3 per cent. of cases. Secondary hemorrhage is very rare.

The operation has been performed upon the abdominal aorta, the external iliac, and the subclavian, as well as upon smaller vessels. Munro, Martin and Parham, Lilienthal, Pringle, and Lozano performed endo-aneurysmorrhaphy for subclavian aneurysm (Eliot, in "Annals of Surgery," July, 1912).

*Halsted's Method by Partial, Progressive, and, Finally, Complete Occlusion by Metal Band.*—This method is applied only to the aorta and other very large arteries. A number of surgeons have sought a method to gradually and safely occlude the abdominal aorta in order that they may attempt to cure aneurysm of the aorta or of the iliac arteries. The usual thought was to leave a metal instrument fixed to the aorta, the handle projecting from the abdominal wound, and a metal clamp or a snare of silk or catgut being around the vessel, so that by means of a screw arrangement pressure could be gradually increased.

Halsted showed that by this plan sepsis almost certainly occurs along the track of the instrument. (See W. S. Halsted, in "Johns Hopkins Bulletin," 1905, xvi, 346; "Jour. Am. Med. Assoc.," 1906, xlvii; "Jour. Experimental Medicine," 1909, vol. xi, No. 2.) Halsted sought for a method "permitting, in each entre-act, complete closure of the wound." He uses a band of aluminum curled in cylinder form about the vessel. This material admits of easy readjustment at a future operation, and it is tightened by the fingers.

Halsted's band, when used to partially occlude, seldom causes macroscopic alteration in the wall of the vessel; when used to completely occlude, the vessel may undergo atrophy. Ideal closure is when the lumen was nearly but not

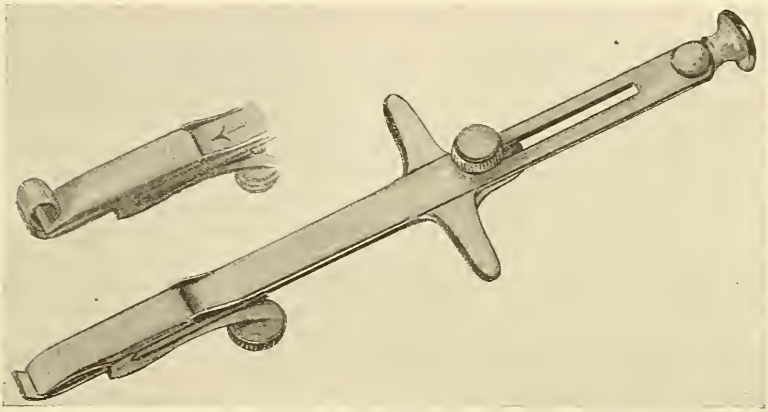


Fig. 197.—Halsted's improved band roller: The instrument shown in full length is unloaded; in the abbreviated cut the band is about to be expelled from the roller (Halsted).

quite occluded, spontaneous obliteration having arisen, the arterial wall embraced by the band having undergone conversion into "a solid cylinder of living tissue." Animals tolerate gradual occlusion very well indeed.

Halsted has made numerous experiments on dogs and has used the band with encouraging results on the human being (partial occlusion of the innominate, twice; common carotid, four times). In a case of aneurysm of the abdominal aorta Halsted partially occluded the aorta near the diaphragm. Seventeen days later he placed another band on the aorta below the aneurysm. The patient lived twenty-four days after the second operation. The operation checked growth and arrested pain ("Trans. Am. Surg. Assoc.," 1909–10). In another case of abdominal aneurysm Halsted put a band above the renals. Seventeen days later Finney inserted wire into the aneurysm. The patient lived forty-five days after the first operation (Ibid.).

In preparing for application it is rolled by means of the instrument shown in Fig. 197. It is well to fasten the band with a silver ligature after application. The ligature prevents unrolling (Ibid.).

**Treatment After Operation for Aneurysm.**—After operating for aneurysm of an extremity by the ligature or by sutures, elevate the limb slightly, keep

it warm by wrapping in cotton and surrounding with bags of warm water, and subdue arterial excitement. When gangrene of a limb follows ligation, await a line of demarcation, and when it forms, amputate. Rupture of the sac after ligation may produce gangrene or be associated with suppuration, the first condition demanding amputation; the second, incision for drainage.

*Injection of coagulating agents* into the sac (ergot, perchlorid of iron, etc.) is very dangerous and is to be utterly condemned. It may lead to suppuration, gangrene, rupture, or embolism.

*Manipulation* to break up the clot was suggested by Sir Wm. Fergusson and has been practised by some. The object aimed at is to have a fragment of clot block up the vessel upon the peripheral side of the artery and act like a distal ligature. The method is dangerous, especially in carotid aneurysm, and should never be employed.

*Amputation*, instead of distal ligation, is performed in some perilous cases of subclavian aneurysm.

*Electrolysis.*—An attempt may be made to at once coagulate the blood in the sac, or from time to time an endeavor may be made to produce fibrinous deposits, but the first method is the better. It is, however, seldom possible to at once occlude a sac, and pulsation, which is for a time abolished, usually recurs as the gas present is absorbed. Use the constant current. Take from three to six cells which stand in point of size between those used for the cautery and those used for ordinary medical purposes. A platinum needle is attached to the positive pole and a steel needle to the negative pole, each needle being insulated by vulcanite at the spot where the tissues would touch it. The asepticized needles are plunged into the sac where it is thick, and they are kept near together. The current is passed for a variable period (from half an hour to an hour and a half). This operation is not dangerous. Pressure stops the bleeding. Electrolysis often ameliorates and sometimes greatly improves aortic aneurysms.

*Acupuncture* consists of the partial introduction of a number of ordinary sewing needles into an aneurysmal sac and leaving them in it for five or six days or more. Professor Macewen introduces a needle, and with it irritates the interior of the sac of an aneurysm, hoping thus to cause deposition of leukocytes, thickening of the sac, and clot formation.

*Introduction of Wire.*—This operation is performed by inserting into the sac a hypodermatic or small aspirating needle, and pushing in through the needle or cannula a considerable quantity of aseptic gold or silver wire, which is allowed to remain permanently. Wiring is used for aneurysms otherwise inoperable. Electrolysis should be combined with the introduction of wire. Wiring was first proposed by Moore, of London, in 1864. The details were improved by Corradi in 1879. Loreta and Barwell both inserted wire into an aneurysm before Corradi, but Corradi inserted wire and also used electricity. The first American wiring operation was performed by Ransohoff in 1886. Corradi's operation can be used when distal ligation cannot be carried out, and can be used even when the vessel is extremely atheromatous. It finds its chief use in aneurysms of the thoracic aorta and innominate. In some cases of abdominal aneurysm the belly has been opened and the operation carried out. It is used for sacculated, never for fusiform, aneurysms. The operation has not many elements of danger. Sepsis would inevitably cause death. If the wire is carried on into the aorta and heart death will follow. A cause of death is embolism. In one of Finney's cases gangrene of the arm resulted from embolism. Too strong a current may cause sloughing of the aneurysmal wall. The wall of the aneurysm may rupture because of deviation of the strong blood-current in another direction. Some cases have been notably improved. The operation is performed with aseptic care. If the thoracic aorta is to be oper-

ated upon, an anesthetic is not required. If the abdominal aorta is to be wired, the patient must be anesthetized, because the abdomen needs to be opened. The wire used must have been previously drawn, so that it will easily pass through a hypodermatic needle and will coil up spirally within the sac. The best wire is of silver or gold. A special reel is used to keep the wire from getting kinked. It is a great mistake to introduce a large quantity. Stewart decided that a globular sac 3 inches in diameter requires from 3 to 5 feet, and a sac 5 inches in diameter requires from 8 to 10 feet. A hypodermatic needle, insulated up to ¼ inch of the point, is carried into the interior of the aneurysm through a fairly thick portion of the sac. The shoulder of the needle is not insulated and must not be permitted to touch the skin, because if it did so it would cause a burn by electrolysis. The required amount of wire is introduced. The wire is attached to the positive pole of the battery. The negative pole is fastened to a large flat piece of clay or a pad of moistened absorbent cotton, and the negative electrode is placed upon the back or abdomen. The current is turned on gradually until the necessary strength is obtained (40 to 80 ma.). When ready to terminate the operation the current is lowered gradually to zero, the needle is withdrawn, the wire is cut off close to the skin, the end is pushed under the skin, the puncture is covered with iodoform collodion, and pressure is applied to keep blood from gathering in the tissue. Such a hematoma might cause the formation of a slough. The entire operation requires from three-quarters of an hour to one and a half hours. A clot forms with considerable rapidity and expansile pulsation may lessen or cease. It requires from a number of days to several weeks for the clot to become hard. The operation can be repeated if necessary. Injections of gelatin after wiring may be beneficial. Rest is imperative for months after wiring.

Notable improvement is common, but genuine cure is not obtained. As Hare says, "adjacent tissues of the vessel sooner or later give way, because the effort is like an effort to mend a rotten hose; though mended at one spot it breaks at another." One of Jones's cases remained well for six years and then pain recurred. The operation causes prompt and marked diminution of pain, an amelioration usually spoken of by the patient before he leaves the operating table. In 2 of my cases pain, which had been severe, disappeared completely during the operation. "Dyspnea is also benefited and just as rapidly" (H. A. Hare, in "Therapeutic Gazette," April, 1908). Finney ("Annals of Surgery," May, 1912) reported 23 cases (mostly thoracic, some abdominal). Of these cases only 2 were known to be living at the time of the report.

**Treatment of Aneurysm Following Wound of a Healthy Artery.**—The prognosis in such a case is usually extremely good. The treatment is as for the other forms. Extirpation is particularly adapted to such direct traumatic aneurysms in the neck and Matas's operation to those in the extremities.

**Diffuse Traumatic Aneurysm.**—When an artery ruptures or an aneurysm ruptures and a large mass of blood is extravasated into the tissues, no complete sac exists, and the condition is usually called diffuse traumatic aneurysm. In diffuse traumatic aneurysm a large oblong, fluctuating swelling is found. If the rent is large there may be bruit and pulsation. There is no pulsation in the artery below the aneurysm, and the limb is cold and swollen. The skin is at first of a natural color, but later becomes thin and purple.

**Treatment.**—If an aneurysm ruptures, cut down upon the aneurysm, incise the sac longitudinally, and perform Matas's operation. Some surgeons cut down to the aneurysm, tie on each side of the tear, open the sac, and pack it (the operation of Antyllus), but Matas's operation is the preferable procedure. If an artery is ruptured, empty the limb of blood, apply an Esmarch band above, and expose the seat of rupture by incision. If possible, suture the opening; if this is not possible, tie the vessel on each side of the rupture and

excise the intervening portion. If the main vein is also ruptured, suture the vessels if possible. If suture is impossible, apply ligatures. The attempt will probably not succeed in saving the extremity, and in most cases amputation will be required.

**Arteriovenous aneurysm** was first described by Wm. Hunter in 1757. By this term we mean an unnatural passageway between a vein and an artery, through which passage blood circulates. There are two forms: (*a*) *aneurysmal varix*, or *Pott's aneurysm*, a vein and an artery directly communicating (Fig.

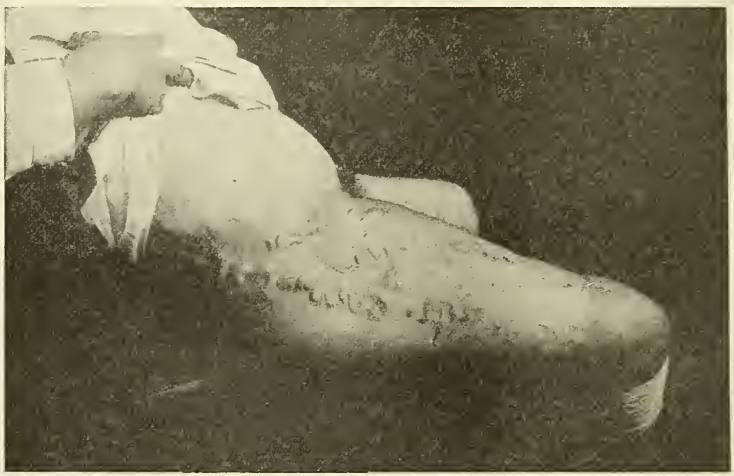


Fig. 198.—Dilatation of veins in arteriovenous aneurysm of the femoral vessels.

199); and (*b*) *varicose aneurysm*, a vein and an artery communicating through an intervening sac. These conditions arise usually from punctured wounds, the instrument passing through one vessel and into the other, blood flowing into the vein, the subsequent inflammation gluing the two vessels together, and the aperture failing to close (aneurysmal varix, Fig. 199). After the infliction of the wound the two vessels may separate; the blood continuing to flow from artery into vein, and the blood-pressure, by consolidating tissue, forming

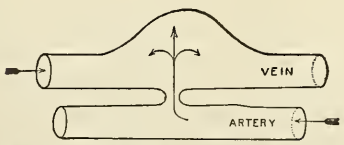



Fig. 199.—Plan of aneurysmal varix.

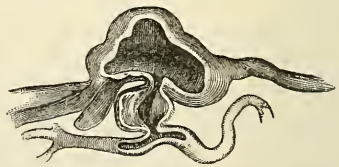


Fig. 200.—Varicose aneurysm (Spence).

a sac of junction (varicose aneurysm, Fig. 200). Wounds produced by a small bullet may result in arteriovenous aneurysm (Matas, in "Transactions of Amer. Surg. Assoc.," vol. xix). Aneurysmal varix is a less grave disorder than varicose aneurysm. Arteriovenous aneurysm used to be most frequent at the bend of the elbow, the vessels being injured during venesection. The condition may occur in the neck, the axilla, the extremities, or the groin. I assisted Professor Keen in an operation upon an aneurysmal varix of the common carotid and internal jugular vein, and assisted Professor Hearn in operating on a varicose aneurysm involving the external iliac vessels. Sir Frederick Treves operated on a case involving the internal maxillary vessels. Very

rarely an arteriovenous aneurysm forms spontaneously. Spontaneous arteriovenous aneurysm is most frequent between the aorta and vena cava. There is no tendency to spontaneous cure in arteriovenous aneurysm. Edema is the rule, muscular atrophy is common, and ulceration or even gangrene of a limb may occur. Matas has collected 17 cases of arteriovenous aneurysm of the subclavian vessels ("Transactions of Amer. Surg. Assoc.," vol. xix). In this list is the celebrated case of his own, a traumatic (gunshot) arteriovenous aneurysm, in which cure followed operation; in the operation it was necessary to obliterate the artery by ligatures, but the venous orifice was closed by sutures without obliterating the lumen of the vein. In the analysis of Matas's paper 15 cases are used, 2 having been noted too late for incorporation; 9 of the cases resulted from "stab or penetrating cut wounds," 6 from bullets—in 5 of the cases the brachial plexus was injured. In 8 out of the 11 unoperated cases the time after the injury when symptoms of arteriovenous aneurysm was noted is stated; in 1 signs were definite within four hours, in 3 they were noted on the second day, in 3 on the third day, in 1 on the sixth day, in 1 on the eighth day, in 1 on the ninth day, and in 1 a few days later. In 3 of the 15 cases secondary hemorrhage followed the injury. Eleven of the 15 cases were treated expectantly; 1 died from secondary hemorrhage and sepsis three weeks after the injury and 10 "survived the immediate effects of the injury, their wounds healing after the cessation of the primary hemorrhage."

In 4 of the 15 cases operation was performed. In 3 the operation was done soon after the injury because of violent secondary hemorrhage. In 1 (Matas's own case) operation was done deliberately to prevent complications. Three of these cases recovered (including Matas's); 1 died of renewed secondary hemorrhage on the twenty-fourth day after operation. Matas points out the fact that in stab-wounds of the subclavian vessels the largest proportion of cases die of primary hemorrhage before assistance is obtained, but in a considerable number of cases temporary hemostasis occurs, which is followed by secondary hemorrhages or arteriovenous aneurysm.

**Symptoms of Aneurysmal Varix.**—The arterial blood is cast forcibly into the vein, and as a consequence the vein becomes enlarged, tortuous, and thickened. The scar of a wound is almost invariably apparent. At the seat of vascular trouble the most marked dilatation exists and it is of bluish color. The swelling pulsates markedly, imparts a sensation to the finger like that felt when the hand is laid upon the back of a purring cat. This thrill or vibration is very characteristic. A sound of a hissing or buzzing nature can be easily heard. The swelling at once disappears on pressure being made upon it or on the artery between it and the heart. It is diminished in size by raising the limb, is increased in size by a dependent position of the limb and by compressing the vein between the heart and the tumor. The adjacent veins are dilated and often the dilatation is manifested over a wide area above and below (Fig. 198), and the thrill and bruit are transmitted a considerable distance. If an extremity is involved it is usually edematous. The parts, as a rule, are painful. The condition progresses, but very slowly, and sometimes years may elapse without any notable aggravation.

**Symptoms of Varicose Aneurysm.**—In this condition we find many of the symptoms of aneurysmal varix, but in varicose aneurysm pressure over the artery of supply between the heart and the lesion does not cause the entire disappearance of the swelling; the veins collapse, it is true, but a distinct sac remains, which may be emptied by direct pressure.

**Treatment.**—The prognosis after operation is better than in ordinary aneurysm, but nevertheless it is wisest to refrain from operating on aneurysmal varix so long as the condition is not progressing obviously, is borne without inconvenience, and is not leading to complications. Varicose aneu-

rysm should be operated upon. If we refrain from operating upon aneurysmal varix the patient should wear a support; but if the part becomes painful or if there seems to be danger of rupture of the vein, operation should be performed. Until recently, when operation was indicated, surgeons advised that each vessel should be tied above and below the opening and a portion of each vessel should be excised, the excised area including the opening. In varicose aneurysm it was the custom to tie each vessel above and below the sac, and excise the sac with a portion of vessel. At the present time surgeons prefer the Matas operation for both varicose aneurysm and aneurysmal varix. In some cases of varicose aneurysm, however, the sac is extirpated and the openings in the vessels closed by suture, and in some cases of aneurysmal varix the adherent vessels are separated and the opening in each is sutured. In a case of aneurysmal varix of the popliteal due to a gunshot-wound, I opened the vein and closed the fistula in the arterial wall. In accomplishing this I gathered so much of the vein wall in the sutures as to render it impossible to suture the vein and retain its lumen. I cut the vein across, a little above and a little below the sutured fistula, used the trapdoor-like piece of vein to reinforce the arterial suture line, and did end-to-end anastomosis of the vein ends. The result was a complete success ("Annals of Surgery," April, 1912).

I attempted a similar operation on a varicose aneurysm of the brachial in the middle of the arm. I found that the superior profunda also ran into the sac, and that a saccular aneurysm had formed on the brachial a little below the varicose aneurysm. I was obliged to ligate the profunda and tie the artery and vein above and below before extirpating the aneurysm. I feared gangrene, but, fortunately, the patient escaped it.

**Cirsoid aneurysm,** or **aneurysm by anastomosis,** consists in great dilatation with pouching and lengthening of one or several arteries. The disease progresses and after a time involves the veins and capillaries. The walls of the arteries become thin and the vessels tend to rupture. Cirsoid aneurysm is most commonly met with upon the forehead and scalp of young people, where it sometimes takes origin from a nevus. It is sometimes seen upon the back or upper extremity. The cause is unknown. Usually there is no assignable cause, but occasionally the condition follows an injury. Pregnancy causes a cirsoid aneurysm to grow rapidly, and so usually does the onset of puberty. Occasionally some of the enlarged vessels fuse and form a great cavity. If rupture occurs, desperate hemorrhage inevitably ensues.

**Symptoms.**—There is a pulsating mass, irregular in outline, composed of dilated, elongated, and tortuous vessels that empty into one another. The mass is soft, can be much reduced by direct pressure, and is diminished by compression of the main artery of supply. A thrill and a bruit exist.

**Treatment.**—In treating a cirsoid aneurysm the ligation of the larger arteries of supply is a wretched failure. Subcutaneous ligation at many points of the diseased area has effected cure in some cases, but it has failed in more. Direct pressure is also entirely useless. Ligation in mass has been successful. Destruction by caustic has its advocates. Electropuncture with circular compression of the arteries of supply has once or twice effected a cure. Injection of astringents has been recommended. Verneuil ligated the afferent arteries, incised the tissues around the tumor, and sank a constricting ligature into the cut. The proper method of treatment is excision after exposure and ligation of every accessible tributary of supply. In a very extensive mass extirpation is impossible; hence one of the other methods suggested must be employed. A very considerable mass can be excised, and the resulting wound should be covered with Thiersch skin-grafts.

**Wounds of arteries** are divided into contused and incised, lacerated, punctured and gunshot-wounds, and vascular ruptures.

**Contused and Incised Wounds.**—A contusion may destroy vitality and be followed by sloughing and hemorrhage. A contusion may rupture a blood-vessel, and is especially apt to do so if the vessel is diseased. Blood is at once effused at the seat of rupture. If an artery is ruptured, there may or may not be a bruit and pulsation over the seat of rupture, pulse is absent below, and the leg below the injury swells and becomes cold. If a large vein ruptures, a blood tumor forms, which does not pulsate and has no bruit, and the limb below becomes intensely edematous. Gangrene is apt to follow the rupture of a main blood-vessel of an extremity. A contusion may rupture the internal and middle coats of an artery, the external coat remaining intact. When this happens the internal coat curls up and the middle coat contracts and retracts, the blood-stream is arrested, and a large clot forms within the artery. If the clot blocks up many collaterals, gangrene may follow, and, as has been pointed out, the gangrene will not be preceded by swelling at the seat of injury, which always occurs if a vessel is ruptured. A contused wound may do little damage, may produce gangrene from thrombosis, or may cause secondary hemorrhage. In an incised wound of an artery there is profuse hemorrhage. The artery after a time is apt to contract and retract, bleeding being thus arrested. A transverse wound causes profuse bleeding, but there is a better chance for natural arrest than in an oblique or in a longitudinal wound. The clot which forms within a cut artery is known as the

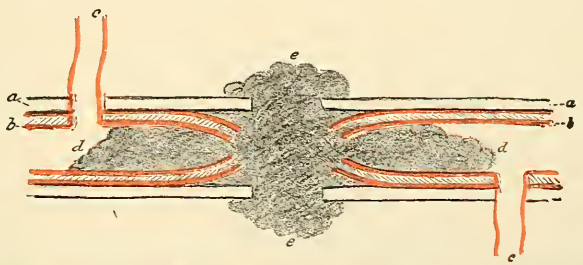



Fig. 201.—Clots formed after division of an artery: 1, 2, 3, Outer, middle, and inner coats; *c*, *c*, branches; *d*, *d*, internal clot; *e*, *e*, external clot.

"internal clot." It used to be taught that the internal clot always reaches as high as the first collateral branch, and subsequently is replaced by fibrous tissue, which permanently obliterates the vessel, and converts it into a shrunken fibrous cord. As a matter of fact, when the parts are aseptic after a ligation the clot is rarely bulky and is often very scanty, repair being quickly effected by proliferation of endothelial cells. Between the vessel and its sheath, over the end of the vessel, and in the surrounding perivascular tissues is the "external clot" (Fig. 201).

A **lacerated wound** of an artery causes little primary hemorrhage. The internal coat curls up, the circular muscular fibers of the media contract upon it, the longitudinal fibers retract and draw the vessel within the sheath, and the external coat becomes a cap over the orifice of the vessel. All of these conditions favor clotting. The vessel wall is so damaged that secondary hemorrhage is usual.

**Punctured Wounds.**—In punctured wounds primary hemorrhage is slight unless a large vessel is punctured. Secondary hemorrhage is not common. Traumatic aneurysm and arteriovenous aneurysm are not unusual results.

**Gunshot-wounds** of arteries by pistol balls and the balls of large-caliber rifles are apt to be contusions which may eventuate in sloughing and secondary hemorrhage or thrombosis and gangrene. A shell-fragment makes a lacerated wound. A military rifle-bullet usually makes a clean-cut division of

an artery, but may contuse it. Secondary hemorrhage after gunshot-wounds is most likely to occur during the third week after the injury. Partial rupture of an artery may cause sloughing and secondary hemorrhage, thrombosis and gangrene, or aneurysm. A complete rupture constitutes a lacerated wound, and is a condition accompanied by diffuse hemorrhage into the tissues.

**Wounds of veins** are classified as are wounds of arteries. The symptom of any vascular wound is hemorrhage.

## Hemorrhage, or Loss of Blood

**Hemorrhage** may arise from wounds of arteries, veins, or capillaries, or from wounds of the three combined. In arterial hemorrhage the blood is scarlet and appears in jets from the proximal end of the vessel, which jets are synchronous with the pulse-beats; the stream, however, never intermits. The stream from the distal end is darker and is not pulsatile. Venous hemorrhage is denoted by the dark hue of the blood and by the continuous stream. In capillary hemorrhage red blood wells up like water from a squeezed sponge, and the color is between the bright red of arterial blood and the dark color of venous blood.

In *subcutaneous hemorrhage* from rupture of a large blood-vessel there are great swelling, cutaneous discoloration, and systemic signs of hemorrhage. If a main artery ruptures in an extremity, there is no pulse below the rupture, and the limb becomes cold and swollen. At the seat of rupture a large fluctuating swelling forms, and sometimes there are bruit and pulsation. If a vein ruptures in an extremity, a large, soft, non-pulsatile swelling arises, there is no bruit, and intense edema occurs below the seat of rupture. Profuse hemorrhage induces constitutional symptoms, and death may occur in a few seconds. Loss of half of the blood (from 4 to 6 pounds) will usually cause death, though women can stand the loss of a greater relative proportion of blood than men. Young children, old people, individuals exhausted by disease, drunkards, sufferers from Bright's disease, diabetes, and sepsis stand loss of blood very badly. An individual with *obstructive jaundice* is apt to suffer from persistent oozing of blood after operation, an oozing which is particularly persistent and dangerous in obstruction of the bile-ducts due to malignant disease. It not unusually causes death. After profuse bleeding has gone on for a time, syncope usually occurs. Syncope is Nature's effort to arrest hemorrhage, for during this state the feeble circulation and the increased coagulability of blood give time for the formation of an external clot. When reaction occurs the clot may hold and be reinforced by an internal clot, or it may be washed away with a renewal of bleeding and syncope. These episodes may be repeated until death supervenes. Nausea exists and there may be regurgitation from the stomach. Vertigo is present. The room may seem to be turning around. There is dimness of vision or everything looks black; black specks float before the eyes (muscæ volitantes), or the patient sees flashes of light or colors. There is a roaring sound in the ears (tinnitus aurium). The patient yawns, is restless, tosses to and fro, casts off the bedclothes, and great thirst is complained of. The mind may be clear, but delirium is not unusual, and convulsions often occur. After a very severe hemorrhage an individual is intensely pale and his skin has a greenish tinge; his lips are blanched, the tongue is cold and white; the brow is covered with cold moisture, the breath is cold; the eyes are fixed in a glassy stare and the pupils are widely dilated, and react slowly to light; the respirations are shallow and sighing; the skin is covered with a cold sweat; the hands are clammy and look like wax; the legs and arms are extremely cold, and the body temperature is below normal. The pulse is very frequent, soft, small, compressible, flut-

tering, or often cannot be detected; the heart is very weak and fluttering, and the arterial tension is almost abolished. There is muscular tremor; the patient tosses about, and asks often and in a feeble voice for water. The suffering from thirst is terrible and no amount of water gives relief. There is often dreadful dyspnea; at each inspiration the nostrils open widely and the accessory muscles of respiration are all in action. A man who is bleeding to death grasps at his chest, makes efforts to rise, and then falls back in a dead faint. Usually reaction occurs after a faint, though the patient is obviously weaker than before; again a faint may happen, and so there is fainting spell after fainting spell until death ensues. Convulsions frequently precede death. In hemorrhage the hemoglobin is greatly diminished in amount. In an *intra-abdominal hemorrhage* the above symptoms are noted, and, except in splenic hemorrhage, blood gathers in both loins, and dulness on percussion exists which gradually rises and shifts as the patient's position is shifted. The blood also gathers in the rectovesical pouch in the male, and in the recto-uterine pouch in the female, and may be detected by digital examination. If the spleen is wounded the blood usually clots quickly, and if it does, an area of dulness, which does not shift and which progressively increases, is noted in the splenic region.

**Treatment.**—When serious hemorrhage exists the surgeon should, when possible, arrest bleeding temporarily, and should then bring about reaction and arrest bleeding permanently. Temporary arrest is not possible in an intra-abdominal hemorrhage. In any case of severe hemorrhage lower the head, and have compression made upon the femorals and subclavians, so as to divert more blood to the brain, or bandage the extremities (autotransfusion). Apply artificial heat. The value of adrenalin in restoring or maintaining arterial tension has been demonstrated by Crile. We should give the patient by hypodermoclysis 1 pint of hot normal salt solution containing 1 dr. of the 1:1000 solution of adrenalin chlorid. The fluid is allowed to run in the subcutaneous tissue beneath the breast. The infusion of 1 pint or more of hot salt solution into a vein is a very valuable remedy; it gives the heart something to contract upon and thus maintains cardiac action. If the depression is very severe, inject ether hypodermatically, then brandy, and then atropin. Strychnin may be given hypodermatically in doses of $\frac{1}{20}$ gr., but atropin is of more service. Digitalin is advised by some, but it is not sufficiently rapid in action. Give enemata of hot coffee and brandy. Apply mustard over the heart and spine. Lay a hot-water bag over the heart.

In hemorrhage from a vessel of an extremity temporarily arrest bleeding while bringing about reaction. Do so by digital pressure in the wound, or, if the bleeding is arterial, by the application of an Esmarch band. In some cases forced flexion is used. As soon as reaction is established, permanently arrest bleeding by the ligature. In intra-abdominal or concealed hemorrhage it is not possible to temporarily arrest it and wait for reaction, but the abdomen must be opened and the work proceeded with in spite of the patient's condition. Every moment we wait he is growing worse.

A severe hemorrhage is apt to be followed by fever, due to the absorption of fibrin ferment from extravasated blood and its action upon a profoundly debilitated system. After a severe hemorrhage leukocytes are increased, not only relatively, but absolutely. Red corpuscles are diminished both relatively and absolutely. Hemoglobin diminishes; many of the corpuscles become irregular and microcytes are noticed.

In treating a patient who has thoroughly reacted after a severe hemorrhage, apply cold to the head. Fluids and ice are grateful. Frequently sponge the skin with alcohol and water. Milk-punch, koumiss, and beef-peptonoids are given at frequent intervals.

**Hemostatic agents** comprise: (1) the ligature and suture; (2) torsion; (3) acupressure; (4) elevation; (5) compression; (6) styptics; (7) the actual cautery; (8) forced flexion of a limb.

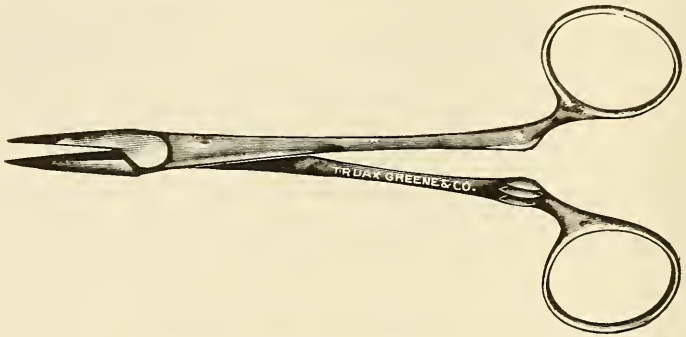



Fig. 202.—Halsted's straight artery forceps.

The *ligature* was known to the ancients, but was rediscovered by Ambroise Paré. The ligature may be made of silk, floss-silk, or catgut. Whatever

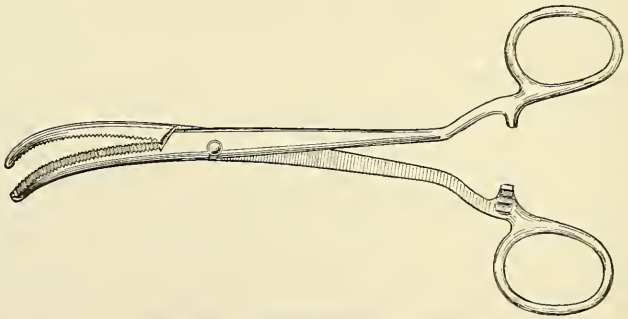


Fig. 203.—Curved hemostatic forceps.

material is used must, of course, be rendered aseptic. A ligature should be about 10 inches long. The vessel to be tied must be drawn out with for-

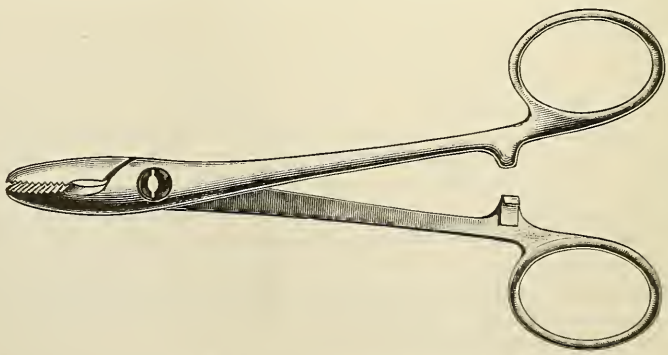


Fig. 204.—Straight hemostatic forceps.

ceps and separated for a short distance from its sheath, but must not be separated to any considerable extent; to do so may lead to necrosis of the

vessel and secondary hemorrhage. The hemostatic forceps (Figs. 202, 203, 204) is in most cases a better instrument than the tenaculum (Fig. 205). The tenaculum makes a hole in the vessel, and sometimes a slit-like tear. A portion of this opening may remain back of the tied ligature, the vessel may retract a little, or the ligature may slip slightly, and bleeding may occur. When the artery lies in dense tissues or is retracted deeply in muscle or fascia,

Fig. 205.—Tenaculum.

the tenaculum, when carefully used, is the better instrument. The ligature is tied in a reef-knot (Fig. 206), not in a granny-knot (Fig. 207) and not in a surgeon's knot (Fig. 208). It is often the purpose of the surgeon to divide the internal and middle coats of the vessel, and if such is his desire the first knot is firmly tied. The second knot must not be tied too tightly or it will cut the ligature. The ligature must not be jerked as it is being tied. If a third knot

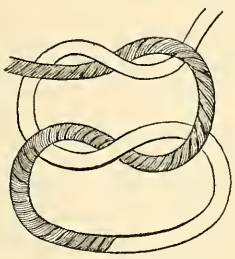


Fig. 206.—Method of tying square or reef-knot.

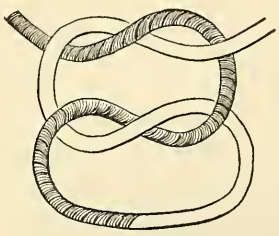


Fig. 207.—Method of tying granny-knot.

overlies the first two, the ligature can be cut off close to the knot, otherwise it is cut off so that short ends are left. Both ends of a divided vessel should be ligated. If a vessel is atheromatous, it is not desirable to divide the internal and middle coats. In this case a ligature should be applied firmly rather than tightly, and another ligature should be put on above it, or ligation can be effected by the stay knot. If an artery is incompletely divided, a ligature should be applied on each side of the wound and the vessel divided between the ligatures.

When the parts about an artery are so thickened that the vessel cannot be drawn out, arm a curved Hagedorn needle (Fig. 209) with catgut and pass the latter around the vessel in such a manner that the catgut will include the vessel with some of the surrounding tissue. Then tie the ligature (Fig. 211). This method is known as the application of a *suture-ligature*, and is pursued in necrosis, atheroma, scar-tissue, sloughing, etc. Never include a nerve of any size in the ligature. If this mode of ligation fails, we may try acupressure.

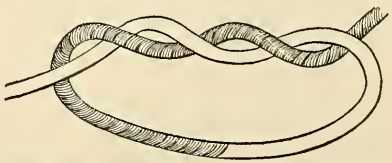


Fig. 208.—Method of tying surgeon's knot.

Doyen, when about to tie a thick pedicle, crushes it by means of a very powerful instrument and then ties a ligature about the crushed and attenuated area. The vessels are closed by laceration wide of the ligature and the ligature does not tend to slip. Some trust such a stump without a ligature, but most surgeons prefer to ligate. This instrument is known as the vasotribe or angiotribe and is used particularly in hysterectomy. Figure 212 shows a vasotribe.

Veins are ligated as are arteries. If a large vein is torn, we wish, if possible, to control hemorrhage without obliterating the lumen of the vein by ligation.

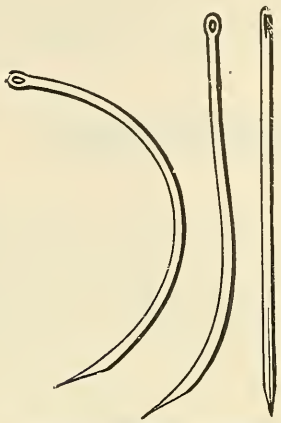


Fig. 209.—Hagedorn's needles.

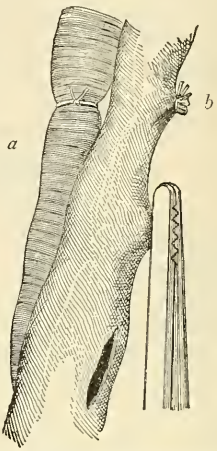



Fig. 210.—Method of controlling hemorrhage by ligature (after Esmarch): *a*, Artery ligated; *b*, lateral ligature of vein.

If the wound is not greater in length than the measure of the diameter of the lumen, a lateral ligature may be used. It is practically always used in small transverse wounds. In order to apply a lateral ligature the two lips of the vein wound are seized by forceps and drawn out into a tit (Figs. 210 and 227). A ligature is placed around the base of the cone and tied. The pull in the cone is relaxed while the first knot is being tied in order that the ligature may constrict tightly. In a large vessel the thread should be passed by a needle through the outer coats of the vein before it is used to encircle the cone. This plan prevents slipping. In some cases when a lateral ligature or suture cannot be applied, forcipressure will succeed. One or more clamp forceps are applied and are left in place for several days.

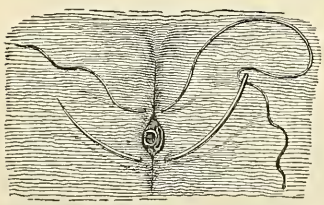


Fig. 211.—Arrest of hemorrhage by passing a suture-ligature.

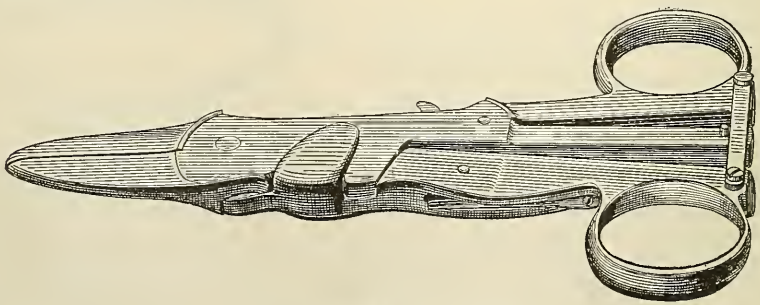


Fig. 212.—Vasotribe of Doyen.

*Phleborrhaphy* is suture of a vein, with preservation of the lumen of the vessel. It is used when complete ligation is undesirable (as in a large vein), and when lateral ligation without obliteration of lumen is impossible. It is

commonly employed for longitudinal wounds and for wounds in any direction when the length of the wound is greater than the diameter of the vessel. Fine catgut or silk may be used. An intestinal needle threaded with silk is entirely satisfactory. The thread is passed through the external coat and part of the middle coat on each side of the wound. Interrupted sutures are employed and thus the two lips of the wound are approximated. A vein completely divided across can be united by end-to-end suturing. Figure 213 shows the operation of phleborrhaphy.

By suturing I successfully closed a tear in the innominate vein inflicted during the removal of a retrosternal goiter, and also a considerable longitudinal tear in the internal jugular vein inflicted during the removal of lymph-nodes.

Murphy and Damar Harrison each succeeded in suturing a wound of the inferior vena cava. Bier successfully applied sutures in 2 cases of wound of the inferior vena cava.

Israel points out how difficult it is to suture in the depths of a cavity full of blood. This complication always exists in wounds of the inferior cava, and in them it may be necessary to use forcipressure. He did this in 2 cases.

In a case of Körte's the cava was wounded during nephrectomy. Forcipressure and lateral sutures were found impracticable. The cava was tied. Thrombophlebitis arose in both legs, but the patient eventually recovered ("Zentralbl. für Chir.," August 8, 1911).

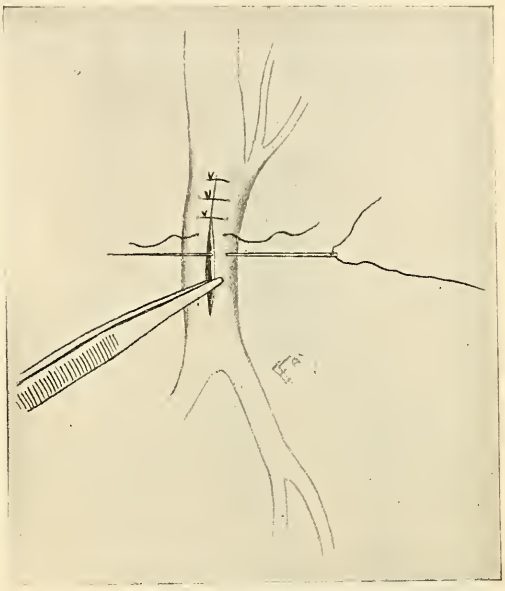


Fig. 213.—Phleborrhaphy: Forceps are seen everting lip of wound for passage of needle and interrupted sutures (Bickham).

*Arteriorrhaphy.*—The studies of Carrel, Murphy, Matas, Abbe, and others have shown that wounded arteries can be repaired by suturing; that a portion of an artery can be removed and repair be obtained by end-to-end suturing, implantation, or lateral anastomosis; that an artery can be obliterated by suturing the intima from within; that an artery can be anastomosed into a vein, and that after resection of a portion of an artery vascular integrity may be restored by suturing into the gap a portion of a vein or artery recently resected.

We now suture certain wounds in large vessels which until very recently would have caused us to completely ligate the artery. In extirpating malignant tumors it is sometimes necessary to remove large arteries or veins. This may cause grave danger of gangrene, and we now may attempt to prevent gangrene by the restoration of vascular continuity.

The wonderful experiments of Carrel on the transplantation of organs and the brilliant studies and operations of Matas and Murphy have been the great influences that have brought vessel suture into the field of practical surgery.

There is yet much to learn. What we do know is really little, but we are probably at the threshold of great events.

We know that we can close by suture a lateral wound or a transverse wound

of less than half the circumference of the vessel; that we can perform end-to-end suturing; that we can insert a piece of resected vein to re-establish vascular continuity; and that after such an operation the blood-current will be re-established. We do not know how long the circulation will continue after re-establishment. A sutured artery will certainly carry blood for a time, but in most cases, at least, only for a time, the ultimate fate of the vessel being obliteration by endothelial proliferation. If the vessel operated upon is diseased, obliteration by clot is practically certain to ensue. But even temporary re-establishment of circulation is of the greatest value. Even though the lumen is finally closed, the closure is gradual. While the vessel is closing the collaterals are dilating. By the time one source of supply for the tissues is cut off, another

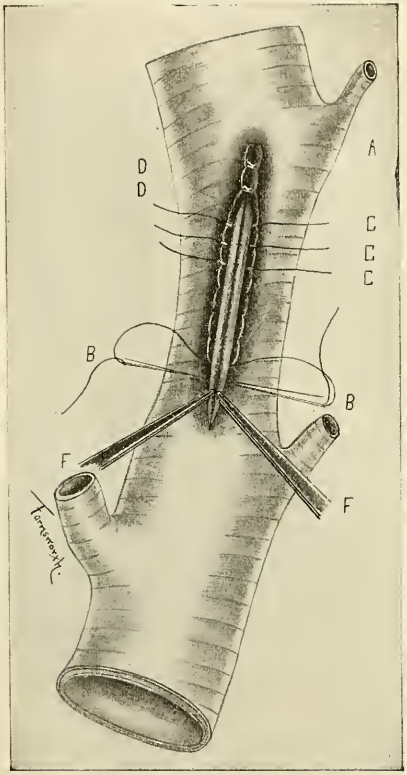



Fig. 214.—Repair of longitudinal wound of artery by combination cobbler's stitch through all coats, and interrupted sutures through outer coats, as suggested by the author: A, Beginning of cobbler's stitch through all coats; B, B, needles in act of passing through same opening in opposite directions, in characteristic cobbler fashion; C, C, C, three interrupted sutures through outer coats, ready to be tied; D, D, two interrupted sutures tied, passing through outer coats (Bickham).

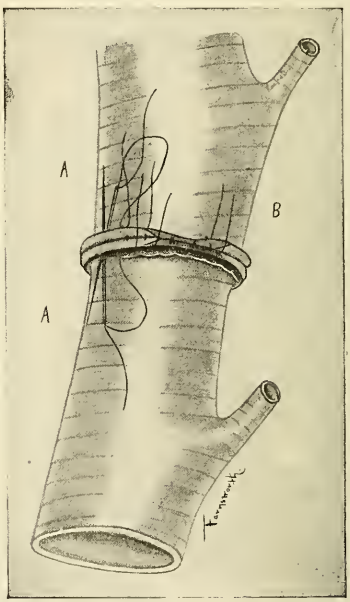



Fig. 215.—Repair of complete transverse division of artery by combination cobbler's stitch through all coats, followed by interrupted sutures through outer coats, as suggested by the author: A, A, Needles passing in opposite directions through all coats, in act of placing cobbler's stitch; B, superficial tier of interrupted stitches through outer coats, showing three untied and two tied (Bickham).

has come into being. Thus gangrene is prevented. There appears to be a certain amount of danger of the development of aneurysm at the seat of suturing. In a longitudinal wound of an artery or in a transverse wound of not over half the circumference of the vessel, the wound may be closed by interrupted sutures, passing the threads through the two outer coats and bringing the wound edges together without inversion. Floss silk is used, and it should be as large as the eye of a curved conjunctival needle will carry in order to lessen the danger of leaking of blood through the stitch holes. The sheath is sutured over the stitch line ("Bickham's Operative Surgery").

A better plan in such wounds is that of Bickham, viz., a cobbler's stitch through all the coats to bring the intima of each lip together and interrupted sutures through the outer coats (Fig. 215). An oblique wound may be repaired

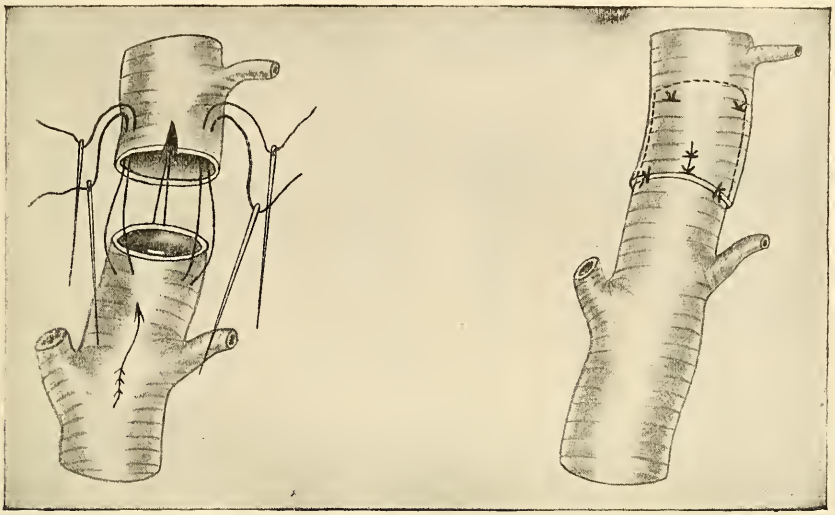


Fig. 216.—Arteriorrhaphy in complete circular division of an artery (Murphy's method): A, Intussusception, with sutures passing through outer and middle coats; B, intussuscipiens (split to aid invagination), with sutures passing through all coats; C, showing all sutures tied (Bickham).

in the same manner. If a vessel is divided transversely through more than half of its circumference Murphy believes that the division should be made complete as a preliminary to suturing.

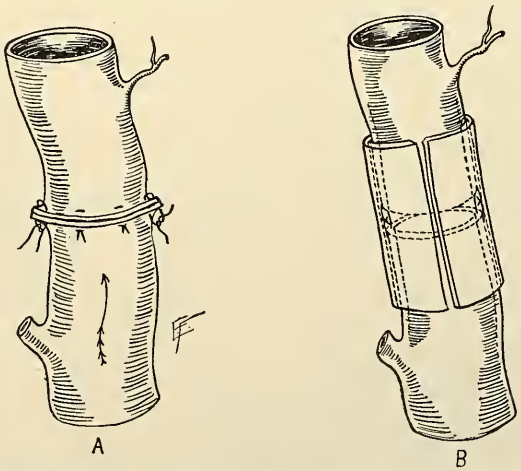



Fig. 217.—Circular arteriorrhaphy in complete division of an artery: A, Method of Salomoni and Tomaselli—interrupted sutures through all coats; B, method of Gluck—interrupted sutures through outer coats, protected by cylinder of decalcified bone, ivory, or rubber (Bickham).

Complete circular division may be treated by Murphy's plan, viz., invagination of one end of the cut vessel into the other end split to receive it (Figs. 215 and 216), by Bickham's plan of a cobbler's stitch followed by interrupted sutures, or by one of the plans shown in Fig. 217.

The author successfully sutured a small wound in the axillary artery. Stewart removed a clot from the femoral and sutured the vessel. Murphy removed a clot from the external iliac artery and sutured the vessel. Braun ("Zentralblatt für Chirurgie," August 29, 1908) resected a portion of the aorta along with a tumor and performed circular suture. Depage successfully sutured the common carotid artery. Pringle sutured a wound of the external iliac artery. The wound was $\frac{1}{4}$ inch in length. Dejemil Pasha sutured a lacerated wound of the axillary artery. Torance sutured a wounded brachial artery. Martin sutured a lacerated wound of the femoral artery ("Annals of Surgery," Oct., 1905). Henderson sutured a transverse wound extending half across the femoral artery ("American Medicine," Jan. 14, 1905). A. E. Halstead cut two-thirds through the circumference of the axillary artery and sutured the wound directly instead of making the wound first of all a complete division, as advised by Murphy. Two months later the radial pulse was present.

Faykiss made an end-to-end suture of a divided carotid artery ("Centralb. für Chirurgie," 1908, xxxv).

Lexer ("Archiv. für klinische Chirurgie," 1907, No. 2) resected the sac of a traumatic arteriovenous aneurysm of the popliteal vessels, removing at the same time 5 cm. of artery and as much vein.

He performed end-to-end anastomosis of each vessel over a thin magnesium tube (*Payr's tube*). The pulse was restored. Nine months after operation the pulse was present below the seat of operation, but was weaker than it had been some months before. In this paper Lexer speaks of a case of diffuse aneurysm of the axillary artery. The surgeon resected the sac and a portion of the artery. The gap of over 3 inches was filled by a piece of internal saphenous vein sutured in place (*autoplasty*). The circulation was re-established in the limb, but the patient died of complications. Postmortem demonstrated that the transplanted vein was patent and the sutures firm. There was a clot above the piece of vein due to the action of the clamp upon a sclerotic artery. Gangrene began on the fourth day after operation.

Manteuffel successfully sutured a wound of the femoral artery. This was the first case to be recorded (Bloodgood, in "Progressive Medicine," Dec. 1, 1908), although Hallowell, in 1759, successfully closed a wound in the brachial artery by means of a harelip pin and a ligature (Mumford). Bloodgood in the same article notes that Körte, in 1904, performed lateral suture of the popliteal artery and vein, and that Garré closed a wound in the popliteal artery by suture, and in the vein of the same patient by lateral ligature.

Murphy was the first surgeon to succeed in resecting a portion of a large artery (the femoral) and doing end-to-end suturing ("Med. Record," Jan. 16, 1897).

Lund ("Annals of Surgery," March, 1909) sutured a double stab-wound of the femoral artery and vein, and six months later there was a pulse in the dorsalis pedis. Routier tore the aorta in removing a hypernephroma of the kidney. He sutured the wound with catgut. The patient died on the fourteenth day, but not from hemorrhage ("Gaz. des Hôpitaux," 1911). Mantelli closed a gap in the femoral artery by a segment of a vein (autoplastic graft). Buchanan performed circular resection and suture of the axillary artery, the vessel having been lacerated by the bone fragments after fracture-dislocation of the anatomical neck of the humerus. Buchanan has collected 29 instances of end-to-end suture of divided arteries ("Surgery, Gynecology, and Obstetrics," Dec., 1912).

Among other surgeons reporting successful cases of arterial suture are Heidenhain, Orlow, Pean, Baum, Sherman, Krause, Payr, Kummell, Seggel, Lindner, and Brougham.

*Torsion* was practised by the ancients, but was reintroduced in modern times, particularly by Amussat, Velpeau, Syme, and Bryant, of London.

By means of torsion the internal and middle coats are ruptured and the external coat is twisted. The middle coat retracts and contracts, and the inner coat inverts into the lumen of the artery. It is a safe procedure, and is practised upon vessels as large as the femoral by many surgeons of high standing. Before the days of asepsis torsion possessed the signal merit of not introducing possible infection in ligatures. At the present time it offers no particular advantage. It is no quicker than the ligature, and damages the vessel so much that necrosis may occur. It cannot be used if the vessels are diseased. In what is known as free torsion the vessel is grasped, drawn out, and twisted until the free end of the vessel is twisted off. Limited torsion is more often used. The vessel is drawn out of its sheath by a pair of forceps held horizontally, and is grasped a little distance above its extremity by another pair of forceps held vertically (Fig. 218). The first instrument is used to twist the artery six to eight times.

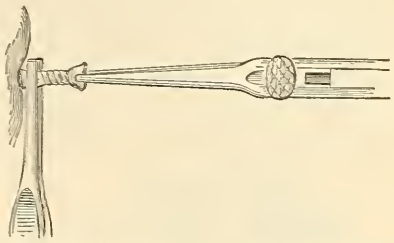


Fig. 218.—Method of controlling hemorrhage by torsion.

*Acupressure* is pressure applied by means of a long pin. The method of hemostasis by acupressure was devised by Sir James Y. Simpson. A pin is simply passed under a vessel (transfixion), leaving a little tissue on each side between the pin and vessel. A pin can be passed under a vessel, and a wire be thrown over the pin and twisted (circumclusion). The pin can be inserted upon one side, passed through ½ inch of tissues up to the vessel, be given a quarter twist, and be driven into the tissues across the artery (torsoclusion). Some tissue may be picked up on the pin, folded over the vessel, and pinned to the other side (retroclusion). Acupressure is occasionally used to arrest hemorrhage if vessels are inflamed or atheromatous, in sloughing wounds, in scar-tissue, and when a ligature will not hold firmly.

*Elevation* is used as a temporary expedient or in association with some other method. It is of use in a wound of a bursa, in bleeding from a ruptured varicose vein, and its use is frequently associated with compression.

*Compression* is either direct or indirect—that is, in the wound or upon its artery of supply. In the removal of the upper jaw arrest bleeding by plugging. In injury of a cerebral sinus plug with gauze. Compression and hot water (115°–120° F.) will stop capillary bleeding. A graduated compress was formerly recommended in hemorrhage from the palmar arch (Fig. 220). A compress will arrest bleeding from superficial veins. The knotted bandage of the scalp will arrest bleeding from the temporal artery. Long-continued pressure causes pain and inflammation.

Fig. 219.—Tamponade of intercostal artery (after Von Langenbeck).

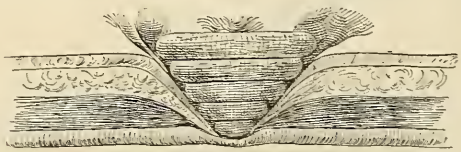


Fig. 220.—Conical aseptic tampon (graduated compress) compressing an artery (Senn).

Indirect compression is used to prevent hemorrhage or to temporarily

arrest it. It may be effected by encircling a limb above a bleeding point with an Esmarch band or by applying a tourniquet or an improvised tourniquet (Fig. 221). It may also be effected by a clamp. Crile has devised a clamp to effect temporary closure of the carotid artery. In operations about the head one or both carotids may be closed for a considerable time and bleeding may thus be largely prevented. In 10 cases Crile temporarily closed both carotids. A hypodermatic injection of atropin is given to prevent inhibition, the vessels are exposed, and the clamps are applied with just sufficient firmness to approximate the vessel walls. No clot will form if the walls are not compressed. The patient is in the Trendelenburg position. If it is found that respiratory difficulty occurs, one clamp must be loosened. After the completion of the operation the patient must be brought to the horizontal before the clamps are removed (Crile, in "Annals of Surgery," April, 1902).

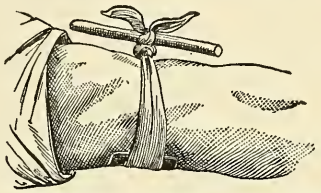


Fig. 221.—Impromptu tourniquet for compressing an artery with a handkerchief and a stick.

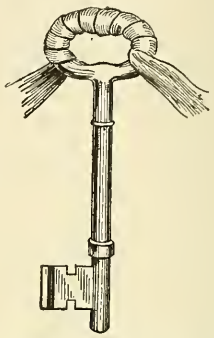


Fig. 222.—Handle of door-key, padded.

Digital compression is a form of indirect compression. It can be maintained for only a few minutes by one person, but a relay of assistants can carry it out for a considerable time. In compressing the subclavian artery wrap a key as shown in Fig. 222, and compress the artery against the outer surface of the first rib. The shoulder must be depressed and pressure applied in the angle between the posterior border of the sternocleidomastoid and the upper border of the clavicle. The direction of the pressure should be downward, backward, and inward.

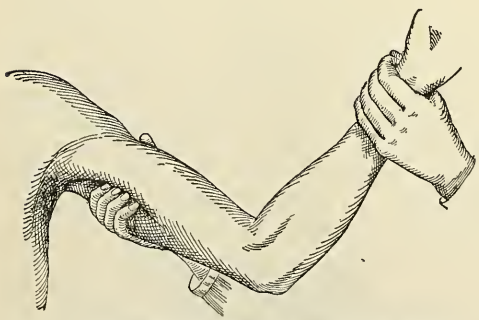


Fig. 223.—Digital compression of the brachial artery.

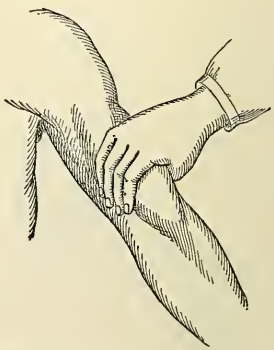


Fig. 224.—Digital compression of the brachial artery.

The brachial artery can be compressed against the humerus. In the upper part of the course of the artery the pressure should be from within outward (Fig. 223), in the lower part from before backward (Fig. 224). The abdomi-

nal aorta can be compressed by Macewen's method (*q. v.*). The common iliac can be compressed through the rectum by means of a round piece of wood known as Davy's lever. The femoral artery can be compressed just below Poupart's ligament against the psoas muscle and head of the femur (Fig. 225). The pressure should be directly backward. In the middle third of the thigh digital compression is unsatisfactory, and a tourniquet should always be used or an Esmarch band be employed.

*Forced flexion* is a variety of indirect compression introduced by Adelmann. It will arrest bleeding below the point compressed, but soon becomes intensely painful. Forced flexion can be maintained by bandages. Brachial hyperflexion is maintained by tying the forearm to the arm. It is often associated with the use of a pad in front of the elbow. Genuflexion is maintained by tying the foot to the thigh. It is increased in efficiency by placing a pad in the popliteal space.

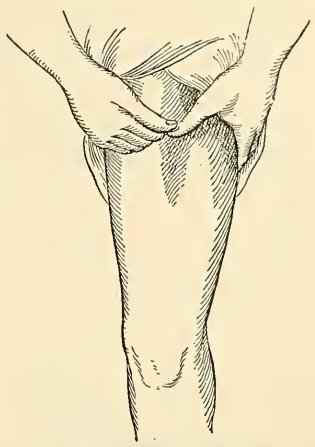


Fig. 225.—Digital compression of the femoral artery.

*Styptics.*—Chemicals are now rarely used to arrest hemorrhage. In epistaxis we may pack with plugs of gauze saturated with a 10 per cent. solution of antipyrin. In bleeding from a tooth-socket freeze with chlorid of ethyl spray, and then pack with gauze soaked with 10 per cent. solution of antipyrin or pack with dry sponge or styptic cotton (absorbent cotton soaked in Monsel's solution and dried). A bit of cork may be forced into the socket. In bleeding from an incised urinary meatus pack with styptic cotton and compress the lips of the meatus. Cold water, chlorid of ethyl spray, and ice act as styptics by producing reflex vascular contraction. Hot water produces contraction and coagulates the albumin. The temperature should be from 115° to 120° F. A mixture of equal parts of alcohol and water stops capillary oozing.

*The Use of Gelatin in Controlling Hemorrhage.*—It seems very positively proved that gelatin increases the coagulability of the blood if given hypodermatically. It has been shown by Horatio C. Wood, Jr. ("American Medicine," May 3, 1902), that, even when administered by the stomach, digestion does not destroy its coagulating effect upon the blood. Carnot, of Paris, used it locally and with success to control epistaxis in a sufferer from hemophilia He then employed it to arrest bleeding from hemorrhoids, tumors, and incised wounds; and demonstrated in animals that it will arrest oozing from the cut surface of the liver. Carnot used a 5 or 10 per cent. solution. It has been employed with success to control hemorrhage in many situations, is of value when applied locally, and possibly of use when injected subcutaneously.

Intravenous injections are extremely dangerous, and are apt to be followed by embolism. Subcutaneous injections are decidedly painful and are not altogether safe, producing albuminuria and occasional embolism. Another danger that may follow the subcutaneous administration of gelatin is the development of tetanus, and several cases have been reported. The existence of disease of the kidneys contra-indicates the hypodermatic use of gelatin.

It has been successfully used as an enema in intestinal hemorrhage, and as an injection in hemorrhage from the bladder. I have used it with success in arresting bleeding from the cut surface of the human liver; to check bleed-

ing from an incised wound in a victim of leukemia; to arrest postoperative oozing in sufferers from cholemia; and in several cases of severe epistaxis.

When employed locally in solution, it should be of a strength of from 2 to 10 per cent. in normal salt solution. For hypodermatic use some employ a 5 per cent., some a 2 per cent., and some a 1 per cent. solution. In using a 1 or 2 per cent. solution a very large amount of fluid must be injected. This causes pain; and Sailer maintains that the pain is slight or absent if the solution is not turbid and if but 10 c.c. of a 10 per cent. solution are injected. The injection may be repeated until from 1 to 3 gm. of gelatin have been administered. It should be injected on the outer side of the thigh, under the breast, or between the shoulder-blades. If the drug is given by mouth, 100 c.c. of a 10 per cent. solution is the dose, and this may be repeated every two or three hours.

On account of the possible danger of the development of lockjaw after infection great care in sterilizing must always be exercised. The method of preparation suggested by Joseph Sailer will be found of the greatest value. (For the formula for this see page 422.)

In view of the fact that gelatin is such an excellent culture material, whenever it is used in the rectum, nose, pharynx, vagina, or bladder it should be mixed with some antiseptic agent.

The exact mode in which gelatin acts in producing coagulation is not certain. Floresco maintains that it acts like an acid. Laborde states that undissolved particles of gelatin serve as centers for coagulation. Other experimenters insist that gelatin destroys the leukocytes, and thus liberates fibrin ferment.

*Suprarenal extract* is a valuable agent to control capillary oozing. It constricts capillaries, and if applied to a mucous membrane will rapidly blanch it. It is extensively used to check bleeding during operations on the nose, throat, larynx, and ear, and to arrest epistaxis and bleeding from the uterus. The solution to employ is adrenalin chlorid of a strength of from 1:10,000 to 1:1000. A piece of cotton soaked in this solution is pressed lightly upon the part or it is sprayed upon the part by an atomizer.

*Chlorid of calcium*, given internally, is said to favor coagulation of the blood and is used to check oozing or to prevent hemorrhage. It is used particularly in jaundice cases when operation must be performed. If given several times a day for two or three days it increases the coagulability of the blood; but if given for more than four days, actually diminishes it. The initial dose is from 15 to 30 gr., then 5 gr. every hour are given until five or six doses have been taken. It is apt to provoke gastric irritability, and it is often given by the rectum. I have never been convinced of the value of the drug.

**Blood-serum.**—In hemophiliacs and in the postoperative oozing of jaundiced patients blood-serum is of great value. The wound may be tamponed with fresh animal blood or blood-serum. A suitable material for local use can be made by grinding up the fresh liver of an animal, soaking it in water, filtering the mixture through gauze, soaking gauze in the filtrate, and tamponing the wound with the wet gauze. This fluid contains the thrombokinase necessary in coagulation (Kottmann and Lidsky, in "Deutsch. medizin. Wochen," 1910, vol. i). Human serum is even more efficient locally. In ordinary cases of hemorrhage serum may be given subcutaneously in very severe hemorrhage intravenously. In jaundice cases a serum injection should precede operation. As a prophylactic or for therapeutic effect human serum, horse serum, rabbit serum, antistreptococcus serum, or diphtheria antitoxic serum may be used. The dose is 10 to 40 c.c. subcutaneously.

The *actual cautery* is a very ancient hemostatic. It is still used occasionally after excising the upper jaw, in bleeding after the removal of some malignant growths, in continued hemorrhage from the prostatic plexus of veins after lateral lithotomy, and to stop oozing after the excision of venereal warts. We are often driven to its use in "bleeders," that is, those persons who have a hemorrhagic diathesis, and who may die from having a tooth pulled or from receiving a scratch. It will arrest hemorrhage, but the necrosed tissue separates, and when it separates secondary hemorrhage is apt to set in. The iron for hemostatic purposes must be at a cherry heat. The old-fashioned iron, which was heated in a charcoal furnace, is rarely used. It is large, clumsy, and cools quickly if the bleeding is profuse. In an emergency we may heat a poker or a coil of telegraph wire. The best instrument is the Paquelin cautery. The Paquelin cautery consists of an alcohol lamp, a metal chamber containing benzene, a tube of entrance for air containing two bulbs, an exit tube, and a wooden-handled cautery instrument, the tip of which is hollow and composed of platinum (Fig. 226). The tip can be kept hot even when bleeding is profuse. If the iron is very hot, it will not stop bleeding completely. In order to use the Paquelin cautery, light the lamp, heat the cautery-tip in the flame until it becomes red, remove it from the flame, and squeeze the bulb repeatedly until the tip becomes bright red. Each time the bulb which is not covered with netting is squeezed air is driven through the metal chamber into the tube and cautery, and this air carries with it the vapor of benzene, which passes to the hot tip and takes fire. The degree of heat maintained depends upon the rapidity with which the bulb is squeezed.

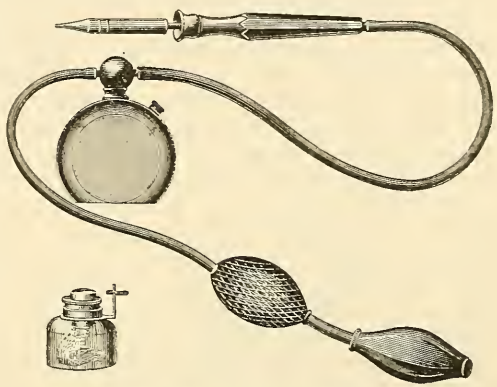


Fig. 226.—Paquelin cautery.

Skene has devised a method known as electrohemostasis. He grasps the vessel or tissue with specially constructed forceps, an electric current generates heat, the tissue is cooked, and the walls of the vessel united. For the small instrument Skene uses a current of 2 ma. and for the larger instrument a current of 8 ma.[1]

Downes has devised an instrument to apply electrothermic hemostasis in abdominal and pelvic operations. He asserts that by this method an intra-abdominal operation can be rendered bloodless; that the lymph-ducts are sealed and the stump rendered sterile; that adhesions are less apt to form; and that there is less postoperative pain than if the ligature were used ("Boston Med. and Surg. Jour.," July 10, 1902).

*Rules for Arresting Primary Hemorrhage.*—1. In arterial hemorrhage tie the artery in the wound, enlarging the wound if necessary (*Guthrie's rule*). In tying the main artery of the limb in continuity for bleeding from a point below we fail to cut off the bleeding from the distal extremity, and hemorrhage is bound to recur. If the surgeon does not look into the wound, he cannot know what is cut: it may be only a branch and not a main trunk. The same rule obtains in secondary hemorrhage.[2]

2. We can safely ligate veins as we would arteries.

[1] "New York Medical Journal," Feb. 18, 1898.

[2] For observations on suture of vessels, see page 444.

3. In a wound of the superficial palmar arch tie both ends of the divided vessel.

4. In a wound of the deep palmar arch enlarge the wound, if necessary, in the direction of the flexor tendons, at the same time maintaining pressure upon the brachial artery. Catch the ends of the arch by hemostatic forceps and tie both ends. If the artery can be caught by, but cannot be tied over the point of the forceps, leave the instrument in place for four days. If the artery cannot be caught by forceps, use a tenaculum. The ends of the divided vessel can be caught and must be caught even if large incisions are needed to effect it. An incision which will probably always expose the vessel is as follows: Make a cut on a line with the injury from the web of the fingers to above the carpus, separating the metacarpal and carpal bones, until the artery is reached. (This is really Mynter's incision for excision of the wrist.) In former days, if the surgeon found trouble in grasping the ends of the vessel, he applied a graduated compress (see Fig. 220). This is applied as follows: Insert a small piece of gauze in the depths of the wound, put over this a larger piece, and keep on adding bit after bit, each successive piece larger than its predecessor, until there exists a conical pad, the apex of which is at the point of hemorrhage and the base of which is external to the surface of the palm. Bandage each finger and the thumb, put a piece of metal over the pad, wrap the hand in gauze, bandage each finger, the thumb, palm, and wrist, place the arm upon a straight splint, apply firmly an ascending spiral reverse bandage of the arm, starting as a figure-of-8 of the wrist, and hang the hand in a sling. Instead of applying a splint, we may place a pad in front of the elbow and flex the forearm on the arm. The palmar pad is left in place for six or seven days unless bleeding continues or recurs. The graduated compress is unreliable, hence it is a dangerous method of treatment. It is an evasion. It should be employed at the present time only as a temporary expedient until ligatures can be applied. The old rule of surgery was as follows: If bleeding is maintained or begins again after application of a graduated compress, ligate the radial and ulnar arteries. If this maneuver fails, we know that the interosseous artery is furnishing the blood and that the brachial must be tied at the bend of the elbow. If this fails, amputate the hand. At the present day it is hard to conceive of such radical procedures being necessary for hemorrhage from a palmar vessel.

5. In primary hemorrhage, if the bleeding ceases, do not disturb the parts to look for the vessel. If the vessel is clearly seen in the wound, tie it; otherwise do not, as the bleeding may not recur. This rule does not hold good when a large artery is probably cut, when the subject will require transportation (as on the battlefield), when a man has delirium tremens or mania, or when he is a heavy drinker. In these cases always look for the artery and tie it.

6. When a person is bleeding to death from a wound of an extremity, arrest hemorrhage temporarily by digital pressure in the wound and apply above the wound a tourniquet or Esmarch bandage. Bring about reaction and then ligate, but do not operate during collapse if the bleeding can be controlled by pressure.

7. If a transverse cut incompletely divides an artery, it may be found possible and may be considered desirable to suture the cut. Longitudinal cuts can certainly be sutured (see page 441). If suturing is impossible, or if the surgeon prefers not to attempt it, apply a ligature on each side of the vessel wound and then sever the artery so as to permit of complete retraction.

8. If a branch comes off just below the ligature, tie the branch as well as the main trunk.

9. If a branch of an artery is divided very close to a main trunk, the rule

used to be, tie the branch and also the main trunk. It was thought that if the branch alone were tied the internal clot, being very short, would be washed away by the blood-current of the larger vessel. We now know that the clot is not required in repair, and under aseptic conditions it is trivial in size and rarely reaches the first collateral branch. Repair is effected by endothelial proliferation.

10. If a large vein is slightly torn, put a lateral ligature upon its wall (Fig. 227). Gather the rent and the tissue around it in a forceps and tie the pursed-up mass of vein wall. It is a wise plan to pass the ligature through the two outer coats by means of a needle and tie the knot subsequently. This expedient prevents slipping. If a longitudinal wound exists in a large vein, take an intestinal needle and fine silk and sew it up (see page 440). Transverse wounds can also be sutured.

11. When a branch of a large vein is torn close to the main trunk, tie the branch and not the main trunk. Apply practically a lateral ligature.

12. If, after tying the cardial extremity of a cut artery, the distal extremity cannot be found, even after enlarging the wound and making a careful search, firmly pack the wound.

13. In bleeding from diploë or cancellous bone, use Horsley's antiseptic wax, or break in bony septa with a chisel, or plug with threads of gauze or scrapings of catgut. If the bleeding is very free, wax will not stick and mash-

Fig. 227.—Application of lateral ligature to a vein.

ing the bone edges usually fails. The expedient suggested by Vaughan should then be employed, viz., a piece of muscle or other tissue is cut off, and, by means of the fingers or a knife handle, forcibly rubbed against the bleeding bone surface. Minute fragments of the soft tissue plug the open vessels and arrest the bleeding (George Tully Vaughan, in "Jour. Am. Med. Assoc.," Nov. 9, 1907).

14. In bleeding from a vessel in a bony canal, plug the canal with an antiseptic stick and break the wood, or fill up the orifice of the canal with antiseptic wax or a separated bit of tissue. If this fails, ligate the artery of supply.

15. In bleeding from the internal mammary artery the old rule was to pass a large curved needle holding a piece of silk into the chest, under the vessel and out again, and tie the thread tightly; but it is better to make an incision and ligate the artery.

16. In bleeding from an intercostal artery make pressure upward and outward by a tampon (see Fig. 219), or throw a ligature by means of a curved needle entirely over a rib, tying it externally; or, what is better, resect a rib and tie the artery.

17. In collapse due to puncture of a deep vessel, the bleeding having ceased, do not hurry reaction by stimulants. Give the clot a chance to hold. Wrap the sufferer in hot blankets. If the condition is dangerous, however, stimulate to save life.

18. In punctured wounds, as a rule, try pressure before using ligation.

19. After a severe hemorrhage *always* put the patient to bed and elevate the damaged part (if it be an extremity or the head).

20. A clot which holds for twelve hours after a primary hemorrhage will probably hold permanently; but even after twelve hours be watchful and insist on rest.

21. If recurrence of a hemorrhage from a limb is feared, mark with anilin or iodin the spot on the main artery where compression is to be applied, apply a tourniquet loosely, and order the nurse to screw it up and to send for the physician at the first sign of renewed bleeding. This must be done in many gunshot-wounds.

22. When the femoral vein is divided high up, the advice commonly given is to ligate the vein and also the femoral artery. Braune taught that because of the venous valves there is no collateral circulation, and to tie the vein alone renders gangrene inevitable. Niebergall shows that the valves may be overcome by moderate arterial pressure, and thus collateral circulation be established. Hence, when the femoral vein is divided tie the vein, but leave the artery untied, so as to furnish the necessary pressure.[1]

23. In extradural hemorrhage, trephine. The side to be trephined is determined by the symptoms, and not by the situation of the injury. The opening is made on a level with the upper orbital border and $1\frac{1}{4}$ inches behind the external angular process. This opening exposes the middle meningeal and its anterior branch. If this does not expose a clot, trephine over the posterior branch, on the same level and just below the parietal eminence. When the clot is found, enlarge the opening with the rongeur, scoop out the clot, and arrest the bleeding by passing ligatures on each side of the injury in the vessel through the dura, under the artery and out again, and then tying them. If the artery lies in a bony canal, plug the canal with Horsley's wax. In some cases packing must be used to arrest bleeding. In subdural hemorrhage open the dura and endeavor to ligate. If this procedure is impossible, pack with iodoform gauze.

24. In hemorrhage from a cerebral sinus catch the edges of the opening with forceps, if possible, and apply a lateral ligature, or leave the forceps in place for forty-eight hours, or compress firmly with iodoform gauze.

25. In extramedullary spinal hemorrhage rapidly advancing and threatening life perform laminectomy and arrest the hemorrhage.

26. In bleeding from a tooth-socket use chlorid of ethyl spray or ice. If this treatment fails, plug with gauze infiltrated with tannin or soaked in antipyrin solution of a strength of 10 per cent., or in Carnot's solution of gelatin. Close the jaws upon the plug, and hold them with Barton's bandage. If this expedient fails, soak the plug in Monsel's solution, or plug with a bit of cork or dry sponge, and if this is futile, use the cautery. Pressure on the carotid and ice over the jaw and neck are indicated. It may be necessary to tie the external carotid artery.

27. In intra-abdominal hemorrhage open the belly. In intra-abdominal hemorrhage it is necessary to operate during shock. If the blood accumulates so rapidly as to prevent the location of the bleeding point, compress the aorta or pack the abdominal cavity with large sponges. In seeking for the bleeding point remove the sponges one by one, or have the pressure momentarily relaxed from time to time. In parenchymatous hemorrhage from the liver, suture the torn edge or use the cautery. In some cases the liver is sutured to the abdominal wall and the wound is packed. Severe wounds of the spleen demand splenectomy. Wounds of the kidney may be sutured, but may require partial or complete nephrectomy. Wounded mesenteric vessels are ligated *en masse* with silk (Senn). If a portion of intestine is found to be deprived

[1] Niebergall, "Deut. Zeit. f. Chir.," vol. xxxvii, Nos. 3 and 4.

of blood it must be resected. Wounds of the stomach and intestines causing hemorrhage require stitching of their edges. When there are a great many points of bleeding, take a number of gauze sponges, tie a piece of tape firmly to each one, pack many places in the belly with the sponges, bring the tapes out of the wound, and remove the sponges from below upward one at a time, securing the bleeding points as they come into view.

28. In abdominal section for disease of the female pelvic organs bleeding is limited by the clamp or by pressure-forceps. Ligation *en masse* is often practised. A large mass can be transfixed and tied in sections. Bleeding edges are stitched. Areas of oozing are treated by temporary pressure and hot water or, if this fails, by the cautery. Packing can be used as a tamponade, which is a gauze pouch, pieces of gauze being packed into this pouch after its insertion into the belly (see Fig. 42).

29. A ruptured varicose vein requires a compress, a bandage from the periphery up, and elevation.

30. Most cases of capillary bleeding can be controlled by compression with gauze pads soaked in water at a temperature of 115° to 120° F. This contracts the vessels and seals them with coagulated albumin. Keetly in 1878 impressed the profession with the value of hot water as a styptic. Centuries ago surgeons used hot oil for the same purpose. Capillary bleeding can often be controlled by the application of gauze soaked in Carnot's solution of gelatin. A solution of suprarenal extract may control capillary oozing. If other means fail to control capillary hemorrhage, the cautery must be used. Understand that the term "capillary bleeding" does not so much mean bleeding from genuine capillaries as it does bleeding from arterioles and venules.

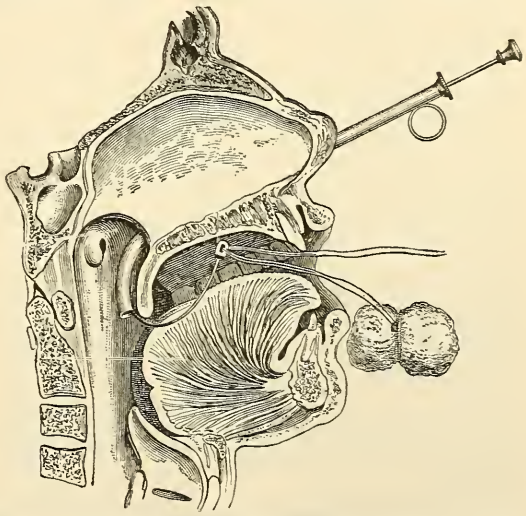


Fig. 228.—Plugging the nares for epistaxis by the aid of Bellocq's cannula (Guérin).

31. Pressure above a wound may arrest arterial hemorrhage, but aggravate venous bleeding. Pressure below a wound may arrest venous hemorrhage, but increase arterial bleeding.

32. A moderate epistaxis, or bleeding from the nose, may be arrested by an injection of peroxid of hydrogen, an injection of a solution of antipyrin, or an injection of Carnot's solution of gelatin. Favorite domestic expedients are keeping the arms raised above the head and applying ice to the back of the neck. In severe epistaxis examine the nose by means of a head-mirror and a speculum. If a little point of ulceration is found, touch it by a cautery. If the bleeding is a general ooze, if it is high up, or if the cautery does not arrest it, pack the nares. It may be necessary to pack one nostril or both. Pass a Bellocq cannula (Fig. 228) along the floor of one nostril into the pharynx, project the stem into the mouth, tie a plug of lint or gauze wet with Carnot's solution of gelatin to the stem, and withdraw it. Hold the double string which emerges from the nostril in the hand and

pack gauze wet with gelatin solution from before backward. Tie the strings together over the plug; if both nostrils are plugged, the strings from one nostril are fastened to the strings from the other. Do not use subsulphate of iron, as it forms a disgusting, clotty, adherent mass. If Bellocq's cannula is not obtainable, push a soft catheter into the pharynx, catch it by a finger, pull it forward, and tie the plug to it. Remove the plug in two or three days. Do not leave it longer. It blocks up decomposing fluids and may lead to blood-poisoning. Pick out the front plug first, hold the string of the second plug in the hand, push the plug back into the pharynx, catch it by forceps, and withdraw plug and string through the mouth.

33. In gunshot-wounds the primary hemorrhage is slight unless a large vessel is cut. The bleeding may be visible or may be internal (concealed), the blood running into a natural cavity or among the muscles. Capillary oozing is arrested by very hot water and compression. Venous bleeding is usually arrested by compression. If a large vessel is the source of bleeding, enlarge the wound and tie the vessel. If the artery cannot be found in the wound, tie the main trunk.

34. In prolonged bleeding from a leech-bite try compression over a plug saturated with alum or with tannin. If this fails, pass under the wound a harelip pin and encircle it with a piece of silk. If this fails, use the actual cautery or excise the bite and suture the incision.

35. In severe bleeding from the ear elevate the head, put an ice-bag over the mastoid, give opium and acetate of lead, and, if blood runs into the mouth, plug the Eustachian tube with a piece of catheter.

36. Umbilical hemorrhage in infants requires pressure over a plug containing tannin, alum, or gelatin solution. If compression fails, pass harelip pins under the navel and apply a twisted suture. If this fails, use the actual cautery.

37. Rectal bleeding requires elevation of the buttocks, insertion of pieces of ice, ice to the anus and perineum, astringent injections (alum), injections of gelatin, injections of adrenalin, and the internal use of opium and acetate of lead. If these means fail, plug the bowel over a catheter, or insert and inflate a Peterson bag or a colpeurynter, or tampon and use a T-bandage. If the bleeding persists or if a considerable vessel is bleeding, stretch the sphincter, catch the bowel and draw it down, seize the vessel, and tie it if possible; if not, leave the forceps in place. Failing in this, the actual cautery must be used.

38. Subcutaneous hemorrhage, if severe and persistent, demands that an incision be made and ligatures be applied.

39. Bleeding from a cut urethral meatus requires the insertion of styptic cotton and the application of pressure. Moderate bleeding from the deeper urethra can usually be arrested by a very warm bougie, by very warm injections, or by tying a condom over a catheter, and, after inserting it, inflating the condom by blowing through the catheter and plugging the orifice of the instrument, thus using pressure. Sitting with the perineum on a thickly folded towel is useful. Ice to the perineum does good. The patient can lie down, have a folded towel applied to the perineum, and a crutch-handle pushed upon the towel, the lower end of the crutch being jammed against the foot of the bed. If a solid bougie has been first introduced, firm pressure can be made by this method. If these means are futile, perform external urethrotomy and reach the bleeding point.

40. Hemorrhage from the prostate requires hot injections, the introduction of a large bougie first dipped in very warm water, and the retention of a catheter for two days. Perineal section may be required, or suprapubic cystotomy with packing which does not occlude the ureteral orifices.

41. Vesical hemorrhage usually ceases spontaneously, in which case the urine must be drawn off and the viscus be washed out frequently with a solution of boric acid, to prevent septic cystitis. If blood-clots prevent the flow of urine, break them up with a catheter or a lithotrite and inject vinegar and water, a 2 per cent. solution of carbolic acid, or a solution of bicarbonate of sodium. Perfect quiet is to be maintained, cold acid drinks are given, ice-bags are put to the perineum and hypogastric region, and opium with acetate of lead, or gallic acid is to be given by the mouth. If the hemorrhage is severe or persistent, perform suprapubic cystotomy, wash out the bladder, and, if necessary, plug the bladder with gauze, leaving the ureters uncovered.

42. In hemorrhage after lateral lithotomy, ligate if possible. If the vessel can be caught but cannot be ligated, leave the forceps in place. If it is not possible to catch the vessel with forceps, use a tenaculum. If the tenaculum fails, pass a threaded curved needle through the tissues around the vessel and tie the ligature (suture ligature). Plugs of ice and injections of hot water may be tried. These means failing, pressure is indicated. Take a cannula, fasten to it a chemise (Fig. 229), empty clots from the bladder, insert the instrument into the viscus, and pack gauze between the sides of the cannula and the chemise. The chemise is bulged out and pressure is made. Tie the cannula by means of tapes to a T-bandage. Pressure is thus combined with vesical drainage. Buckstone Brown makes pressure by inflating a rubber bag with air. The hot iron may occasionally be demanded.

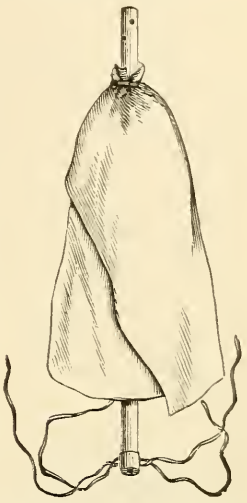


Fig. 229.—Cannula à chemise.

43. Renal bleeding requires ice to the loin, tannic acid and opium, gallic acid or sulphuric acid internally, and perfect quiet. The use of a cystoscope will show from which ureter blood is emerging. If the bleeding threatens life and the diseased organ is identified, make a lumbar incision and suture or perform nephrectomy.

44. Vaginal hemorrhage requires the ligature or the tampon.

45. Severe uterine hemorrhage (unconnected with pregnancy) requires the tampon. Persistent hemorrhage due to morbid growths may require removal of the tubes and appendages, ligation of the uterine and ovarian arteries, or hysterectomy.

46. Hematemesis, or bleeding from the stomach, is treated by the swallowing of ice, giving tannic acid (dose, 20 or 30 gr.), or Monsel's solution (3 drops). Gelatin by the mouth is recommended. Never give tannic acid and Monsel's solution at the same time, as they mix and form ink. Opium is usually ordered. Acetate of lead and opium and gallic acid are favorite remedies, and ergot is used by many. Give no food by the stomach. If life is threatened by bleeding from an ulcer, open the belly and excise the ulcer and suture the wound. If severe hemorrhage follows injury, perform an exploratory laparotomy. Always remember that furious and even fatal gastro-intestinal hemorrhage may be due to cirrhosis of the liver, and a slight injury may be the exciting cause of such a hemorrhage. In this condition, of course, operation is useless.

47. In bleeding from the small bowel give acetate of lead and opium, sulphuric acid, or Monsel's salt in pill form (3 gr.), allow no food for a time, and insist on liquid diet for a considerable period. If hemorrhage threatens life, do a celiotomy and find the cause. If ulcer exists, excise it and suture, or

suture a perforation without previously excising. If violent hemorrhage follows injury, explore to discover the vessel.

48. In bleeding from the large bowel, use styptic injections (10 gr. of alum or 5 gr. of bluestone to 1 oz. of water). If bleeding is low down, use small amounts of the solution; if high up, large amounts. Do not use absorbable poisons. In dangerous cases perform an exploratory operation to find the vessel. (For rectal bleeding, see page 454.)

49. Hemoptysis, or bleeding from the lung, is treated by morphin hypodermatically, by perfect rest, by dry cups or ice over the affected spot if it can be located, and by the administration of gallic acid, which drug aids coagulation.[1] Nitrite of amyl by inhalation has given good results.

50. In hemorrhage from wound of the lung do not open the chest unless life is threatened. If life is endangered, resect a rib, allow the lung to collapse, and see if this arrests bleeding. If bleeding still continues, remove several ribs, find the bleeding-point, ligate or employ forcipressure. A small cavity may be packed with gauze. If a large surface is bleeding, fill the pleural sac with gauze and pack more gauze against the oozing surface.[2]

**Reactionary or Recurrent Hemorrhage** (called also *Consecutive, Intermediate*, or *Intercurrent*).—This form of hemorrhage comes on during reaction from an accident or an operation, that is, during the first forty-eight hours, but in most cases within twelve hours. It is usually bleeding from a vessel or vessels which did not bleed during the shock which accompanied operation, and which vessels were overlooked and not tied. It may be due to faultily applied ligatures. It is favored by vascular excitement or hypertrophied heart. The bleeding is rarely sudden and severe, but is usually a gradual drop or trickle. The Esmarch apparatus is not unusually the cause. The constricting band paralyzes the smaller arteries, which do not bleed during shock and do not contract as shock departs; hence bleeding comes on with reaction. To lessen the danger of the Esmarch apparatus use a broad constricting band rather than a rubber tube. After an amputation, when the larger vessels have been tied, gauze pads wet with hot water (115° to 120° F.) should be placed between the flaps. This not only arrests capillary oozing, but stimulates vessels and shows points of bleeding which were not previously visible, and these points are ligated. During reaction after an amputation, if slight hemorrhage occurs, elevate the stump and compress the flaps. If the hemorrhage persists or at any time becomes severe, make pressure on the main artery of the limb, open the flaps, turn out the clots, find the bleeding point, ligate, asepticize, drain, close, and dress. In any severe reactionary hemorrhage open the wound at once and ligate.

**Secondary hemorrhage** may occur at any time in the period between forty-eight hours after the accident or operation and the complete cicatrization of the wound. Secondary hemorrhage may be due to atheroma, to slipping of a ligature, to inclusion of nerve, fascia, or muscle in the ligature, to sloughing, to erysipelas, to septicemia, to pyemia, to gangrene, and to overaction of the heart. The great majority of cases of secondary hemorrhage are due to infection, and the application of modern surgical principles has rendered secondary bleeding a rare calamity. If during an operation the vessels are found atheromatous, a thread should be passed, by means of a curved needle, around the vessel, including a cushion of tissue in the loop of the ligature (this prevents cutting through, see Fig. 211). Acupressure may

[1] The use of ergot is a general but questionable practice. Bartholow and others hold that this drug does harm; it contracts all the arterioles, and hence more blood flows from an area where there is damage. Purgatives do good in bleeding from the lung by taking blood to the abdomen and lowering blood-pressure.

[2] See author's case, "Annals of Surgery," Jan., 1898.

be used in such a case. If the surgeon decides to employ the ligature, he must not tie tightly, but must endeavor to approximate the coats rather than to cut them. Two ligatures can be applied or the stay-knot may be used. One great trouble with atheromatous arteries is that their coats cannot contract; another trouble is that the ligature is apt to cut entirely through them. If after an operation the pulse is found to be forcible, rapid, and jerking, give aconite, opium, and low diet. The bleeding may come on suddenly and furiously, but is usually preceded by a bloody stain in wound-fluids which had become free from blood.

**Treatment of Secondary Hemorrhage.**—Suppose a case of leg amputation in which, several days after the operation, a little oozing is detected: the treatment is to elevate the stump, apply two compresses over the flaps, and carry a firm bandage up the leg. If the bleeding is profuse or becomes so, make pressure on the main artery, open and tear the flaps apart with the fingers, find the bleeding vessel and tie it, turn out the clots, asepticize, drain, close, and dress. If the bleeding begins at a period when the stump is nearly healed, cut down on the main artery just above the stump and ligate. In secondary hemorrhage from a blood-vessel in nodular tissue apply a suture-ligature or tie higher up, or, if this fails, amputate. When secondary hemorrhage arises in a sloughing wound apply a tourniquet or an Esmarch bandage, tear the wound open to the bottom with a grooved director, look for the orifice of the vessel, dissect the artery up until a healthy point is reached, cut it across, and tie both ends. If this fails, apply a suture-ligature or use acupressure. In secondary hemorrhage from atheromatous vessels use the suture-ligature, double ligature with a stay-knot, or employ acupressure.

Secondary hemorrhage may occur after ligation in continuity, the blood usually coming from the distal side. If the dressings are slightly stained with blood, put on a graduated compress. If the bleeding continues or is severe, make pressure on the main artery of the limb, open the wound and ligate, wrap the part in cotton, elevate, and surround with hot bottles. If this religation is done on the femoral and fails, do not ligate higher up, as gangrene will certainly occur, but amputate at once above the point of hemorrhage. If dealing with the brachial artery, do not amputate, but ligate higher up and make compression in the wound. In a secondary hemorrhage from the innominate, tie the innominate again and also tie the vertebral.

**Hemorrhage After an Abdominal Operation.**—Hemorrhage may occur after an abdominal operation. It may come on gradually with reaction. If it does so, it causes thirst, restlessness, an increasing pulse-rate, pallor, increasing rate of respiration, and coldness of extremities. The temperature is normal or subnormal. If the hemorrhage is small in amount the condition may be recovered from. If it continues it produces the grave symptoms set forth on page 436. A rapid secondary or reactionary hemorrhage produces those grave symptoms almost suddenly. If a severe hemorrhage is occurring in the abdomen nothing but an operation can save life (see page 452).

**Hemophilia, Hemorrhagic Disease, or Hemorrhagic Diathesis.**—The term "hemophilia" expresses the existence in an individual of a tendency to profuse or even uncontrollable hemorrhage spontaneously or as a result of some very trivial injury.

Hemorrhage may take place from mucous or serous membranes or from wounds of the cutaneous surface, into tissue, into organs, into a joint, under the scalp, or into the external genitals. In a hemophiliac, if a cut is made, the hemorrhage from the larger vessels is easily arrested, but capillary oozing continues.

The condition is far more common in males than in females, and if it exists in a female, which it rarely does, it is not usually provocative of dangerous hemorrhage. The disease is nearly always transmitted by heredity. It

is usually transmitted to a son by a mother who is free from the disease, but whose father had it, and the son bleeds dangerously from slight causes. Some reported cases were transmitted in the male line (Goodall, in "Scottish Med. and Surg. Jour.," Feb., 1905). There is, however, an acquired form of hemophilia. The existence of the tendency is rarely suspected until the first dentition, and possibly not till puberty; "70 per cent. of cases appear before the fifth year."[1] The discovery of the existence of such a condition may not be made until a tooth is pulled, and extraction is followed by persistent bleeding. It is alleged that the tendency may disappear in middle life. The victims of this hemorrhagic tendency are called "bleeders."

The cause of hemophilia is unknown. It has been assumed that there is a condition of the blood which prevents coagulation. The blood-changes are not characteristic. The blood is similar to that found in secondary anemia, the red corpuscles are diminished, but the hemoglobin is diminished more distinctly, hence there is a low color index. The white corpuscles are not increased as in scurvy and purpura, and often there is a positive leukopenia. Blood coagulation is slow and often imperfect. In some cases coagulation occurs in nine minutes, but in one of Wright's cases it required over an hour. It is important to remember as against failure of coagulating elements as the cause that Agnew had a case in which hemophilia was limited to the head and neck, and that there have been cases in which bleeding occurred into one joint or from one kidney. Some maintain that there is structural defect in the capillaries. In a case of hemophilia in the Jefferson Medical College Hospital, in which it was absolutely necessary to amputate a finger because of a crush, a careful study of the vessels of the finger by Dr. Coplin failed to show any disease of the blood-vessels. Wright believes with Morawitz that the serum of circulating blood contains fibrinogen and a ferment called thrombogen. The leukocytes and other cells of the body produce thrombokinase. Thrombokinase is only set free when leukocytes or body-cells break down. Thrombokinase in the presence of calcium converts thrombogen into thrombin, and thrombin converts fibrinogen into fibrin (Wood, in "Australian Med. Jour.," 1910, vol. xv). Sahli believes that hemophilia depends on defects in certain cells which entail a deficiency of thrombokinase, and hence impairment of coagulating power in the blood ("Deutsch. Archiv. für klinische Medizin," 1910, vol. xcix). A surgeon must be on the lookout for hemophilia, and should inquire for it before deciding to do an operation. If it exists, only an operation of imperative necessity should be undertaken. It is now well recognized that joint lesions may occur in hemophilia (*hemophilic arthritis*). The condition is most common in children. As a rule, more than one joint is involved, but only a few joints suffer. In Rugh's case only one knee-joint suffered ("Annals of Surg.," May, 1907). If more than one joint is attacked, the involvement may or may not be symmetrical.

The acute form resembles acute rheumatism and lasts about ten days. In the subacute form the temperature is lower, the symptoms less intense, and the duration shorter. In both forms joint function is restored (Frolich, "Centralb. für Chir.," 1905, vol. xxxii). The chronic form resembles tuberculous arthritis or osteomyelitis. In this form there is a tendency to loss of function, but there is no reaction to tuberculin.

A child who is a "bleeder" must be unceasingly watched and guarded. A tendency to profuse oozing exists in leukemia because of the condition of the blood, but this is not hemophilia. A tendency to oozing also exists during jaundice.

**Treatment.**—The oozing is difficult and often impossible to control, although most of these cases, in the long run, recover. In the acquired form

[1] R. C. Cabot, in "International Text-Book of Surgery."

the prognosis is better than in the congenital (Weil, "Centralb. für Chir.," 1907, vol. xxxiv). Internal administration of such drugs as ergot, gallic acid, and acetate of lead is useless. It is claimed that chlorid of calcium internally is of service. Lactate of calcium, 15 to 20 gr., is sometimes given three times a day by the mouth. It may also be given by the rectum. Milk may be given by rectum, in order to obtain the combination of salts of milk unchanged by gastric juice (Solt, Ibid.). The local use of astringents is of no avail. Prolonged elevation may in rare cases succeed. In the case in the Jefferson Medical College Hospital the bleeding was arrested, after numerous expedients failed, by compression and hot water. Nurses sat by the bed for several days, constantly compressed the wound with gauze pads soaked in hot water, and changed the pads as soon as they cooled. The local use of Carnot's solution of gelatin has apparently saved several cases from death. A valuable plan is to tampon the wound with gauze containing fresh animal or human blood or blood-serum.

Serum in doses of from 5 to 10 c.c. may be given hypodermatically or, if bleeding is very severe, intravenously. We may use human serum, horse serum, rabbit serum, diphtheria serum, or antistreptococcus serum (see page 448). After each injection of serum there is leukocytosis, and leukocytosis means increase of cells containing fibrin ferment or thrombokinase. (See Tremburg, in "Medizinische Klinik," Berlin, Nov. 7, 1910.) A rapid method of obtaining thrombokinase is to take the liver from a rabbit or other animal, chop it, grind it, soak in water, and filter through cloth. Gauze soaked in this fluid is used to tampon a wound (Koltmann, Ibid., June 2, 1910).

Witte applies locally an extract of lymphoid organs (spleen, lymph-glands, or thymus) and injects hypodermatically a 5 per cent. solution of sterile peptone in .5 salt solution. The dose is 3 c.c. every other day until three or four doses have been taken. Then the injections are intermitted for three or four weeks (Nolf and Herry, "Reme de Medécine," Feb., 1910, and "Gaz. des Hôpitaux," 1911). Thyroid extract should be tried, as in some cases it seems to have been of value. The dose is 5 gr. after each meal. Eugene Fuller's case of hemophilia ("Med. News," Feb. 28, 1903) was apparently cured by the administration of 5 gr. of thyroid extract three times a day. In a case of hemophilia in the Jefferson Hospital thyroid extract apparently arrested the bleeding. In Rugh's case, after excision of a knee-joint, bleeding was profuse and continued, but ceased in eight days. The patient was given 5 gr. of thyroid extract three times a day. The cases are particularly interesting in connection with W. J. Taylor's observation that thyroid extract increases the rapidity of blood coagulation in jaundice cases and lessens the tendency to postoperative oozing in such cases.

## Operations On the Vascular System

**Paracentesis auriculi,** or tapping the heart-cavity, has been suggested for the relief of an overdistended heart from pulmonary congestion. The right auricle can be tapped. Push the aspirator needle directly backward at the right edge of the sternum, in the third interspace. This operation is not recommended, as it is highly dangerous and is of questionable value.

**Paracentesis pericardii,** or tapping the pericardial sac, is done only when life is endangered by effusion. Introduce the needle 2 inches to the left of the left edge of the sternum, in the fifth interspace, and push it directly backward (thus avoiding the internal mammary artery). The operation of tapping is extremely dangerous. The heart is lifted up and pushed forward by an effusion and the needle is apt to enter it. The puncture of a ventricle may do no harm, although it is very apt to, but the puncture of an

auricle is almost certain to be followed by fatal hemorrhage. It is wiser and safer to expose the pericardium and incise it, as is done for pericardial suppuration.

**Operation for Pericardial Effusion or Suppuration.**—The operation of tapping should be abandoned in favor of a safer but more radical procedure. There is no spot where we can introduce the needle with perfect safety, and the heart or pleura may be wounded; further, as Brentano shows,[1] tapping will not completely empty the sac. In a purulent case tapping gives practically no chance of cure. No general anesthetic should be used. A portion of the fourth rib or the cartilage on the fourth rib on the left side should be excised, the pericardium exposed and punctured in order to determine the nature of the fluid present. If the fluid is serous, it can be drained away through a small incision and the pericardium may be sutured. If the fluid be purulent, the pericardium should be stitched to the chest wall and opened. Clots should be removed by hot salt solution irrigation and a drainage-tube should be introduced.

**Operation for Wound of the Heart.**—In many cases it is obviously impossible to administer an anesthetic, but when possible it should be given because the movements of the patient while under the knife make operation difficult and increase bleeding. Ether may be used or we may take Hill's advice and give chloroform. Hill would give an anesthetic unless the patient is unconscious and the corneal reflex is abolished. Personally, I would be disposed to use chloroform unless the patient's general condition forbade it (see page 405). The pericardium can be exposed freely and Rotter's incision gives excellent access, although it always opens the pleura. This exposure is described by Hill in the "Medical Record," November 29, 1902, and was employed in his successful case. Begin an incision over the third rib $\frac{5}{8}$ inch from the left edge of the sternum and carry it outward along the rib for 4 inches. Begin an incision over a corresponding point of the sixth rib and carry it out for a like distance. Join the outer extremities of these cuts. Cut through the ribs and pleura by bone forceps and scissors. Raise the flap upon its hinges of cartilages, and have an assistant grasp the lung to prevent collapse. The pericardium thus exposed is opened widely if necessary. Hill advises us to steady the heart by pressing the hand under it and lifting it. Parrozzani did this by inserting a finger in the wound. Other surgeons have used traction sutures of silk. For wound closure interrupted sutures are preferred to the continuous suture. Either silk or catgut can be used. They should be inserted by a round-edged needle. "As few as possible should be passed commensurate with safety against leakage, as they cause a degeneration of the muscular fiber." It does no harm apparently if they enter a heart chamber, but it is wiser not to have them do so. If the heart fails, use heart massage (L. L. Hill, Ibid.). The pericardial and pleural sacs are cleansed by salt solution. The question of drainage is still *sub judice*. The pleural sac is treated according to indications in each case.

George Tully Vaughan, in reporting his second case of heart suture and a table of 150 operations ("Jour. Am. Med. Assoc.," Feb. 6, 1909), mentions five methods for exposing the heart, and states that no single method has yet been agreed on as the best. The kind of operation is often determined by the external wound, and, begun as an exploration, the subsequent steps depend on the necessities which arise during its progress. The five methods mentioned by Vaughan are: (*a*) Through an intercostal space, with or without the division of one or two cartilages; (*b*) resection of one or more cartilages, with or without a portion of rib; (*c*) flap method across the sternum, dividing the sternum and cartilages (this avoids opening the pleura); (*d*) flap of cartilages and

[1] "Deut. Med. Woch.," Feb. 11, 1890.

ribs with an external hinge; (*e*) flap of cartilages and ribs with an internal hinge.

Vaughan's table shows that in 46 patients the pericardium was drained, with 25 recoveries and 21 deaths; in 44 the pericardium was not drained, with 25 recoveries and 19 deaths; in 42 both the pericardium and pleura were drained, with 21 recoveries and 21 deaths; in 19 both pericardium and pleura were closed without drainage, with 12 recoveries and 7 deaths; in 72 the pleura was drained, with 30 recoveries and 42 deaths; in 21 the pleura was not drained, with 13 recoveries and 8 deaths. These figures would indicate that drainage should not be the rule. It should be used though if bleeding continues or if we greatly fear infection. Of course, drainage causes irritation, prevents the lung expanding, and makes secondary infection more probable (Vaughan, in "Jour. Am. Med. Assoc.," Feb. 6, 1909).

In Vaughan's table of 150 cases we find that 98 died and 52 recovered, a mortality of 65 per cent.; 32 patients died in less than twenty hours after the operation for the injury and 15 died on the table or just after the operation. In all but 1 of these cases death was due to hemorrhage. In 1 it was due to pneumothorax on opening the pleura. The remaining 66 deaths occurred in from twenty-four hours to five months after the operation: 6 died of pleurisy, 5 of pericarditis, 21 of combined pleurisy and pericarditis, 3 of pneumonia, 3 of peritonitis, 2 of pericarditis and nephritis, 1 of pleurisy and cerebral abscess, 1 of pleurisy and wound of the tricuspid valve, 1 of pleurisy and double pneumonia, 1 of gangrene of the lung, 1 of two wounds, one of which was not sutured, 3 of bleeding into the pleura, 2 of bleeding into the pericardium, 1 of clot in the tricuspid opening, and 15 of unassigned causes.

**Cardiolysis.**—As a result of pericarditis the heart may adhere to the pericardium and the pericardium to the chest wall. This condition is dangerous, and, if unrelieved, will eventually prove fatal.

Delorme has suggested that the pericardial sac be opened and the adhesions be broken down with the finger, a very dangerous procedure, which is almost certain to inflict serious injury upon the heart.

Brauer's method, which he suggested in 1902, consists in removing the several ribs and portion of the sternum to which the pericardium adheres. The periosteum is to be removed with the bone to prevent the formation of new bone. This is the preferable operation. The safest plan is to remove the anterior, but not the posterior, periosteum. The danger of fresh ossification is slight and we avoid injuring the left pleura (Poynton and Trotter, quoted by Simon, in "Brit. Med. Jour.," Dec. 14, 1912).

**Decompression for Heart Hypertrophy.**—In a case of cardiac hypertrophy in which the heart knocked violently against the ribs, Morrison performed thoracotomy. He removed several inches of the fifth rib and an equal length of the sixth rib. The patient was much improved, the capacity of the chest was increased, and the painful attacks were practically cured ("Lancet," July 4, 1908).

**Operation for Varix of Leg.**—Many cases do not require operation. In some, operation is positively harmful. In some selected cases operation is very useful to remove certain complications (ulcer, eczema, etc.), and to relieve the patient from annoyance, but the operation rarely absolutely cures the condition. As Blake points out, a cure cannot be claimed until at least one year has passed after operation without reappearance of the varix ("Boston Med. and Surg. Jour.," Sept. 25, 1902). The indications and contra-indications are discussed on page 412. Never operate if phlebitis exists except to treat thrombosis. After any operation for varicose veins of the leg follow Bennett's advice and keep the patient in bed for three weeks and do not let him resume active work for three weeks more ("Lancet," Nov. 22, 1902).

**Trendelenburg's Operation.**—I have employed this with much satisfaction in cases of varix of the leg following involvement of the saphenous in the thigh. Trendelenburg believes that in varix the valves in the saphenous become incompetent because of high central pressure. The veins of the leg distend, as they are unable to support such a long column of blood, and finally the blood begins to flow in the wrong direction in the saphenous, a "vicious circle" being established. We determine whether a case is a suitable one for Trendelenburg's operation as follows: While the patient is lying down raise the extremity as though we intended to empty it of blood previous to amputation. After three minutes compress the saphenous vein about the lower third of the thigh by means of a moist gauze bandage, which must not be so tight as to shut off the deeper vessels. Lower the leg and have the patient stand up. If blood flows into the saphenous from above and distends the portion of the vein above the compress, the valves are incompetent and Trendelenburg's operation may be performed. The operation is performed as follows: Make an incision about 4 inches in length over the internal saphenous vein at the junction of the lower and middle thirds of the thigh. Expose the vein, ligate each visible branch, ligate the saphenous at the lower end of the wound and also at the upper end, and remove the portion of vein included between the ligatures. By this operation the central pressure is intercepted and the dilated veins in consequence shrink. Some surgeons have advised the removal of the entire length of the long saphenous vein. If Trendelenburg's operation fails and a relapse occurs, extirpate the varicose veins of the leg.

*Madelung* cuts down over the varices and ligates at various points. *Schede* makes a circular cut (a circumcision) completely around the leg at the junction of the upper and middle thirds, the incision reaching to the deep fascia. All bleeding points are ligated and the edges of the incision are stitched together. This operation is so often followed by persistent hard edema that it is now seldom performed. A recent operation is to place the internal saphenous beneath the deep fascia. *Delbet* implants the saphenous into the femoral 10 cm. below its normal point of junction. The valvular arrangement of the femoral restores normal tension in the saphenous. *Fergusson* ties the saphenous vein near the femoral and removes a section from it. This makes the varices clearly evident. A semilunar incision is made to surround the varices, which incision reaches to the deep fascia. The flap is raised and dissected up, the vessels are tied, and the flap is sutured in place. *Phelps* advises multiple ligation. Sir Wm. H. Bennett thinks that in ordinary cases the best operation consists in removing a portion of the long saphenous in the thigh and also in removing 3 inches of the vein from below the knee. If there are cystic dilatations above the knee he removes the saphenous from the thigh. Some local varices he dissects out ("Lancet," Nov. 22, 1902). As a matter of fact, excision of a short piece of vein seems to do as much good as excision of a long piece. Excision of a piece at several points is valuable. The object of excision is to reduce pressure on the vein walls by breaking up the column of blood (Barker, in "Practitioner," Oct., 1910). Any of the suggested operations may be followed by relapse.

**Phlebotomy, or Venesection.**—**Operation.**—The patient sits on a chair "with the arm abducted, extended, and inclined outward" (Barker). The parts are ascepticized and a tape is tied around the arm just above the elbow. The patient grasps a stick firmly and works his fingers in order to cause the veins to distend. The surgeon stands to the right of the arm, holds the elbow with his left hand, and puts his thumb upon the vein below the intended point of puncture. Either the median cephalic or the median basilic may be opened (Figs. 230, 231). The median basilic is the more distinct, and is the vein usually

selected. In opening it do not cut too deep, as nothing but the bicipital fascia separates it from the brachial artery. The median cephalic may be selected (we thus avoid endangering the brachial artery); under this vein lies the external cutaneous nerve (Fig. 231). Steady the vein with the thumb and open it by transfixion, making an oblique cut which divides two-thirds of it. Remove the thumb and allow bleeding to go on, instructing the patient to work his fingers. When faintness begins, remove the fillet, put an antiseptic pad over the puncture, apply a spiral reversed bandage of the hand and arm and a figure-of-8 bandage of the elbow, and place the arm in a sling for several days.

**Transfusion of Blood.**—This operation has been a recognized procedure since 1824, though it has been known since 1492, when transfusion was employed in the case of Pope Innocent VIII. Its chief use was in severe hemorrhage, especially postpartum, in which it served to replace the blood lost and supplied something for the heart to contract upon until new blood formed. Senn insisted that the operation had proved an absolute failure, that it did not prevent death from hemorrhage, and that the transferred blood-elements did not retain vitality. Von Bergmann maintained that after severe hemorrhage we do not need to inject nutritive elements, but do need to restore the greatly diminished intracardiac and intravascular pressure. At the present day a saline fluid is usually infused in preference to transfusing blood. In fact, the operation of transfusion had become all but extinct until Crile revived it. The old operation, as it used to be performed, exposed the patient to the danger of embolism and infection. It had come to be believed that transfusion had no single element of value beyond that secured by the use of salt solution, except in cases overcome by illuminating gas, in which a more prolonged good effect was known to be produced than by salt solution.

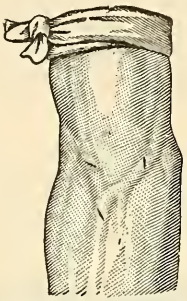


Fig. 230.—Incisions for venesection (Bernard and Huette).

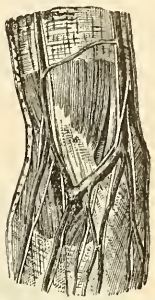


Fig. 231.—Superficial veins in front of elbow (Bernard and Huette).

**Arteriovenous Anastomosis for Transfusion of Blood (Crile's Operation).**—This is a method of the very greatest value in the treatment of the condition resulting from violent or prolonged hemorrhage. It is incomparably the best treatment for severe hemorrhage. It can be employed during the performance of an exhausting operation and its use will bring not a few cases to operation which, without its aid, would be esteemed inoperable. It should be used for severe typhoid hemorrhage and in jaundiced cases requiring operation, but showing very slow coagulation. It seems devoid of value in blood diseases. There are certain dangers in it unless there is time to examine the blood of both donor and recipient. It is known that admixture of certain bloods results in thrombosis. If the corpuscles of the donor are not agglutinated by the serum of the recipient, and vice versâ, the operation is safe.

In Crile's operation the vascular system of the donor is united to the vascular system of the recipient. He places intima in contact with intima. This is accomplished by means of a modification of Payre's magnesium tube or by Crile's tube, which is of German silver. The vein of the recipient is drawn through the tube, is everted, and is tied into the second groove on the tube.

The end of the tube with the everted vessel over it is passed into the vessel of the donor and fixed temporarily by a ligature. The left arm of each subject is usually employed and the radial artery of the donor is anastomosed to a superficial vein of the recipient. Each patient should be on a table the

head of which can be raised or lowered at will. The region over the radial artery of the donor is exposed under local anesthesia. Every small branch over the artery is carefully tied in order to prevent obstruction by blood. The artery is bared for a distance of about 3 cm., tied distally, lightly clamped with a screw clamp proximally, and divided. The vein of the recipient is bared, clamped, and divided, the tube (dipped in sterile olive oil) is inserted into the vein, the cuff of everted vessel is formed over the end, and the artery is pulled over the tube and the cuff of vein, and held by a ligature tied into the first groove (Bevan, in "General Surgery," by Lexer-Bevan).

The flow is at first slow, but after eight or ten minutes becomes more rapid, especially if warm salt solution is run into the wounds. The amount used depends on the strength of the donor and the needs of the recipient.

I have used Brewer's tube in this operation with much satisfaction. It makes the procedure vastly easier of execution. Fig. 232 shows Brewer's tubes.

I am indebted to Dr. George Emerson Brewer for describing in a note to me the technic of his operation. The description follows:

"After thorough sterilization, the tubes are prepared by dipping them in melted paraffin, shaking them out, and allowing them to cool, or immersing

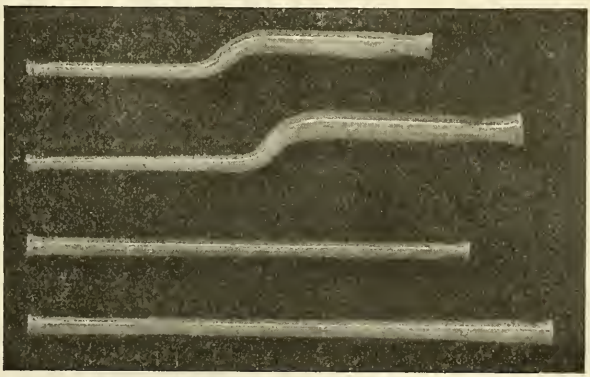


Fig. 232.—Brewer's tubes for direct transfusion.

them in a solution of paraffin in benzine. The radial artery of the donor is exposed in the usual manner, and also the median basilic or some other available vein of the donee. The proximal end of the artery is next drawn over one extremity of the glass tube and secured by a silk ligature. This is facilitated by expanding the lumen of the artery by means of three mosquito forceps or artery clamps. When all is ready to insert the free extremity of the tube into the vein of the donee, the arterial clamp is temporarily released and a few jets of blood allowed to pass through the tube, which is then quickly placed within the lumen of the vein and secured by another silk ligature. If the vein of the donee is large, the distal end of the glass tube may be inserted through a longitudinal slit in the vein, after the manner usually adopted when introducing the cannula for salt infusion. When sufficient blood has been transfused the tube is removed, the vessels ligated, and the cutaneous wounds closed. In certain rare instances where it is advisable to transfuse from an adult into an infant, the popliteal vein of the infant may be employed, as the subcutaneous veins are generally too small to admit of the introduction of the tube. In these cases a tube of diminishing caliber should be used, the larger end for the donor's artery, the smaller for the donee's vein."

**Fauntleroy's Vein-to-vein Anastomosis.**—This is a much simpler operation

than arteriovenous anastomosis. I have used it with great satisfaction. A glass tube is fixed in a vein of the donor and a vein of the recipient. I use the tubes devised by Dr. A. M. Fauntleroy of the United States Navy ("Med. Rec.," Sept. 3, 1910). The tube is $\frac{1}{8}$ inch in diameter and each end is flanged to prevent slipping. The veins in front of the elbow are used. The tube chosen may be full curved or S shaped (Fig. 233). The full curved tube is called the "hand-to-shoulder tube," because when it is used the hand of the donor is toward the shoulder of the recipient and the hand of the recipient is toward the shoulder of the donor. The S-shaped tube is called the "shoulder-to-shoulder tube." It is used when donor and recipient are placed side by side in the same direction, with the shoulders together (Fauntleroy, Ibid.).

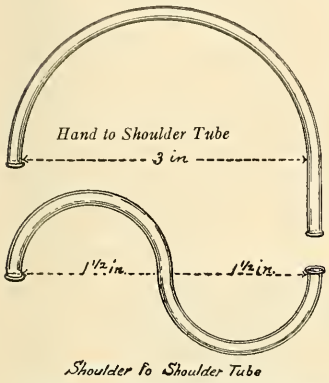



Fig. 233.—Tubes for transfusion, one-half natural size (A. M. Fauntleroy, in "Medical Record").

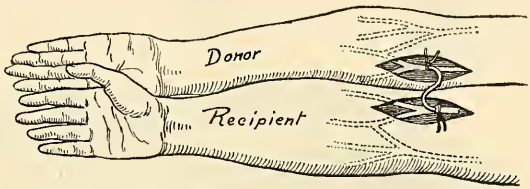



Fig. 234.—Vein-to-vein transfusion with the shoulder-to-shoulder tube (A. M. Fauntleroy, in "Medical Record").

The tubes are prepared with paraffin. A rubber band is placed around the arm of the recipient in order to make the veins prominent. The median basilic or median cephalic vein is exposed and freed for at least 1½ inches. A ligature is passed under the vein at the lower angle of the wound, another under the vein at the upper angle of the wound. The lower ligature is tied and the rubber band removed.

The tourniquet is now applied to the donor and the vein exposed as just described. The ligatures are placed, but only the upper one is tied. The vein below the ligature is gently grasped with rubber-protected forceps and is cut completely across. The intima is grasped with fine forceps and one end of the tube is inserted $\frac{1}{4}$ inch into the vein, and the lower ligature is tied in order to fix the tube in the donor's vein.

The elbows are now brought together, a nick is made in the vein of the recipient, the rubber-protected forceps are released from the vein of the donor, and, while blood flows, the tube is inserted in the vein of the recipient, and is held by the tying of the upper ligature. During the operation the rubber band is kept on the arm of the donor, sufficiently tight "to secure well-marked venous hyperemia in the forearm and a consequent increased venous pressure." The tourniquet must not cause stoppage of the radial pulse.

It is well to have a blood-pressure apparatus on the free arm of the donor and one on the free arm of the recipient. The pulse and general condition of donor and recipient are carefully watched.

Angulation of the veins would cause clotting and must not be permitted. We can tell that blood is passing by the improvement in color, in pulse, and in blood-pressure of the recipient and by the fulness of his vein near the tube. Fauntleroy in 1 case kept up the flow for thirty minutes. At the termination of the operation the tubes are removed, the veins tied, the tourniquet taken from the arm of the donor, and the wounds sutured and dressed.

**Intravenous infusion of saline fluid** is used after severe hemorrhage, in shock, in diabetic coma, in postoperative suppression of urine, and occa-

sionally in sepsis. After a hemorrhage its beneficial effects are often prompt and obvious. The saline fluid increases the arterial tension, gives the heart enough matter to contract upon, and so restores the activity of the circulation, and does not destroy the red corpuscles as plain water would do. We may use a simple apparatus consisting of a rubber tube, a funnel, and an aspirating needle. Some employ an Aveling syringe, and others Collin's apparatus (Fig. 236). The last-named instrument can be used without any danger of air entering with the fluid. Spencer's instrument (Fig. 237) is convenient and useful. Normal salt solution is the fluid usually employed, of a strength of 0.9 per cent. (1 heaping teaspoonful of common salt to 1 quart of warm boiled water). Some surgeons employ an artificial serum which contains 50 gr. of chlorid of sodium, 3 gr. of chlorid of potassium, 25 gr. of sulphate of sodium, 25 gr. of carbonate of sodium, and 2 gr. of phosphate of sodium in 1 quart of boiled water. Szumann's solution consists of 6 parts of common salt, 1 part of sodium carbonate, and 1000 parts of water. The following solution is used by Locke and Hare: Calcium chlorid,

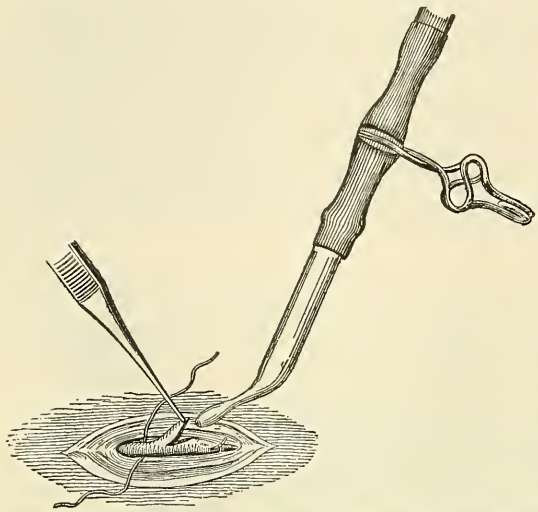


Fig. 235.—Intravenous saline infusion. Manner of incising vein and inserting glass tube (Senn).

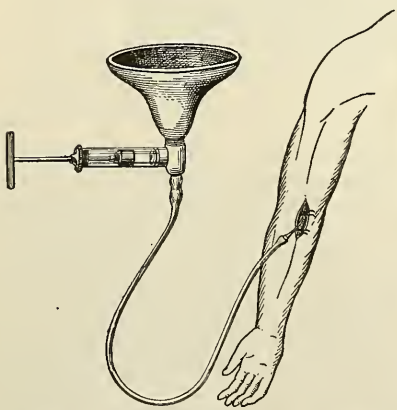


Fig. 236.—Intravenous injection of saline fluid.

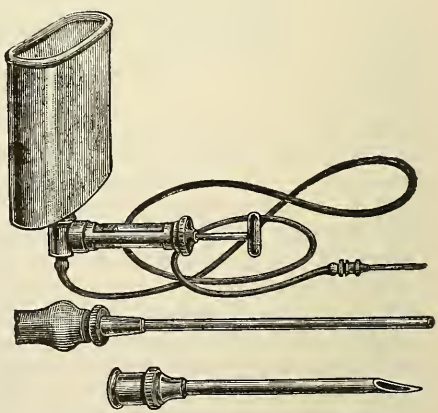


Fig. 237.—Spencer's apparatus for the infusion of saline fluid into a vein. The cannula can be plunged directly into the vessel without preliminary incision.

25 gm.; potassium chlorid, 1 gm.; sodium chlorid, 9 gm.; sterile water sufficient to make 1 liter. One bottle of the commercial fluid when diluted to 1 liter gives a solution of the above composition. The results from artificial serum containing many elements are no better than from normal salt

solution. Adrenalin may be added to the normal salt solution (1 dram of the 1:1000 solution of adrenalin to 1 pint of salt solution). The results of a single dose of adrenalin are very transitory. Whatever fluid is used, it should be at a temperature of 105° F. or over as it enters the vein. The stimulant effect of the heat is of great value. The fluid must not be allowed to cool; and a nurse gives constant attention to the temperature of the fluid in the reservoir. This degree of heat will not damage the corpuscles; in fact, Dawbarn has used saline fluid at a tempertature of 118° F. without doing damage to corpuscles and with great benefit to the patient. From ½ pint to 2 pints or even more are slowly injected, the condition of the patient determining the amount given. In one case of violent hemorrhage the author used over 2 quarts. In order to infuse this fluid tie a fillet well above the elbow, and expose by dissection the median basilic vein, or the basilic vein in the portion of its course where it is superficial to the deep fascia. Tie the vein. Incise it above the ligature, insert a fine cannula toward the heart, and hold the cannula firmly in the lumen by tightening a second ligature (Figs. 235, 236). Remove the fillet. Slowly and gradually introduce the fluid, carefully watching the

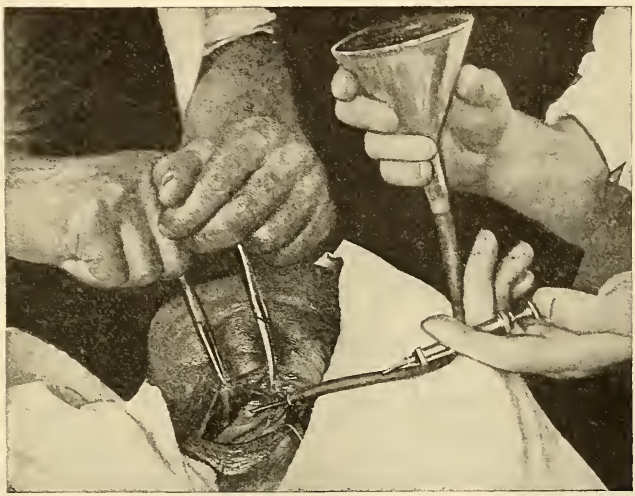


Fig. 238.—Injection of saline solution and adrenalin into an artery by the method of Crile.

pulse. Occupy at least ten minutes in introducing 1 pint, except in a very desperate case of hemorrhage, when the rapidity of the flow may be accelerated. When the tension of the pulse returns, withdraw the cannula, tie the second ligature tightly, sew up the wound, and dress it aseptically. In very severe operations an assistant should conduct the infusion while the surgeon is operating. It may be necessary to repeat the injection if the circulation fails again. The infusion of a very large amount of saline fluid may do harm. It may embarrass the heart and cause acute dilatation, may lead to edema of the lungs or brain, and cause marked anemia which lasts for days. The giving of salt solution intravenously should never be regarded as routine treatment, judgment is required in determining that it should be used, when it should be used, and how much is required, and there is a distinct element of danger in the procedure.

**Arterial Transfusion and Infusion of Saline Fluid in Arteries.**—Hueter preferred the arterial method of transfusion, in order to send the blood more gradually to the heart, and thus prevent sudden disturbance of the circulation. A little air in an artery will do no harm, and the danger of venous embolism is avoided. Saline fluid can be infused into an artery. The radial artery is

exposed and surrounded by three ligatures, and the thread toward the heart is at once tied. The distal ligature is slightly tightened to cut off anastomotic blood-supply. The artery is cut transversely half through; the syringe is inserted, pointed toward the periphery, and fastened by the third ligature; the second ligature is loosened and the material is injected. On finishing, the peripheral thread is tied tightly and that portion of the artery which held the cannula is excised. Dawbarn puts a hypodermatic needle into the radial artery and injects saline fluid.

Crile (Crile and Dolley, in "Jour. of Exper. Med.," Dec., 1906) has shown that when a patient is nearly dead or apparently dead the introduction of saline fluid by a vein may overwhelm the heart. He gives it in these cases by an artery and has succeeded in resuscitating those apparently dead. The tube of the apparatus is quickly inserted into the carotid artery and toward the heart. The reservoir is raised, and as the saline fluid begins to flow the tube is punctured with a hypodermatic needle and adrenalin is added to the saline stream. If the heart starts to beat, blood will appear in the tube and then the administration is discontinued. By this method we may re-establish blood-pressure in the coronary arteries (Fig. 238).

### Ligation of Arteries in Continuity

The **instruments** used in this operation are two scalpels (one small, one medium), two dissecting forceps, several hemostatic forceps, blunt hooks or broad metal retractors, an Allis dissector, an aneurysm needle, for superficial arteries the instrument of Saviard (Fig. 239), for deep vessels the needle

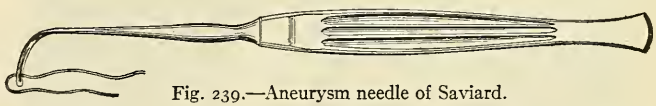
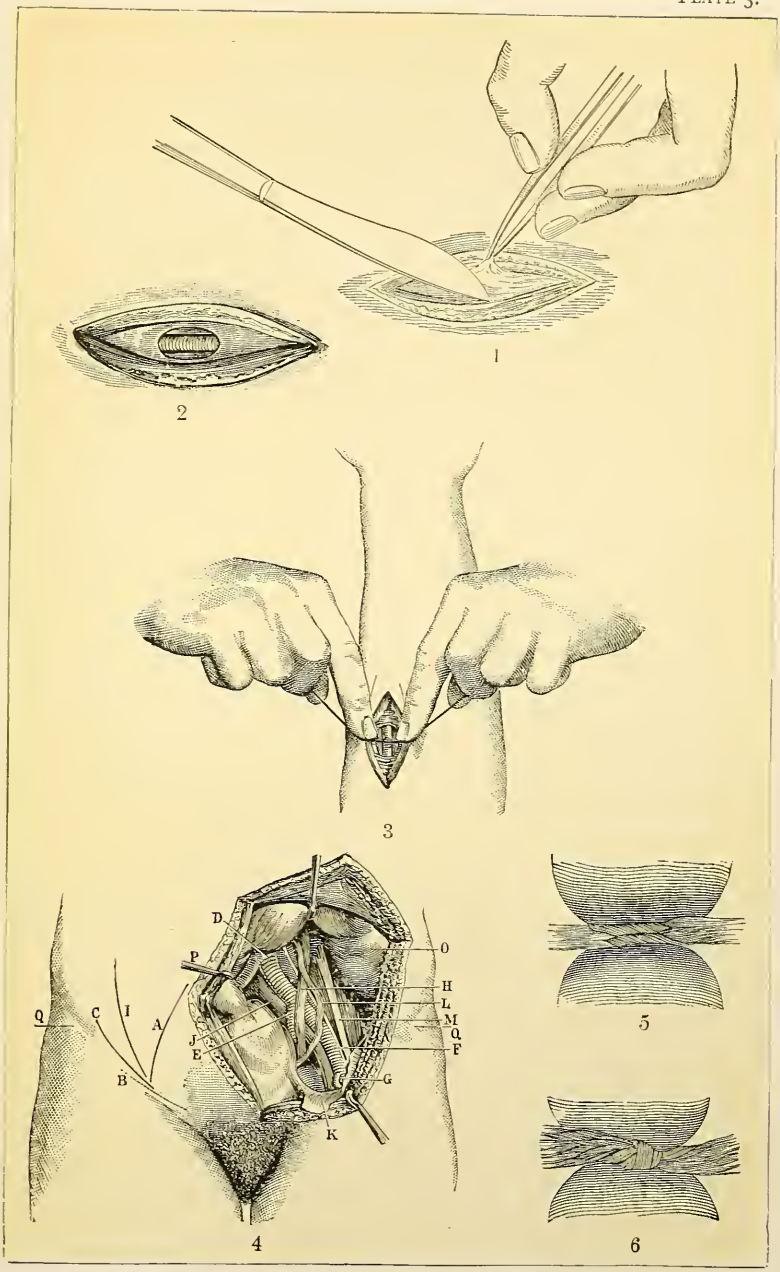


Fig. 239.—Aneurysm needle of Saviard.

of Dupuytren (Fig. 240), ligatures of catgut, of chromicized gut, or of silk, curved needles and a needle-holder, sutures of silkworm-gut, and the reflector or electric forehead-lamp for deep vessels.

The **position** in which the patient is placed varies according to the vessel to be ligated, though the body is supine except when ligation is to be performed on the gluteal, sciatic, or popliteal artery. The operator, as a rule, stands upon the affected side, cutting from above downward on the right side, and from below upward on the left side.

**Operation.**—Accurately determine the *line* of the artery, and make an incision at a slight angle to this line, avoiding subcutaneous veins, and holding the scalpel like a fiddle-bow or a dinner-knife while cutting the superficial parts, and like a pen while incising the deeper parts. On reaching the deep fascia make out the required muscular gap by the eye and finger, so moving the extremity as to bring individual muscles into action. Treves ("Operative Surgery") cautions us not to depend upon the yellow line of fat, which often cannot be seen in emaciated people or when an Esmarch bandage is employed; nor upon the white line due to attachment to the fascia of an intermuscular septum. In opening the deep portion of the wound relax the bounding muscles by altering the posture. Open a muscular interspace by a sharp knife, not by a dissector. Make the depths of the wound as long as the superficial incision. Do not tear structures apart with a grooved director; cut them. Arrest hemorrhage as it occurs. Try to find the situation of the artery with the finger. Pulsation is present, but it may be very feeble and hard to detect. The artery feels like a very thin rubber tube; it is compressible, though not so easily as a vein, and when compressed




1. Opening the Sheath for Ligation of an Artery (Guerin). 2. Sheath of Artery Open (Guerin). 3. Tightening the Knot in Ligation (Guerin). 4. Anatomy of the Iliac Arteries, and showing the lines of incision for their ligation: 1, Abernethy's incision (Guerin). 5, 6. Ballance and Edmunds' Stay-knots.

feels like a flat band which is thinner in the center than at the edges. A nerve feels like a hard, round cord. Veins are soft, larger than their related arteries, and so very compressible that they can scarcely be felt when pressed upon, and compression causes distal distention. If the wound can be seen into clearly, it will be noted, as Treves ("Operative Surgery") asserts, that "the nerves stand out as clear, rounded, white cords; that the veins are of a purple color and of somewhat uneven and wavy contour; that the artery is regular in outline and of a pale-pink or pinkish-yellow tint, the large vessels being of lighter color than the small." Each artery of the upper extremity and each artery below the knee is accompanied by two veins, known as "venæ comites." The arteries of the head and neck, except the lingual, have each a single attending vein; the lingual has venæ comites. Most of the smaller arteries of the trunk (pudic, internal mammary, etc.) have venæ comites. These companion veins may lie on each side of the artery or in front and back of it, and they communicate with one another by transverse branches crossing the artery. On reaching the sheath pick up this structure with toothed forceps so as to make a transverse fold, and thus avoid catching the artery or vein; lift the fold to see that it is free, and open the sheath by cutting toward the edge of the forceps with a scalpel held obliquely with its back toward the vessel, thus making a small longitudinal incision (Pl. 3, Figs. 1, 2). Hold the edge of the incised sheath with the forceps; pass a metal dissector under the vessel and from the forceps; this clears one-half of the vessel. Grasp the other edge of the sheath and pass the blunt dissector all the way around the vessel. Pass an aneurysm needle

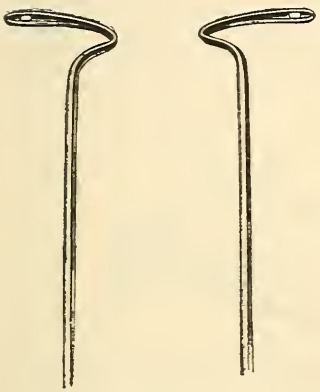


Fig. 240.—Dupuytren's aneurysm needles.

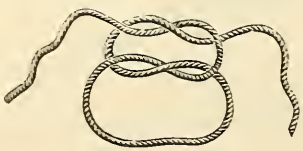


Fig. 241.—Reef-knot.

under the cleared vessel, away from the forceps holding the sheath and away from the vessel's most dangerous neighbor. Thread the needle and withdraw it. If venæ comites are in the way, try to separate them; but if this proves difficult, include them in the ligature. In small vessels always include them if they are in the way, as this saves trouble. If, in passing the needle, a large vein is severely wounded (such as the femoral), Jacobson advises the employment of digital pressure in the lower portion of the wound while the artery is being tied on a level above or below that of the vein injury, and after ligation the maintenance of pressure on the wound for a couple of days. A slight puncture in a vein merely requires a lateral ligature. A small wound can be closed with Lembert sutures of fine silk. After getting a ligature under an artery press for a moment upon the artery over the ligature, which is held taut; this pressure will arrest pulsation below if the ligature is around the main artery and there is not a double vessel. Tie the thread at right angles to the vessel with a reef-knot (Fig. 241), rupturing the internal and middle coats. As the ligature is tightened place the extended index-fingers along the ligature up to the artery (Pl. 3, Fig. 3), using the middle joints as the fulcrum of a lever by placing them against each other.

Ballance and Edmunds claim, as Scarpa and Sir Philip Crampton claimed long since, that it is not necessary to divide the internal and middle coats to

insure obliteration. If this claim be true, the danger of secondary hemorrhage can be greatly lessened. Holmes, however, thinks the older method the more certain of the two. Ballance and Edmunds use floss silk as a ligature material, because it is soft, broad, and flat, and they surround the artery with a double ligature. These surgeons thus describe the application of the stay-knot: "The best way of tying two ligatures is to make on each separately, and in the same way, the first hitch of a reef-knot, and to tighten each separately so that the loop lies in contact with the vessel without constricting it. Then taking the ends on one side together in one hand and the two ends on the other side in the other hand, constrict the vessel sufficiently to occlude it, and finally complete the reef-knot. The simplest way of completing the knot is to treat the two ends in each hand as a single thread and to tie as if completing a single reef-knot." This knot is shown in Pl. 3, Figs. 5, 6. The stay-knot applied by this method is of great value if a vessel be atheromatous.

The chief dangers after ligation are secondary hemorrhage and gangrene. Rigid asepsis usually prevents the first; rest, elevation, and heat antagonize the second.

**Radial Artery.**—The *line* of the radial artery is from the middle of the front of elbow-joint to the ulnar side of the styloid process of the radius. The *line* in the tabatière is from the apex of the styloid process to the posterior angle of the first interosseous space (Fig. 242).

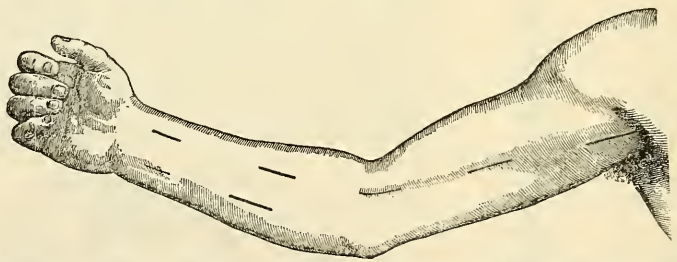


Fig. 242.—Lines of incision for ligation of the axillary (third portion), brachial, radial, and ulnar arteries (MacCormac).

**Anatomy** (Pl. 4, Fig. 5).—The radial artery, though smaller than the ulnar, is the direct continuation of the brachial. It arises from the bifurcation of the brachial ½ inch below the bend of the elbow, runs down the radial side of the forearm to the front of the styloid process of the radius, passes beneath the extensor muscles of the first metacarpal bone and of the first phalanx of the thumb, and over the carpus to the first interosseous space. It is crossed by the tendon of the extensor secundi internodii pollicis, enters into the palm between the heads of the first dorsal interosseous muscle, and forms the deep palmar arch. The artery in the upper two-thirds of its course is somewhat overlaid by the supinator longus muscle; in the lower one-third of the forearm it is superficial. In the upper third of the forearm it lies between the supinator longus on the outside and the pronator radii teres on the inside; in the lower two-thirds of the forearm it lies between the supinator longus on the outside and the flexor carpi radialis on the inside. Two venæ comites attend the vessel. The radial nerve is to the outer, or radial, side of the artery, well removed from the artery in the upper third, nearer to the artery in the middle third, far external to the artery in the lower third, the nerve at this point passing beneath the supinator longus muscle. The radial artery from above downward rests upon the biceps tendon, the supinator brevis, the flexor sublimis, the pronator radii teres, the flexor longus pollicis, the pronator quadratus muscles, and the radius. The best guide to the

radial artery in the forearm is the outer edge of the flexor carpi radialis muscle or the inner edge of the supinator longus muscle.

The tabatière anatomique of Cloquet, or the anatomical snuff-box, is a triangle whose base is the lower edge of the posterior annular ligament, the ulnar side being formed by the extensor secundi internodii pollicis tendon, the radial side by the extensor ossis metacarpi and the extensor primi internodii pollicis tendons; the floor consists of the trapezium, scaphoid, their dorsal ligaments, and the base of the first metacarpal bone.

**Operations.**—*Ligation in the tabatière* is a dissecting-room operation of but little practical use. The patient is placed in a recumbent position, the arm is abducted, and the forearm is placed midway between pronation and supination (Barker). The surgeon stands upon the side operated upon. An incision 2 inches in length is made along the radial border of the extensor secundi internodii pollicis muscle. The skin and superficial fascia are cut and some venous branches are divided. The deep fascia is incised, and the vessel is easily found and tied before it passes between the heads of the first dorsal interosseous muscle (Barker).

*Ligation of the Lower Third.*—In this operation (Pl. 4, Fig. 6, and Fig. 242) the patient is placed supine, the arm is abducted, the forearm is supinated, is rested upon a table, and is held by an assistant. The surgeon stands on the side operated upon, and cuts from above downward on the right forearm and from below upward on the left forearm. The line of the vessel should be determined, and may be indicated with iodin. An incision 1½ inches in length is made at a slight angle to this line and midway between the supinator longus and the flexor carpi radialis muscles, which incision must not extend below the level of the tuberosity of the scaphoid bone. In the superficial fascia watch for the superficial radial vein, and if it comes into view push it aside. Incise the superficial fascia and locate each guide-tendon. Open the deep fascia in the length of the first cut; try to separate the veins, but if they strongly adhere include them in the ligature. There is no special fascial sheath. The radial nerve will not be seen, but a division of the anterior cutaneous nerve is frequently found in relation with the vessel. The needle can be passed in either direction. A high origin of the superficialis volæ artery is confusing.

*Ligation of the Middle Third.*—In this operation the position of the patient should be the same as in the preceding. A 2-inch incision is made. Veins of the subcutaneous tissues are avoided. Lying upon the deep fascia is the anterior division of the musculocutaneous nerve. Open the fascia; find the inner edge of the supinator longus muscle and draw it outward, flexing the elbow partly if necessary. Be sure not to cut external to this muscle. Find the vessel where it is bound down by connective tissue to the pronator radii teres muscle, separate the veins, and pass the ligature from without inward. The nerve is external.

*Ligation of the Upper Third* (Pl. 4, Fig. 6, and Fig. 242).—For this operation the incision is as described above, only higher up. The artery is between the supinator longus and the pronator radii teres, which muscles are at once differentiated by the different direction of their fibers. The artery is usually covered by the supinator longus muscle, which must be retracted externally. The nerve is not seen. The ligature may be passed in either direction.

**Ulnar Artery.**—No one *line* will overlie the entire ulnar artery. The line of the upper third runs from the middle of the front of the elbow-joint to the point of junction of the upper and middle thirds of the ulna. The line of the lower two-thirds runs from the tip of the internal condyle of the humerus to the radial side of the pisiform bone (Pl. 4, Figs. 5, 6; Fig. 242).

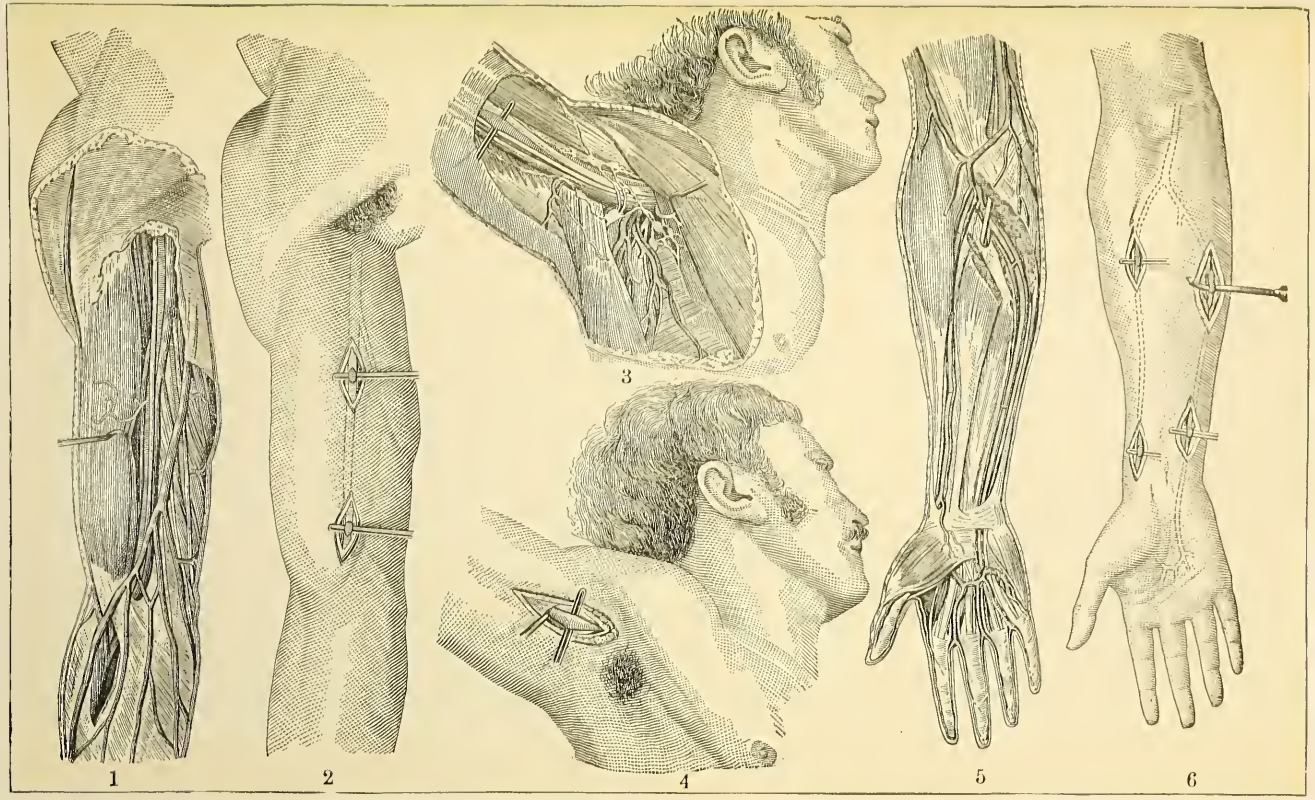
**Anatomy** (Pl. 4, Fig. 5).—The ulnar artery arises from the brachial bifurcation and runs obliquely inward under the median nerve and a group of muscles from the internal condyle; it turns down the arm, being covered in the middle third of its course by the flexor carpi ulnaris muscle. In the lower third it is superficial, between the tendons of the flexor carpi ulnaris on the inside and the flexor sublimis digitorum on the outside, the vessel being a little overlapped by the flexor carpi ulnaris. This vessel rests first upon the brachialis anticus muscle, next upon the flexor profundus, to which it is bound by a distinct process of fascia, and next upon the annular ligament, which structure it crosses to become the superficial palmar arch. Two venæ comites attend the vessel. In the upper third the ulnar nerve is well internal, but in the lower two-thirds the nerve lies near the artery and to its ulnar side. The guide is the outer edge of the flexor carpi ulnaris.

**Operations** (Pl. 4, Fig. 6, and Fig. 242).—*Ligation of the Lower Third.*—The position in this operation is the same as for ligation of the radial artery. Make a 2-inch incision to the radial side of the tendon of the flexor carpi ulnaris, which incision should not be taken lower than a point 1 inch above the pisiform bone. Avoid the superficial ulnar vein in the subcutaneous tissue. Open the deep fascia, find the tendon of the flexor carpi ulnaris, flex the wrist and draw the tendon inward, open a second layer of fascia, clear the vessel, separate the veins, and pass the ligature from within outward to avoid the nerve. On the artery is the palmar cutaneous branch of the ulnar nerve, and this branch must not be included in the ligature.

*Ligation of the Middle Third* (Pl. 4, Fig. 6).—In this operation the position is the same as in the preceding one, the incision being 3 inches long. Avoid the anterior ulnar vein and the branches of the internal cutaneous nerve in the superficial fascia. Open the deep fascia a little external to the superficial cut (Treves). Find the space between the flexor carpi ulnaris and the superficial flexor, feeling with the index-finger, and when the space is discovered flex the wrist, retract the flexor carpi ulnaris inward and the flexor sublimis digitorum outward, open the fascia, find the ulnar nerve, look external to it for the artery, clear the vessel, separate the venæ comites, and pass the needle from within outward. The ulnar artery should not be ligated in continuity in the upper third of its course.

**Brachial Artery.**—The *line* of the brachial artery is from the junction of the anterior and middle thirds of the outlet of the axilla, the arm being abducted and the forearm supinated, to the middle of the front of the elbow-joint (Fig. 242).

**Anatomy** (Pl. 4, Fig. 1).—The brachial artery is the prolongation of the axillary, and extends from the lower edge of the teres major muscle to ½ inch below the bend of the elbow, where it divides into the radial and ulnar arteries. It lies first to the inner side of the arm, but passes to the front of the elbow. It is crossed by no muscle, and is, in fact, superficial, barring its being somewhat overlaid in part of its course by the edge of the biceps muscle. The median nerve is external above, crosses over the vessel about the middle of the arm, and reaches the inner side of the artery. The coracobrachialis and biceps muscles are external, and both often overlap the vessel. The ulnar nerve is internal above, and the median nerve is internal below the middle. The basilic vein is to the inner side of the artery, being outside the deep fascia to near the middle of the arm, at which point it pierces it. The artery above is separated from the long head of the triceps by the musculospiral nerve and superior profunda artery and vein; it rests from above down on the inner head of the triceps, the coracobrachialis, and the brachialis anticus muscles. The artery is covered by skin, by superficial fascia, and by deep fascia. The internal cutaneous nerve lies in front of




1, Anatomy, 2, Ligation, of the Brachial Artery. 3, Anatomy of the Axilla. 4, Ligation of the Third Part of the Axillary Artery. 5, Anatomy, 6, Ligation, of the Radial and Ulnar Arteries. (From Bernard.)

the artery, upon the deep fascia, until it pierces the fascia along with the basilic vein. The artery has venæ comites, and in its upper half has also the basilic vein to its inner side. The guide to the brachial is the inner edge of the biceps muscle. Just in front of the elbow-joint the artery lies in a triangle, the base of which is formed by an imaginary transverse line above the condyles, and the apex by the junction of the pronator radii teres and the supinator longus muscles. The outer line is the supinator longus, the inner line is the pronator radii teres, and the floor is formed by the brachialis anticus and the supinator brevis muscles. From within outward the triangle contains the median nerve, brachial artery, tendon of the biceps, anastomosis of the superior profunda and radial recurrent arteries, and the musculospiral nerve.

**Operations.**—*Ligation at the Bend of the Elbow.*—In this operation (Pl. 4, Fig. 2, and Fig. 242) the patient is placed supine, the arm is moderately abducted and extended, and is allowed to lie upon its posterior aspect. The forearm is supinated. The surgeon stands upon the side operated upon, and cuts from above downward on the right side and from below upward on the left side. The tendon of the biceps and the median basilic vein must be accurately located. An incision is made parallel to the inner edge of the biceps tendon and 2 inches in length, the center of this cut being in the crease of the elbow. On exposing the median basilic vein, retract it downward and inward, open the bicipital fascia, clear the artery of fat, separate the venæ comites, and pass the ligature from within outward to avoid the median nerve. The above operation is not frequently performed.

*Ligation in the Middle of the Arm* (Fig. 242).—In this operation the patient is placed supine, the arm is abducted, and the forearm is supinated. An assistant holds the forearm, but the arm should not rest upon the table, because, if it be allowed to do so, the inner head of the triceps will be forced forward and may overlie the artery, and thus complicate the operation. Locate the inner edge of the biceps, which is the guide. Make an incision 3 inches in length in the line of the artery. Incise the skin and fascia, flex the elbow slightly, retract the biceps outward, feel for the artery, open the sheath, separate its venæ comites, and, having located the median nerve, pass the ligature from it. In the middle of the arm the nerve is in front of the vessel, above the middle it is external to it, and below the middle it is internal to it. High up the arm the inner edge of the coracobrachialis is the guide, rather than the biceps. Above the middle of the arm the basilic vein is beneath the deep fascia and passes along by the inner side of the artery; hence, high up the artery has three companion veins, the venæ comites and the basilic vein, and there is seen the ulnar nerve to the inside of the artery.

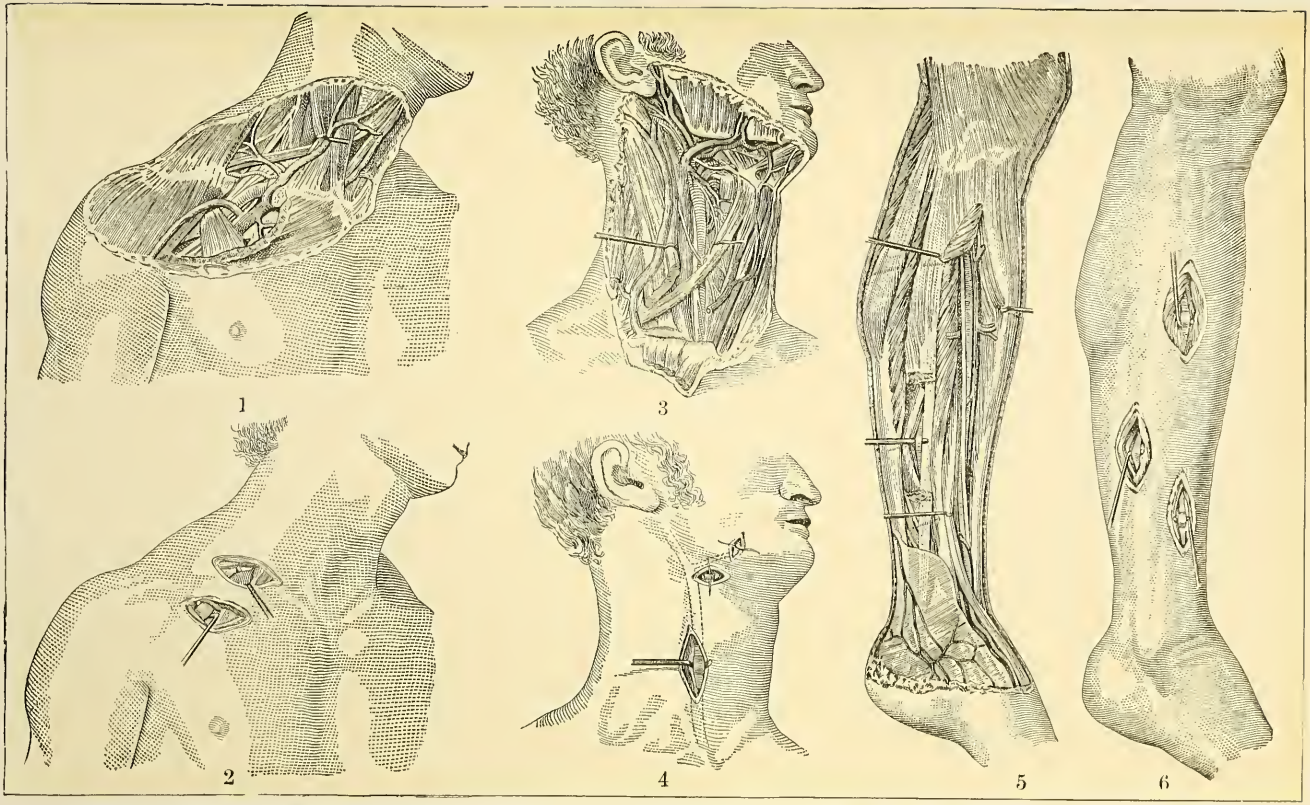
**Axillary Artery.**—To determine the *line* of the axillary artery place the arm at a right angle to the body, with the patient supine, and lay down a line from the middle of the clavicle to the humerus near the inner border of the coracobrachialis. The line of the third portion can be approximated by projecting the line of the brachial upward (Fig. 242).

**Anatomy** (Pl. 4, Fig. 3; Pl. 5, Fig. 1).—The axillary artery is the continuation of the subclavian, and runs from the lower margin of the first rib to the inferior border of the teres major muscle. It is divided into three portions by the pectoralis minor muscle. The first portion is above, the second portion is behind, and the third portion is below, the pectoralis minor. The position of the artery varies with the position of the limb. When the arm is parallel with the body the artery is far from the surface and forms a curve whose convexity is upward and outward. When the arm is at a right angle to the body the vessel is nearer the surface and straight. When the arm is raised above a right angle the artery comes near the surface and forms a curve with the convexity downward.

The first portion of the axillary artery is occasionally ligated. It lies upon the first intercostal muscle and the first serration of the great serratus muscle, and has behind it the posterior thoracic nerve; the brachial plexus is external and posterior to the vessel; on its inner side is the axillary vein; in front of it are the clavicle, the great pectoral muscle, the subclavius muscle, the costo-coracoid membrane, the cephalic and acromiothoracic veins, and the external anterior thoracic nerve. The branches of the first part of the axillary artery are the superior thoracic and the acromiothoracic. The second part of the artery is not ligated. The brachial plexus surrounds the second portion. The third part is covered in front, above, by the great pectoral, but is covered below by skin and fascia; behind, it has the tendon of the subscapularis, the latissimus dorsi, and the teres major muscles; the coracobrachialis is on the outer side; the axillary vein is on the inner side. It is important to remember that there may be three veins, one external and two internal. The axillary vein is formed by the venæ comites of the brachial artery joining, and this new vein effecting a junction with the basilic vein. The median nerve lies upon the axillary artery in the upper part of the third portion of the vessel's course, and passes to the outer side. The musculocutaneous nerve is external, but it is only seen high up; the ulnar nerve is internal; the lesser internal and the internal cutaneous nerves are internal; the musculospiral and the circumflex nerves are behind. The branches of the third portion of the axillary artery are the subscapular and the anterior and posterior circumflex.

**Operations.**—*Ligation of the Third Portion* (Pl. 4, Fig. 4, and Fig. 242).—The position of the patient should be supine, with the shoulders raised and the arm abducted to a right angle. The surgeon stands between the patient's arm and side, with his back toward the subject's feet. An incision is made 3 inches in length. It begins half-way up the axilla opposite to the head of the humerus, and is taken downward parallel to the lower edge of the great pectoral muscle and crosses the junction of the anterior and middle thirds of the outlet of the axilla. The integuments and fascia are incised. The vein or veins will be prominent to the inner side and may ovelie the vessel. To the inner side with the veins are the ulnar and internal cutaneous nerves. The median nerve is upon, and the external cutaneous is to the outer side of, the artery. Feel for the pulsations of the artery, find the median nerve, and draw it outward, draw the nerves and veins which lie to the inner side inward, clear the artery from the venæ comites, and pass the ligature from within outward. Apply the ligature well below the circumflex branches.

*Ligation of the First Part.*—This operation (Pl. 5, Fig. 2, and Fig. 244) was first performed in 1815 by Chamberlaine, of Jamaica. The patient is placed supine, the upper part of the body being raised, a sand-pillow being placed between the scapulæ to insure carrying back of the point of the shoulder, and the arm being brought down along the side. In operating on the left side the surgeon stands on the outer side of the left arm; in operating on the right side he stands to the right of the subject's head and leans over his shoulder. The incision, which is slightly curved downward, begins external to the sterno-clavicular joint and ends internal to the margin of the deltoid, thus avoiding the cephalic vein. The incision is ½ inch below the clavicle (Fig. 244). Incise the skin, platysma myoides muscle, and deep fascia. In the outer angle of the wound watch for the acromiothoracic artery and the cephalic vein. Incise the pectoralis major; draw the pectoralis minor downward; retract the lower margin of the wound, cut through the costocoracoid membrane close to the coracoid process and the upper border of the lesser pectoral muscle. Bring the arm to the side so as to relax the structures. Find the brachial plexus, feel for the artery internal to it, clear the vessel, draw the vein internally, and




1, Anatomy, 2, Ligation, of the Subclavian Artery and First Part of the Axillary Artery. 3, Anatomy of the Neck. 4, Ligation of the Carotid, Lingual, and Facial Arteries. 5, Anatomy, 6, Ligation, of the Anterior Tibial and Peroneal Arteries. (From Bernard.)

pass the needle from within outward. This avoids the dangerous neighbor, which is the axillary vein. This operation is difficult, dangerous, and unusual, and in its performance the axillary vein, which has a close attachment to the costocoracoid membrane, is apt to be torn.

**Subclavian Artery.**—The subclavian artery was first successfully tied by Post, of New York, who applied a ligature about the third portion of the vessel in 1817. In 1809 Sir Astley Cooper attempted to tie the first part of the left subclavian, but abandoned the operation because he feared he had wounded the thoracic duct. The first part of the subclavian was first tied by Colles in 1818 (Treves's "Manual of Surgery"), but the patient died. At the present day the first and second portions are rarely ligated. Professor Halsted in 1892 successfully tied the first portion of the left side for aneurysm. Schumpert tied it successfully for aneurysm. I assisted Dr. Nassau, of St. Joseph's Hospital, Philadelphia, in a ligation of the first part of the right subclavian. The man suffered from a ruptured traumatic aneurysm of the third portion of the vessel. The operation was followed by recovery. Chilton produced a cure of an aneurysm of the third portion of the subclavian of the right side by tying the first portion, and twenty-four hours later tying the first portion of the axillary. Curtis, in 1897, and Allingham, in 1899, ligated the first part successfully. Neff, of Spokane, successfully ligated the first part of the left subclavian ("Annals of Surgery," Oct., 1911). I tied the first part of the right subclavian and the first part of the common carotid for innominate aneurysm. The aneurysm was apparently cured. I also tied the third part of the right subclavian and the first part of the carotid for an innominate aneurysm. The patient apparently recovered, but many months later developed an aneurysm at the point of carotid ligation. There is no *line* for this vessel.

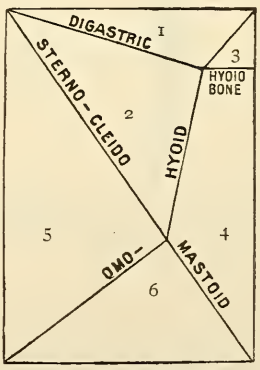



Fig. 243.—The triangles of the neck, right-sided view: 1, Submaxillary triangle; 2, "triangle of election," or superior carotid triangle; 3, submental triangle; 4, "triangle of necessity," or inferior carotid triangle; 5, occipital triangle; 6, subclavian triangle (after Keen).

**Anatomy** (Pl. 5, Fig. 1).—The subclavian artery of the right side arises from the innominate; that of the left side, from the arch of the aorta. The subclavian is divided into three parts: the first part runs from the origin of the vessel to the inner border of the scalenus anticus muscle; the second part lies behind the scalenus anticus muscle, and the third part runs from the outer edge of the muscle to the lower border of the first rib. The third portion is contained in the subclavian triangle (Fig. 243), and is superficial. It rises, as a rule, to ½ inch above the clavicle. The subclavian vein is below the artery, being separated from it by the scalenus anticus muscles. The brachial plexus is above and external to the artery. The vessel rests upon the first rib, and behind it is the scalenus medius muscle. The suprascapular and transversalis colli arteries and veins and branches of the cervical plexus of nerves lie in front of the artery, and the external jugular vein crosses it at its inner side. The third portion gives off no branches.

*Ligation of the Third Part* (Pl. 5, Fig. 2, and Fig. 244).—The patient is placed upon his back, the shoulders are raised, the head is extended and turned toward the opposite side, the arm is pulled down and held by pushing the forearm under the patient's back (Treves). This pulls down the clavicle, thus increasing the size of the subclavian triangle. The operator stands facing the shoulder, with his back toward the patient's feet. The skin over the sub-

clavian triangle, at a point ½ inch above the clavicle, is drawn down until it overlies the bone and is incised. This maneuver enables the surgeon to avoid the external jugular vein and to make an incision in the skin ½ inch above the collar-bone. The incision reaches from the anterior edge of the trapezius to the posterior border of the sternocleidomastoid (Pl. 5, Fig. 2, and Fig. 244), and is about 3 inches long. This incision divides the skin, superficial fascia, the platysma myoides, the vein running from the cephalic to the external jugular, and some superficial nerves. The deep fascia is opened. The external jugular vein is drawn into the inner angle of the wound, and is not divided unnecessarily; if forced to divide the vein, tie with two ligatures and cut between them. The surgeon seeks to find the outer edge of the anterior scalene muscle, and runs the finger down along it to the tubercle on the first rib. The posterior belly of the omohyoid muscle is drawn upward by an assistant. The surgeon, with a finger on the tubercle, recalls the facts that the vein is in front of the finger and the artery is behind it, and that the subclavian vein is on a lower plane than the artery. The artery is felt beating as it lies upon the rib. The artery is cleared and the lower cord of the brachial plexus is exposed. The vein must be guarded with the finger and the needle is passed from above downward, as the plexus, which is in more danger than the vein, is to be avoided. In this operation the transversalis colli and suprascapular arteries must not be cut, as they are necessary to the future anastomotic circulation. If the field of operation is too small, the trapezius or sternocleidomastoid, or both should be partly divided transversely.

Fig. 244.—Position of the lines of incision of temporal, facial, lingual, common carotid (above the omohyoid), subclavian, axillary (first portion), and internal mammary arteries (MacCormac).

**Results.**—Before the days of antisepsis ligation of the subclavian was a very fatal operation. Poland estimated the mortality at 70 per cent. In most cases death was due to secondary hemorrhage. Koenig collected 20 cases of ligation of the first part of the right subclavian, with 19 deaths. Lilienthal believes that the mortality after ligating the first portion on the right side is now only 16 per cent. (quoted by Neff in "Annals of Surg.," Oct., 1911). According to Joseph D. Bryant, there have been 134 deaths in 250 ligations at various points of the subclavian ("Operative Surgery"). I have twice tied this vessel with success. Gangrene seldom follows ligation of the subclavian. In 42 recoveries after ligation of various points there was 1 case of gangrene of the arm (Poiret's statistics, quoted by Neff in "Annals of Surg.," Oct., 1911). In Von Bergmann's 90 ligations for gunshot-wounds there was not a case of gangrene of the arm and only 3 of gangrene of the fingers.

The **vertebral artery** was first successfully ligated by Smythe, of New Orleans, in 1864. He had ligated the innominate for aneurysm of the subclavian and at the same time tied the common carotid. Secondary hemor-

rhage occurred, the blood coming from the brain. He arrested it by tying the vertebral.

**Anatomy.**—This vessel is the largest branch of the subclavian, and is the first branch coming from the first portion of the subclavian. The vertebral artery ascends and enters the foramen in the transverse process of the sixth cervical vertebra (in rare cases the fifth or the seventh), and ascends through foramina in the cervical vertebræ, passes behind the articular process of the atlas and over the posterior arch of this first vertebra, pierces the posterior occipito-atloid ligament, and enters the skull by way of the foramen magnum ("Gray's Anatomy"). It joins its fellow of the opposite side to form the basilar artery. At its point of origin the vertebral artery has in front of it the internal jugular vein and inferior thyroid artery. Near the spine it lies between the longus colli and scalenus anticus muscles, and on the left side has the thoracic duct to the left and in front.

*Ligation.*—The position of the patient is the same as for ligation of the carotid artery. Alexander thus describes the operation: "An incision 3 or 4 inches long is made in an upward and outward direction along the hollow which exists between the scalenus anticus and the sternomastoid muscles. The incision should begin just outside and on a level with the point where the external jugular vein dips over the edge of the sternomastoid muscle, or, if the vein is invisible, about ½ inch above the clavicle. The external jugular vein is drawn inward with the sternomastoid muscle. The connective tissue now appearing, the wound is opened by a blunt dissector until the scalenus anticus muscle, the phrenic nerve, and the transverse cervical artery are seen. It cannot be too well remembered that the pleura is at the inner side of the wound, while below lies the subclavian artery. It is now only necessary to separate the edges of the scalenus anticus and the longus colli muscles to see the vertebral artery lying in the space between them. The artery is generally completely covered by the vein, which is drawn aside, and the artery is then ligatured" (quoted in Bryant's "Operative Surgery"). When the vessel is cleared and tied, branches of the inferior cervical ganglion are damaged and possibly included in the ligature, and as a consequence the pupil contracts. Jacobson tells us to remember that the phrenic nerve lies on the scalene muscle, the pleura is internal, the internal jugular, inferior thyroid, and vertebral veins are over the vessel, and the thoracic duct on the left side crosses it from within outward.

**Results.**—In 36 ligations of the vertebral artery there were 3 deaths (Joseph D. Bryant).

**The Inferior Thyroid Artery.—Anatomy.**—The inferior thyroid artery is a branch of the thyroid axis. It ascends the neck, passes back of the carotid sheath and the sympathetic nerve, and reaches the thyroid gland. The recurrent laryngeal nerve lies behind the artery. The phrenic nerve is external to the artery and near to it in the first part of its course (up to the point of origin of the ascending cervical branch). The ascending cervical branch takes origin just before the artery begins to dip behind the carotid. In front of the beginning of the inferior thyroid artery of the left side the thoracic duct crosses. The artery is ligated in the second part of its course (between its distribution and the origin of the above-named branch).

*Ligation.*—The position of patient and the incision are the same as for the ligation of the common carotid artery in the triangle of necessity (see page 480). After exposing the sternocleidomastoid muscle retract it outward, and then draw outward the common carotid artery and also the internal jugular vein. The inferior thyroid artery will be found a little below the carotid tubercle. It is cleared and ligated. Treves advises ligation close to the level of the carotid, so as to avoid the recurrent laryngeal nerve.

**Innominate Artery.**—First successfully ligated by Smythe, of New Orleans, in 1864.

**Anatomy.**—The innominate artery arises from the beginning of the transverse portion of the arch of the aorta, passes to the back of the right sternoclavicular joint, and divides into the common carotid and subclavian vessels. It rests upon the trachea. It has upon its outer side the pleura, the right innominate vein, and the pneumogastric nerve. Upon its inner side are the remnant of the thymus gland and the beginning of the left carotid artery. In front of it are the inferior thyroid veins of the right side, the left innominate vein, the sternohyoid and sternothyroid muscles, the remnant of the thymus gland, and sometimes a branch from the right pneumogastric nerve.

*Ligation.*—Place the patient supine, with the shoulders a little raised, and the head thrown back. Carry an incision from the upper margin of the sternum for 3 inches along the anterior margin of the sternomastoid. Make another cut of the same length along the upper border of the clavicle to meet the first cut. Dissect up the flap of skin and fascia. Divide the sternal origin and a part of the clavicular portion of the sternocleidomastoid muscle, and cut the sternohyoid and sternothyroid muscles just above their sternal origins (Joseph Bell). Retract the inferior thyroid veins. Divide the dense leaflet of cervical fascia. Find the common carotid artery, and trace back along this vessel until the innominate comes into view. Retract the left innominate vein downward. The needle is passed from without inward to avoid the right innominate vein and right pneumogastric nerve. If the needle is kept close to the artery the pleura and trachea will not be injured.[1]

**Results.**—Burns, of Memphis, collected 45 cases and added 1 of his own, making 46 cases, with 9 recoveries ("Jour. Am. Med. Assoc.," 1908). To these should be added Percy Sargent's successful case ("Lancet," May 6, 1911), making 47 cases and 10 recoveries. Burrell's case in 1895 is counted as a success, although death occurred on the one hundred and fourth day. Smythe's case lived ten years. Sargent's case lived seventeen months and then died of pneumonia and pericarditis. He tied the common carotid as well as the innominate. The case of Coppinger, of Dublin, was alive and well two years after operation. Mitchell Banks's case lived over three months.

**Region of the Neck.—Anatomy.**—The side of the neck is that space between the median line in front and the anterior edge of the trapezius muscle behind, which space is limited below by the clavicle and above by the body of the jaw and an imaginary line running from the angle of the jaw to the mastoid process. The sternocleidomastoid muscle divides this space into an anterior and a posterior triangle, and each of the triangles is subdivided by other structures, the anterior into four spaces and the posterior into two (Fig. 243).

The *anterior triangle* is bounded in front by the median line of the neck, behind by the anterior margin of the sternocleidomastoid muscle, and above by the body of the lower jaw and an imaginary line drawn from the angle of the jaw to the mastoid process. This space is subdivided into four smaller triangles—namely, the inferior carotid, the superior carotid, the submaxillary, and the submental.

The *inferior carotid triangle* is called the "triangle of necessity," because the common carotid artery in this region is ligated, not from choice, but through force of necessity. It is bounded in front by the median line, above by the anterior belly of the omohyoid muscle and the hyoid bone, and below by the anterior edge of the sternomastoid muscle. The floor of this triangle is com-

[1] See the exceedingly clear and terse account in that excellent book, "A Manual of Surgical Operations," by Joseph Bell.

posed of the longus colli, the scalenus anticus, the rectus capitis anticus major, the sternohyoid, and sternothyroid muscles.

The *superior carotid triangle* is known as the "triangle of election," because, if the carotid artery must be tied, the surgeon, whenever possible, elects or chooses to tie it in this triangle. In this region the carotid is superficial, and there can be tied either the external, the internal, or the common carotid artery, as the surgeon elects. The triangle is bounded behind by the anterior edge of the sternocleidomastoid, above by the posterior belly of the digastric, and below by the anterior belly of the omohyoid muscles. Its floor is composed of the inferior and middle constrictors of the pharynx, the thyrohyoid and hyoglossus muscles.

The *submaxillary triangle* is bounded above by the body of the jaw and an imaginary line drawn from the angle of the jaw to the mastoid process, behind by the posterior belly of the digastric muscle and the stylohyoid muscle, and in front by the anterior belly of the digastric muscle. Its floor is composed of the mylohyoid and hyoglossus muscles.

The *submental triangle* is bounded on either side by the anterior belly of one digastric muscle; its base is the hyoid bone and its floor is the mylohyoid muscle.

The *posterior triangle* is bounded in front by the posterior border of the sternocleidomastoid muscle, behind by the anterior edge of the trapezius muscle, and below by the clavicle. The posterior belly of the omohyoid muscle subdivides it into two smaller spaces, the occipital and subclavian triangles.

The *occipital triangle* is bounded in front by the posterior edge of the sternocleidomastoid muscle, behind by the anterior border of the trapezius muscle, and below by the posterior belly of the omohyoid muscle.

The *subclavian triangle* is bounded above by the posterior belly of the omohyoid muscle, below by the clavicle, and in front by the posterior border of the sternocleidomastoid muscle. Its floor is formed by the first rib and the first serration of the serratus magnus muscle.

**Common Carotid Artery.**—The common carotid was tied to arrest bleeding by Abernethy in 1798, and was first ligated successfully for aneurysm by Sir Astley Cooper in 1806. The *line* of the common carotid artery is from the sternoclavicular articulation to midway between the angle of the jaw and the mastoid process, the head being turned toward the opposite side.

**Anatomy** (Pl. 5, Fig. 3).—The right common carotid arises from the innominate opposite the sternoclavicular joint; the left common carotid arises from the arch of the aorta. In the neck the two carotids possess identical relations. The common carotid runs upward and outward from behind the sternoclavicular articulation to a level with the upper border of the thyroid cartilage, at which point it divides into the external and internal carotid. The common carotid is contained in a sheath derived from the cervical fascia. This sheath also contains, in separate compartments, the internal jugular vein on the outer side of the artery and the pneumogastric nerve between the vein and artery, but more deeply placed. The anterior edge of the sternocleidomastoid muscle lies over the artery and is a guide. Low in the neck the common carotid is deep, being covered by skin, superficial fascia, platysma, deep fascia, and the sternocleidomastoid, sternohyoid, and the sternothyroid muscles. Above the omohyoid muscle the vessel is more superficial, being covered by the skin, superficial fascia, platymsa, deep fascia, and the anterior edge of the sternocleidomastoid muscle. Upon the sheath (occasionally within it), above the crossing of the omohyoid muscle, lies the descendens noni nerve—the descending branch of the ninth pair of Willis (the hypoglossal). This nerve is a valuable guide to the sheath in the triangle of election.

The *sternomastoid* branch of the superior thyroid artery crosses the carotid artery a little below its bifurcation, and the superior thyroid vein also crosses it in this region; the middle thyroid vein crosses the artery near its middle, and the anterior jugular vein crosses low down. The common carotid rests upon the longus colli and rectus capitis anticus major muscles, the sympathetic nerve lying between the last-named muscle and the vessel, outside the carotid sheath. The recurrent laryngeal nerve passes behind the carotid below the omohyoid muscle, and the inferior thyroid artery passes behind the carotid just above the omohyoid muscle. The common carotid is in relation internally with the trachea, thyroid gland, larynx, and pharynx. To the outer side are the pneumogastric nerve (which is on a posterior plane) and the internal jugular vein. On the left side, low down in the neck, the jugular vein often lies in front, or partly in front, of the artery.

*Ligation in the Triangle of Necessity.*—In this operation the patient is placed supine, with the shoulders raised, a sand-pillow under the neck, and the head turned to the opposite side, with the chin raised. The operator stands upon the side operated upon. The incision, 3 inches long, at a slight angle to the arterial line, runs from the level of the cricoid cartilage downward and inward toward the sternoclavicular joint, following the inner border of the sternocleidomastoid muscle. The surgeon opens the deep fascia, draws the sternocleidomastoid outward, retracts the sternohyoid and sternothyroid muscles inward, and feels for the carotid tubercle of Chassaignac. This tubercle is the costal process of the sixth cervical vertebra, and lies directly under the artery. The tubercle is found about the point at which the omohyoid crosses the carotid. When the tubercle is found we know the situation of the artery, and that the triangle of necessity is below, and the triangle of election above, the tubercle. The operator draws the omohyoid muscle upward, opens the sheath of the artery on its inner side, clears the vessel, and passes the needle from without inward to avoid the internal jugular vein, remembering that the pneumogastric nerve is in the same sheath as the artery and vein, posterior and external to the artery. In this operation the inferior thyroid veins are much in the way, the anterior jugular vein crosses low down, and on the left side, at the root of the neck, the internal jugular vein may be in front of the carotid artery. If the incision is not sufficiently wide, partially divide the sternocleidomastoid or the sternohyoid and thyroid muscles. In the triangle of necessity the descendens noni nerve does not serve as a guide to the sheath of the vessels. (See Pl. 5, Fig. 4.)

*Ligation in the Triangle of Election* (Fig. 244).—The *position* of the patient for this operation is the same as in the preceding one. An incision, 3 inches in length, is made along the anterior edge of the sternocleidomastoid muscle in the line of the artery, the middle of this incision being opposite the cricoid cartilage (Fig. 244). In cutting the superficial fascia the surgeon avoids the external jugular vein, the course of which should be outlined before making the incision. The line of the external jugular is from the angle of the jaw to the middle of the clavicle. The operator opens the deep fascia, retracts the sternocleidomastoid muscle outward, feels for the carotid tubercle, draws the omohyoid muscle downward, finds the descendens noni nerve upon the sheath, opens the sheath at its inner side, and passes the needle from without inward. This incision permits ligation of either the superior thyroid or the external, internal, or common carotid, and if it be extended up a little there can be tied through it the lingual and even the facial and occipital arteries. (See Pl. 5, Fig. 4.)

**Results.**—In from 20 to 25 per cent. of cases after ligation of the common carotid artery there is cerebral softening or some other intracranial complication. Crile states that of the cases that develop cerebral trouble, one-

half die. The direct operative mortality, according to Crile, is only 3 per cent. Some modern operators regard the mortality as much higher than this. I was obliged to tie the common carotid during an operation for tumor of the carotid gland; the patient developed hemiplegia.

**External Carotid Artery.**—Burke ligated the external carotid in 1827 (Treves, from Chelius). The *line* of the external carotid artery is the upper portion of the common carotid line.

**Anatomy** (Pl. 5, Fig. 3).—The external carotid artery, which is one of the terminal branches of the common carotid, arises on a level with the upper border of the thyroid cartilage and runs to the level of the neck of the condyle of the lower jaw. At its point of origin it is covered only by skin, platysma and fascia, and the edge of the sternomastoid, but as it ascends it passes beneath the digastric and stylohyoid muscles and into the parotid gland. The glossopharyngeal nerve, styloid process, and stylopharyngeus muscle lie between the external and internal carotid arteries. The hypoglossal nerve crosses the vessel just below the digastric muscle, and the facial and lingual veins cross it a little below the nerve. The first branch is the superior thyroid, which arises from the very beginning of the trunk. The lingual arises on a level with the greater cornu of the hyoid bone. The facial and occipital take origin above the lingual. Each of them can be ligated through the incision made for ligation of the external carotid.

**Operation.**—Place the patient in the same *position* as for ligation of the common carotid. The point of election is between the superior thyroid and the lingual arteries. Make an incision 3 inches in length at a slight angle to the arterial line, from near the angle of the jaw to opposite the middle of the thyroid cartilage. Cut through the skin, superficial fascia, platysma and deep fascia, and retract the sternocleidomastoid muscle outward. Watch for the digastric muscle, find the hypoglossal nerve, and feel for the greater cornu of the hyoid bone. Open the sheath a little below the hyoid cornu and pass the needle from without inward. Ligation of the external carotid has been neglected because ligation of the common carotid is easier.

**Results.**—Crile believes the operative mortality to be 2 per cent.

**Internal Carotid Artery.**—The internal carotid was tied by Keith, of Aberdeen, in 1851 (Ashhurst's "International Encyclopedia of Surgery"). The *line* of the internal carotid is parallel with and ½ inch external to the line of the external carotid.

**Anatomy** (Pl. 5, Fig. 3).—The internal carotid artery, the other terminal branch of the common carotid, arises on a level with the upper border of the thyroid cartilage and enters the carotid canal. The first inch of the artery is the only point where a ligature is ever applied, this point being covered only by skin, platysma, fascia, and the sternocleidomastoid muscle; higher up the artery is more deeply placed. It rests upon the vertebræ and the rectus capitis anticus major muscle. The internal jugular vein is in the same sheath and external to the artery; the pneumogastric is in the same sheath, between the artery and the vein, but posterior to both. The superior cervical ganglion of the sympathetic lies behind the origin of the internal carotid, and between the ganglion and the artery is the superior laryngeal nerve.

**Operation.**—In this operation the *position* of the patient is the same as for ligation of the external carotid. The incision is of the same length and direction as that for ligation of the external carotid, and is ½ inch external. The sternocleidomastoid muscle is drawn outward, the external carotid artery is found and drawn inward, the internal carotid is found and cleared, and the needle is passed from without inward. The internal carotid is known by its more external position and by the fact that it gives off no branches.

**Results.**—There is the same danger of cerebral complications after this operation as after ligation of the common carotid. The operative mortality is probably as great.

**Superior Thyroid Artery** (Pl. 5, Fig. 3).—This branches off from the external carotid below the level of the greater cornu of the hyoid bone, in the triangle of election. It is primarily superficial, runs first upward and inward, next downward and forward, passes underneath the omohyoid, sternohyoid, and sternothyroid muscles, and reaches the thyroid gland.

*Ligation.*—The position of the patient and of the surgeon is the same as for ligation of the carotid. The artery may be reached through the incision employed for ligation of the external carotid. Gross made an incision beginning at the edge of the hyoid bone, and running downward and outward to the sternomastoid muscle. The skin and superficial and deep fasciæ are divided, and the artery is found deeply placed in the triangle of election between the carotid sheath and the thyroid gland.

**Lingual Artery.**—Charles Bell ligated the first part of the lingual artery in 1814. The operation beneath the hyoglossus muscle was devised by Pirogoff in 1836 (Treves's "Manual of Operative Surgery").

**Anatomy** (Pl. 5, Fig. 3).—The lingual artery arises from the external carotid opposite the greater cornu of the hyoid bone, passes beneath the digastric and stylohyoid muscles, reaches the margin of the hyoglossus muscle, passes under that muscle, and emerges from beneath it to run along the under surface of the tongue. The place of election for ligation is where the artery is beneath the hyoglossus muscle. Its guide is the hypoglossal nerve, which lies upon the muscle, but at a slightly higher level than the artery.

**Operation.**—In this operation the patient is placed recumbent with the shoulders raised and the face turned away from the side to be operated upon. The surgeon stands upon the affected side. A curved incision is made from a little external to the symphysis of the lower jaw, downward and outward, to just above the greater cornu of the hyoid bone, and upward and outward to just in front of the facial artery at the lower edge of the lower jaw. The skin, the superficial fascia and platysma, and the deep fascia are incised. The submaxillary gland is cleared and retracted well upward. The fascia beneath the gland is divided by a transverse incision. The posterior edge of the mylohyoid muscle and the bellies of the digastric muscle are sought for and identified. One of the digastric tendons is retracted down and out (Treves). The hyoglossus muscle is cleared with a dissector; the hypoglossal nerve and ranine vein are found and drawn a little upward. The hyoglossus muscle is divided transversely a little above the hyoid bone and below the level of the hypoglossal nerve. The artery is found under the muscle and the needle is passed from above downward.

**Facial Artery.—Anatomy** (Pl. 5, Fig. 3).—It arises from the external carotid a little above the lingual, runs upward and forward beneath the body of the inferior maxillary bone, passes along a groove in the posterior and upper surface of the submaxillary gland, crosses the body of the lower jaw at the lower anterior edge of the masseter muscle, and passes forward and upward to the angle of the mouth and side of the nose.

*Ligation* (Pl. 5, Fig. 4).—The facial artery is rarely ligated in the cervical portion, but may be reached through the incision employed for ligation of the external carotid. The vessel may be tied before it crosses the submaxillary gland, the stylohyoid and digastric muscles being drawn aside. The vessel is reached in the facial portion of its course by a 1-inch cut at the anterior edge of the masseter muscle (Fig. 244). Branches of the facial nerve are pushed aside. The needle is passed from behind forward to avoid the vein (Jacobson).

**Temporal Artery.**—The line of the temporal artery passes "upward over the root of the zygoma, midway between the condyle of the jaw and the tragus" (Jacobson).

**Anatomy.**—The temporal artery arises from the external carotid behind the condyle of the jaw and in the parotid gland, passes over the zygoma, and divides into two terminal branches.

*Ligation.*—The patient is placed recumbent and the head is turned to the opposite side. An incision 1 inch in length is made (see Fig. 244), the superficial structures and dense fascia are divided, the vein is retracted backward, and the needle is passed from behind forward.

The **occipital artery** takes origin from the posterior surface of the external carotid, below the digastric muscle and opposite the point of origin of the facial artery. It ascends beneath the digastric and stylohyoid muscles and parotid gland; the hypoglossal nerve hooks around it from behind forward. It crosses the internal carotid artery, the internal jugular vein, the pneumogastric and spinal accessory nerves; passes between the mastoid process of the temporal bone and the atlas; grooves the temporal bone; penetrates the trapezius muscle, and ascends over the occiput.

*Ligation.*—This vessel can be ligated near its origin through the same incision as is employed to reach the external carotid. The hypoglossal nerve is avoided. To tie back of the mastoid process, place the patient in the same position as for ligation of the carotid. Carry an incision from the tip of the mastoid upward and backward, reaching a point midway between the mastoid and the occipital protuberance (Jacobson). Cut the skin, the fascia, the sternocleidomastoid, the splenius capitis, and possibly a portion of the trachelomastoid muscles. Bring the head toward the operator in order to relax the structures, retract the edges of the wound, and clear the artery where it lies between the mastoid process and the transverse process of the atlas (Jacobson). An electric forehead light is of great assistance in finding the vessel. Pass the needle away from the vein or veins (there are often several).

**Dorsalis Pedis Artery.**—The *line* of the dorsalis pedis artery is from the middle of the front of the ankle-joint to the middle of the base of the first interosseous space.

**Anatomy** (Pl. 6, Fig. 1).—The dorsalis pedis is a continuation of the anterior tibial artery, and it runs from the bend of the ankle to the proximal extremity of the first interosseous space, where it divides into the dorsalis hallucis and the communicating arteries. The artery rests, from above downward, upon the astragalus, scaphoid, and internal cuneiform bones, and at its point of bifurcation lies between the heads of the first dorsal interosseous muscle. It may lie in some persons a little external to this course. It is held upon the bones by a distinct layer derived from the deep fascia. This artery is covered by skin, by superficial and deep fascia, and by the annular ligament above, and is sometimes partly overlaid by the extensor proprius pollicis muscle, and is crossed, just before its bifurcation, by the innermost tendon of the extensor brevis muscle. The inner tendon of the extensor communis digitorum is to the outer side of the vessel; the tendon of the extensor proprius pollicis is to the inner side, and is a guide. The artery is ligated in the dorsal triangle of the foot—a space which is bounded above by the lower edge of the annular ligament, externally by the inner tendon of the extensor brevis, and internally by the tendon of the extensor proprius pollicis. The artery has venæ comites; the anterior tibial nerve lies, as a rule, to its inner side, but may be found upon the artery or to its outer side, and the inner division of the musculocutaneous nerve is external to the vessel in the superficial parts.

**Operation** (Pl. 6, Fig. 2).—In this operation the patient is placed supine with the leg and foot extended. Heath flexes the leg partly and rests the sole

of the foot directly upon the table. The surgeon stands below the extremity and cuts from above downward. Make an incision 2 inches in length along the arterial line, beginning opposite the lower edge of the annular ligament and running along by the tendon of the extensor proprius pollicis; cut through the skin and superficial and deep fascia; have the toes extended; retract the tendon of the extensor proprius pollicis inward, and the tendon of the extensor communis digitorum outward; clear the artery, find the nerve, try to separate the venæ comites, and pass the needle from the nerve.

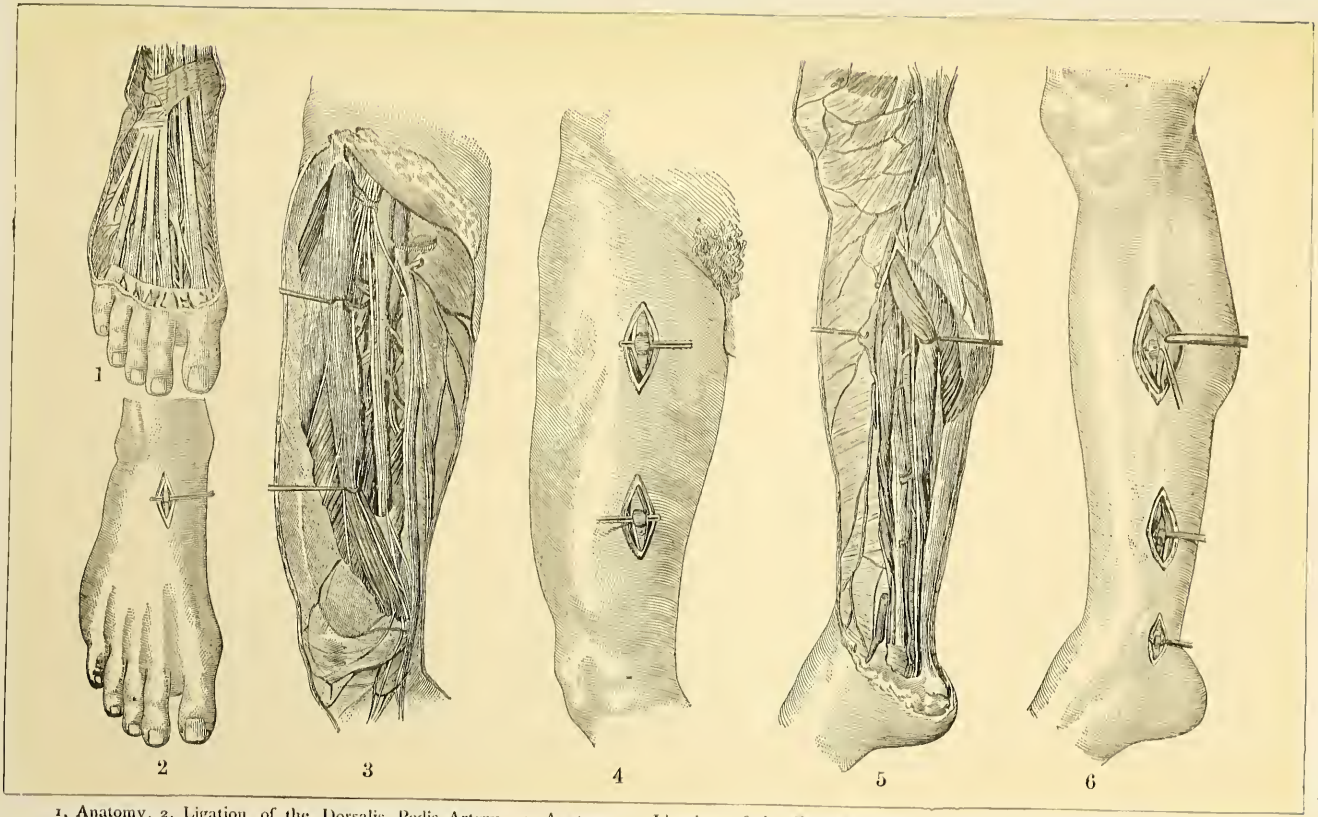
**Anterior Tibial Artery.**—To locate the *line* of the anterior tibial mark a point midway between the head of the fibula and the tuberosity of the tibia, drop 1 inch, and draw a line from the second point to the middle of the front of the ankle-joint.

**Anatomy.**—The anterior tibial artery is one of the terminal branches of the popliteal. It arises opposite the lower border of the popliteus muscle, passes forward between the two heads of the posterior tibial muscle, comes to the front of the leg through an opening in the interosseous membrane, and runs down to the middle of the front of the ankle-joint. In the upper two-thirds of its course it rests upon the interosseous membrane, to which it is fastened by firm fascia; in the lower third it lies first upon the front of the tibia and then upon the anterior ligament of the ankle-joint. For its upper two-thirds the artery has the tibialis anticus muscle just external to it; at the junction of the middle and lower thirds the extensor proprius pollicis comes from the outside and lies either upon the artery or to its inner side for the rest of its course. Externally in its upper third is the extensor communis digitorum; in the middle third is the extensor proprius pollicis; in the lower third, the proprius pollicis having crossed to the inner side, the extensor communis digitorum again becomes the outer boundary. The artery is covered by skin and by superficial and deep fascia. In its upper third it is deeply placed between the muscles; in its middle third it is less overlaid by muscle; in its lower third it is superficial except where it is crossed by the extensor proprius and where it is covered by the annular ligament. The artery has venæ comites. In the lower three-fourths of its course it is accompanied by the anterior tibial nerve, which in its course in the upper third of the leg is external to the artery; in the middle third it is external and a little in front of the artery; and in the lower third it is external to or upon the artery (Pl. 5, Fig. 5).

**Operations.**—The ligations of the anterior tibial (Pl. 5, Fig. 6) are: (1) of the lower third; (2) of the middle third; (3) of the upper third. In all these ligations the patient is placed recumbent with the leg extended, and the surgeon stands to the outer side of the extremity, cutting from above downward on the right side and from below upward on the left side.

*Ligation of the Lower Third.*—Make an incision 3 inches long in the line of the artery and over the annular ligament. This incision is external to the tibialis anticus muscle and ½ inch from the outer border of the tibia (Barker). Divide the skin and fascia, retract the tendon of the tibialis anticus inward, and the tendon of the extensor proprius pollicis outward, along with the tendons of the extensor communis. Flex the ankle-joint to relax the tendons and clear the artery. Draw the nerve external and pass the ligature from without inward. In order to recognize the muscles in this as in other ligations, rely largely upon the finger while the muscles are being moved.

*Ligation of the Middle Third.*—In this operation the procedure is similar to the above. Remember that the nerve lies in front of the vessel and that the extensor proprius pollicis muscle is external. The nerve is retracted outward and the needle is passed from the nerve. A good rule for detecting the artery is to find the outer edge of the tibia and by this locate the inter-




1, Anatomy, 2, Ligation, of the Dorsalis Pedis Artery. 3, Anatomy, 4, Ligation, of the Femoral Artery. 5, Anatomy, 6, Ligation, of the Posterior Tibial Artery. (From Bernard.)

osseous membrane, and then, by passing out along this membrane, discover the artery.

*Ligation of the Upper Third.*—Make an incision 3 inches long in the arterial line. On opening the deep fascia, do not rely on the eye for finding the muscular interspace, as often the latter cannot be seen, and neither a white nor a yellow line is reliable. Place the index-finger deep in the wound and have the tibialis anticus and extensor communis digitorum muscles successively rendered tense by an assistant. In opening the interspace use the handle of the knife. Relax the muscles, retract the tibialis anticus inward and draw the extensor communis digitorum outward. Find the interosseous membrane where it is attached to the edge of the tibia, and the artery will be found upon this membrane, between the tibia and the nerve. Clear the vessel and pass the ligature from without inward to avoid the nerve.

**Posterior Tibial Artery.**—The *line* of the posterior tibial is from the middle of the popliteal space to a point midway between the tip of the inner malleolus and the point of the heel (Pl. 6, Figs. 5, 6).

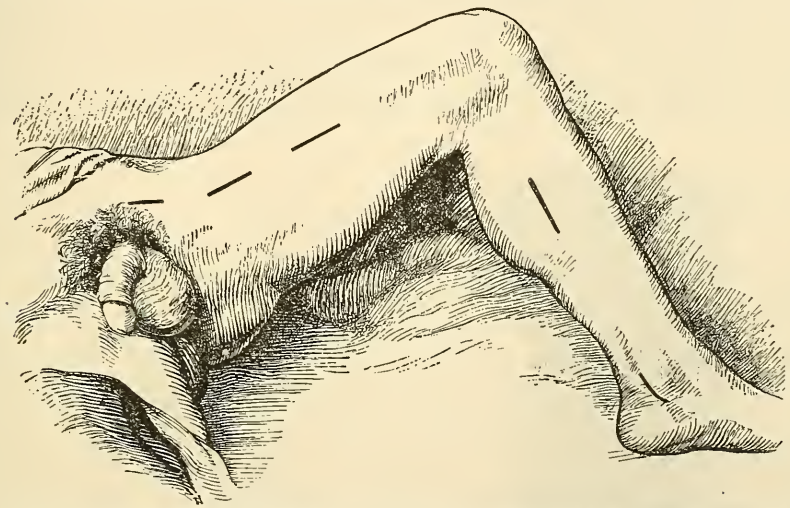


Fig. 245.—The lines indicate the incision to be made for the ligature of the common femoral, of the femoral in Scarpa's triangle and in Hunter's canal, and of the posterior tibial in the calf and behind the malleolus (MacCormac).

**Anatomy.**—The posterior tibial is the larger of the two terminal branches of the popliteal. It arises opposite the lower border of the popliteus muscle, passes down between the deep and superficial flexor muscles to midway between the tip of the malleolus and the point of the heel, and divides into the external and internal plantar vessels. In the upper third of its course it is very deeply placed midway between the tibia and fibula; in its middle third it is less deep, having passed inward; and in its lower third it is superficial. At the ankle the artery is beneath the annular ligament. From above downward the posterior tibial artery rests upon the posterior tibial muscle, the flexor longus digitorum muscle, the posterior surface of the tibia, and the internal lateral ligament of the ankle-joint. For the first inch or two of the course of the artery the posterior tibial nerve is to the inner side; the nerve then crosses to the outer side, and remains in that relative position throughout the rest of the course of the artery. When the knee is partly flexed and the leg is laid upon its outer surface the artery is between the operator and the nerve, and the nerve is between the artery and the table. Back of the malleo-

lus, in the first compartment, lies the posterior tibial muscle; in the next compartment is the flexor longus digitorum muscle; in the next compartment are the artery and nerve; and in the most posterior is the flexor longus pollicis muscle.

**Operations.**—*Ligation Back of the Malleolus.*—In this operation the patient is placed recumbent, with the thigh abducted and the leg flexed and resting upon its outer surface. The surgeon stands to the outer side. Make a 2-inch semilunar incision corresponding in its curve to the malleolus and ½ inch posterior to its margin (Fig. 245). Cut down to the annular ligament, incise the ligament, and find the artery and venæ comites. Clear the vessel and pass the needle from behind forward (to avoid the nerve, which is here posterior and external). Do not make the preliminary incision nearer the malleolus than ½ inch, as the sheath of the tibialis posticus muscle will then surely be opened. In closing the wound, suture the ligament by buried sutures of catgut before closing the superficial parts (Pl. 6, Fig. 6).

*Ligation in the Middle of the Leg.*—In this operation the patient is placed in the same position as for the ligation back of the malleolus. Feel for the inner border of the tibia, and make an incision 4 inches long 1 inch behind the osseous border, parallel with it, and extending through skin and superficial and deep fascia (Fig. 245). Draw the gastrocnemius muscle outward. Incise the soleus muscle, but not the fascia beneath the soleus; cut this fascia,

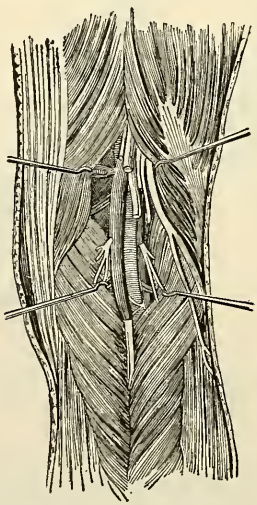


Fig. 246.—Anatomy of popliteal artery (Bernard and Huette).

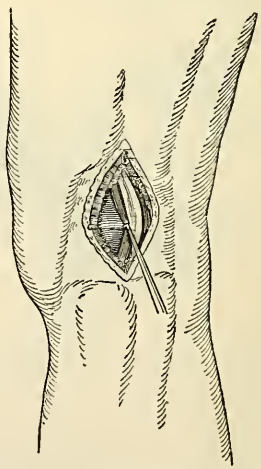


Fig. 247.—Ligation of popliteal artery in its upper third (Bernard and Huette).

after dropping the handle of the knife so that the blade is at right angles with the plane of the tibia. Clear the artery; pass the needle from without inward (Pl. 6, Fig. 6).

The **popliteal artery** is almost never ligated in continuity. It can be tied at the upper portion of the popliteal space, at the lower portion of the popliteal space, or at the inner side of the thigh.

**Anatomy** (Fig. 246).—The popliteal artery is the continuation of the femoral, and runs from the opening in the adductor magnus muscle to the lower margin of the popliteus muscle. This vessel runs downward and outward behind the knee-joint and in the popliteal space. The ham, or popliteal space, is a lozenge-shaped space, which above the joint is bounded on the

outer side by the biceps muscle, and on the inner side by the semitendinosus, semimembranosus, gracilis, and sartorius muscles, while below the joint it is bounded externally by the plantaris and outer head of the gastrocnemius muscles, and internally by the inner head of the gastrocnemius muscle. The floor of this space is formed by the surface of the femur, the posterior ligament of the knee-joint, the head of the tibia, and the popliteus fascia. The internal popliteal nerve passes down the middle of the popliteal space; it is superficial to the vessels in the upper half of the space, and is external to them; it is internal to the vessels in the lower half of the space. The external popliteal nerve is in the outer side of the space. The popliteal vein is between the nerve and the artery. Above the knee-joint it is to the outer side of the artery, but below the knee-joint it is to the inner side. The artery lies deeply in the space.

*Ligation in Upper Third.*—Place the patient prone. The surgeon stands to the outer side of the limb and makes a vertical incision 3 inches in length along the outer margin of the semimembranosus muscle, exposes the popliteal nerve, retracts the muscle inward and the nerve outward, exposes the artery, separates it from the other structures, and passes the needle from without inward (Fig. 247).

*Ligation in Lower Third.*—Make a 3-inch vertical incision between the heads of the gastrocnemius muscle. Avoid the external saphenous vein and nerve, and retract them with the popliteal nerve. Separate the artery from the vein and pass the needle from within outward.

**Femoral Artery.**—The *line* of the femoral artery is from midway between the anterior superior spine of the ilium and the symphysis pubis to the adductor tubercle on the inner condyle of the femur, the thigh being abducted and resting upon its outer surface (Pl. 6, Fig. 3).

**Anatomy.**—The femoral artery is the continuation of the external iliac trunk; it extends from the lower border of Poupart's ligament to the opening in the adductor magnus muscle, and hence occupies the upper two-thirds of the thigh. The artery for its first 5 inches is superficial, lying in Scarpa's triangle, a space which is bounded externally by the sartorius muscle and internally by the adductor longus, its base being Poupart's ligament and its floor being composed of the psoas, iliacus, pectineus, and adductor longus muscles, and often the adductor brevis. The artery enters the triangle as the common femoral, but after a 2-inch course it divides into the profunda (which passes deeply) and the superficial femoral. The latter vessel is the one alluded to in this section.

At the base of Scarpa's triangle the vein is internal, the artery is between, and the nerve is external (V. A. N.). At the apex of the triangle the vein is internal and a little posterior. At the apex of the triangle the superficial femoral passes under the sartorius muscle and enters into Hunter's canal, which occupies the middle third of the thigh and which terminates at an opening in the adductor magnus muscle. Hunter's canal is bounded externally by the vastus internus muscle, internally by the adductors longus and magnus, and its roof is fascia which stretches from the adductor longus to the vastus internus. In Hunter's canal the vein is behind the artery in the upper part, but external to it in the lower part, and is firmly attached to the artery. There may be two veins. Inside Hunter's canal, but outside the femoral sheath, is the long saphenous nerve, which crosses the artery from without inward.

A way to remember the relation of the femoral vein to the femoral artery is to recall the fact that the relation of the vein to the artery is always contrary to the relation of the sartorius muscle to the artery: when the sartorius muscle is external to the artery the vein is internal, as at the base of Scarpa's

triangle; when the sartorius muscle is crossing in front toward the inside of the artery, the vein is passing at the back to the outside, as at the apex of Scarpa's triangle; when the muscle is over the artery the vein is back of it, as in the upper third of Hunter's canal; and when the muscle is to the inside of the artery the vein is to the outside, as in the lower two-thirds of Hunter's canal. In a ligation at the apex of Scarpa's triangle the inner edge of the sartorius is the guide. In a ligation in Hunter's canal the long saphenous nerve is the guide.

**Operations.**—*Ligation of the Superficial Femoral at the Apex of Scarpa's Triangle.*—In this operation the *position* of the patient is supine, with the thigh and leg partly flexed, and the thigh abducted, everted, and rested upon its outer surface on a pillow. The operator stands to the outer side of the extremity. From a point corresponding to the middle of Scarpa's triangle, and 2½ inches below Poupart's ligament, make a 3-inch incision in the arterial line (Fig. 245). Cut the skin and superficial fascia. The saphenous vein will not be seen unless the incision is internal to the arterial line; if this vein is seen, draw it inward. Open the fascia lata, find the inner border of the sartorius muscle, and draw it outward. The fibers of this muscle run downward and inward, thus distinguishing it from the adductor longus, whose fibers run downward and outward. Open the common sheath for the artery and vein, and then incise the individual arterial sheath. Clear the artery and pass the ligature from within outward (Pl. 6, Fig. 4).

*Ligation of the Superficial Femoral in Hunter's Canal.*—This operation was first performed for aneurysm by John Hunter in 1785. In this operation the *position* of the patient is the same as in the ligation at the apex of Scarpa's triangle. Make a 3-inch incision in the middle third of the thigh, parallel with the arterial line and ½ inch internal to it (Barker) (Fig. 245). Incise the skin and superficial fascia, look out for the internal saphenous vein, open the fascia lata, find the sartorius muscle, and retract it inward, thus exposing the roof of Hunter's canal, which is to be opened for 1 inch or more. Within the canal is seen the long saphenous nerve, usually upon the sheath. Open the sheath of the artery, clear the vessel, and pass the needle from without inward.

Results: Ligation at the apex of Scarpa's triangle is a method for treating popliteal aneurysm. It is a very successful procedure. I have performed it 3 times with success and have assisted other operators in 3 successful cases. Syme successfully ligated the femoral about its middle 23 consecutive times, and in Guy's hospital the same operation was done 24 times, with 1 death ("Practice of Surgery," by Thomas D. Bryant).

**Iliac Arteries.**—The *line* of the common and external iliac arteries is from a point ½ inch below and ½ inch to the left of the umbilicus to midway between the anterior superior spine of the ilium and the pubic symphysis. The upper third of this line represents the common iliac, and the lower two-thirds the external iliac (Pl. 3, Fig. 4).

**Anatomy.**—The common iliac arteries arise from the aorta opposite the left side and lower border of the fourth lumbar vertebra, and extend to the upper margin of the right and left sacro-iliac joints, where they each bifurcate into an external and an internal iliac. The common iliac arteries lie upon the fifth lumbar vertebra, are covered with peritoneum, and are crossed by the ureters. In women the ovarian arteries cross the common iliacs. Each common iliac vein lies to the right side of its associated artery. The right common iliac artery has in front of it, besides the peritoneum and ureter (in women also the ovarian artery), the ileum, branches of the superior mesenteric artery, and branches of the sympathetic nerve. The left common iliac artery has in front of it, in addition to structures common to both sides (ureter,

ovarian artery, sympathetic branches), branches of the inferior mesenteric artery, and the sigmoid flexure with its mesocolon. The internal iliac artery runs from the sacro-iliac joint to the upper margin of the great sacrosciatic foramen. It is very rarely ligated (only for gluteal aneurysm, for uncontrollable hemorrhage from the gluteal or sciatic arteries, or to produce atrophy of the prostate gland). The external iliac artery runs from the sacro-iliac joint along the pelvic brim, upon the inner edge of the psoas muscle, to Poupart's ligament. The external iliac vein is internal to the artery. On the right side, high up, it passes behind the artery. The external iliac artery has in front of it peritoneum and subserous tissue (Abernethy's fascia). The ileum crosses the right, and the sigmoid flexure crosses the left, external iliac artery. The genital branch of the genitocrural nerve crosses the artery low down, and the circumflex iliac vein crosses it just before it terminates in the femoral. The spermatic vessels and the vas deferens in the male, and the ovarian vessels in the female, lie upon the artery near its termination. Sometimes the ureter crosses the vessel near its point of origin.

*Ligation of the Iliac Arteries After Abdominal Section.*—The best method for ligating the common, the internal, and sometimes the external iliac is by abdominal section. The patient is placed in the Trendelenburg position. The abdomen is opened in the midline below the umbilicus or in the semilunar line of the diseased side. The intestines are lifted toward the diaphragm, and are held up by gauze pads. The edges of the incision are retracted. The vessel to be tied is located and the point for ligation is selected. The posterior layer of the peritoneum is opened over the selected point, the vessel is cleared, and the threaded Dupuytren's aneurysm needle is passed in a direction away from the vein. In ligating either common iliac pass the needle from right to left. In ligating the external iliac pass the ligature from within outward. It is not necessary to suture the posterior layer of peritoneum. The abdomen is closed without a drain. In these operations be sure to push the ureter out of the way. This operation has been performed by Dennis, Hearn, Marmaduke Shield, Mitchell Banks, the author, and others.

Results: Bryant ("Operative Surgery") alludes to 5 reported cases of transperitoneal ligation of the common iliac artery, with 1 death.

*Ligation of the Common Iliac Artery by the Extraperitoneal Method.*—The common iliac artery was tied unsuccessfully by Dr. Wm. Gibson in 1812. It was first successfully ligated by Valentine Mott in 1827. The patient is placed recumbent or in the Trendelenburg position. The body is then turned a little to the opposite side and the thighs are partly flexed. Bryant says there are two linear guides for this artery. *Crampton's line* is drawn from "the apex of the cartilage of the last rib downward and a little forward nearly to the crest of the ilium, then carried forward parallel with it to a little below the anterior superior spine" ("Operative Surgery," by Joseph D. Bryant). *McKee's line* is "drawn from the tip of the cartilage of the eleventh rib to a point 1½ inches within the anterior superior spine, then curved downward, forward, and inward, and terminating abruptly above the internal abdominal ring" (Ibid.).

The incision can be begun just external to the internal abdominal ring and be curved upward and outward as in ligation of the external iliac, but Crampton's incision gives more room. The superficial tissues are divided down to the transversalis fascia, this structure is nicked and divided, and the exposed and unopened peritoneum is rolled upward and inward. The muscular guide is the inner border of the psoas magnus muscle. By its side an artery is felt. If the sacrovertebral prominence is above the vessel touched, the artery is the external iliac; otherwise it is the common iliac. If the external iliac is the vessel first exposed, follow it up to find the common trunk.

When the common iliac is found, separate the fatty tissue about it and pass the ligature from the right toward the left in order to avoid the associated vein.

Results: Jos. D. Bryant tells us that this vessel has been ligated by the extraperitoneal method 69 times, with only 16 recoveries, but it is to be remembered that many of these operations were in pre-antiseptic days. The artery has been tied 80 times, with 56 deaths (70 per cent.).

Twenty-one of these operations were done since 1880; there were 10 deaths (mortality of nearly 48 per cent.). In these 21 cases gangrene occurred 7 times. (See Wm. J. Gillette, in "Annals of Surgery," July, 1908.)

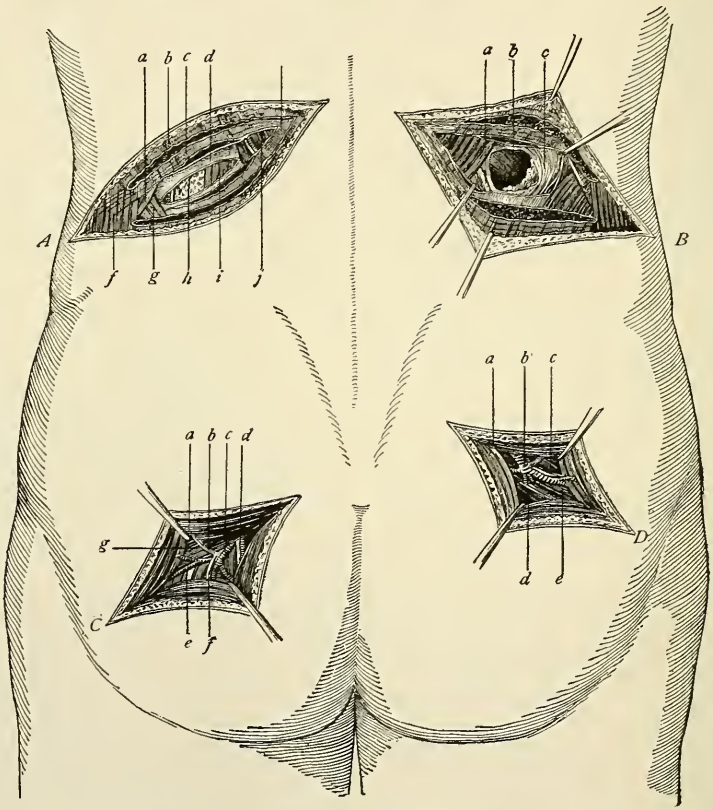



Fig. 248.—*A*, Nephrotomy: *a*, last dorsal n.; *b*, latissimus dorsal m.; *c*, serratus post. inferior m.; *d*, middle layer of lumbar fascia; *e*, outer layer; *f*, ext. oblique m.; *g*, int. oblique m.; *h*, perinephritic (extraperitoneal) fat; *i*, quadratus lumborum m.; *j*, erector spinæ m. *B*, Nephrotomy: *a*, first lumbar n.; *b*, kidney; *c*, transversalis fascia. *C*, Ligature of the sciatic and internal pudic arteries, and exposure of the great sciatic, small sciatic, and internal pudic nerves: *a*, gluteus maximus m.; *b*, inf. gluteal n.; *c*, sciatic a.; *d*, int. pudic a. and n.; *e*, great sciatic n.; *f*, small sciatic n.; *g*, pyriformis m. *D*, Ligature of the gluteal artery and exposure of the superior gluteal nerve: *a*, gluteus maximus m.; *b*, gluteal a.; *c*, superior gluteal n.; *d*, pyriformis m.; *e*, gluteus medius m. (Kocher).

*Extraperitoneal Ligation of the Internal Iliac Artery.*—This operation was first performed by Stevens, of Vera Cruz, in 1812 ("Practice of Surgery," by Thomas Bryant). The incision and the method of exposing the vessel are identical with like steps in the ligation of the common iliac.

Results: Of 26 ligations of this vessel recorded, 18 were fatal, but only a few of the cases were done antiseptically (Joseph D. Bryant's "Operative Surgery").

*Ligation of the External Iliac by Abernethy's Extraperitoneal Method* (Pl. 3, Fig. 4).—The external iliac artery was first ligated by Abernethy in 1796. The operation failed, but he did the first successful operation in 1806. The patient is placed recumbent with the thighs extended during the first incisions; but in the later stages of the operation the thighs are flexed a little to relax the abdominal structures. The operator stands to the outer side. The surgeon will find the artery by the side of the psoas muscle. Mark a point 1 inch above and 1 inch external to the middle of Poupart's ligament, and another point 1 inch above and 1 inch internal to the anterior superior iliac spine (Barker). Join these two points by a curved incision 4 inches long and convex downward. Cut the skin, the fat, the two oblique muscles, and the transversalis muscle; open the transversalis fascia, separate the peritoneum toward the vessels, and draw it inward by a broad retractor, and look for the artery along the pelvic brim. The anterior crural nerve is seen to the outer side of the artery, the external iliac vein is to the inner side of the artery, and the genitocrural nerve is upon the artery. Clear the artery near its middle and pass the ligature from within outward. In Sir Astley Cooper's method of ligation the inguinal canal is opened; in Abernethy's method the inguinal canal is not opened.

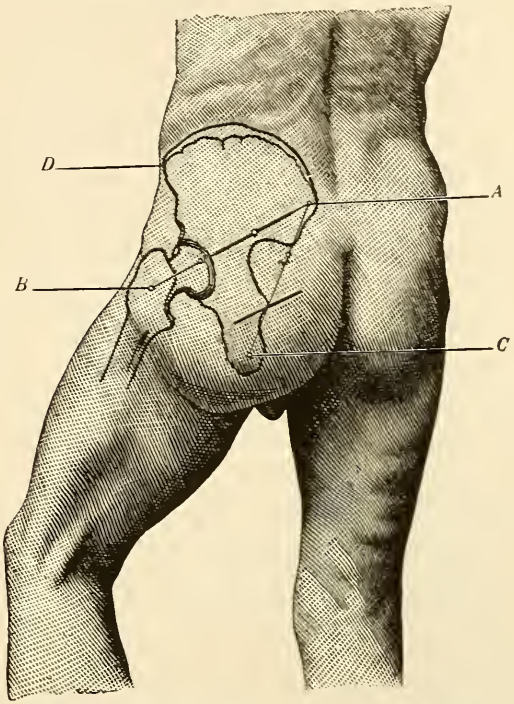



Fig. 249.—Position and direction of the superficial incisions which must be made in order to secure the gluteal artery and the sciatic and pudic arteries: *A*, Posterior superior iliac spine; *B*, great trochanter; *C*, tuberosity of the ischium; *D*, anterior superior iliac spine; *A–B*, iliotrochanteric line, divided into thirds. This line corresponds in direction with the fibers of the gluteus maximus muscle. The incision to reach the gluteal artery is indicated by the darker portion of the line. Its center is at the junction of the upper and middle thirds of the iliotrochanteric line, and exactly corresponds with the point of emergence of the gluteal artery from the great sciatic notch. *A–C*, ilio-ischiatic line. The incision to reach the sciatic artery and internal pudic is indicated by the lower dark line. It is also to be made in the direction of the fibers of the gluteus maximus muscle. The center of the wound corresponds to the junction of the lower with the middle third of the ilio-ischiatic line (MacCormac).

**The Gluteal Artery.**—This vessel is a continuation of the posterior division of the internal iliac. It emerges from the great sacro-sciatic foramen at the upper border of the pyriformis muscle. It rests upon the gluteus minimus, divides into three branches, and is covered by the gluteus maximus muscle. The superior gluteal nerve lies inferior to the artery (Fig. 248).

*Ligation.*—The patient should be prone. The surgeon stands to the outer side. The incision corresponds to a line drawn from the posterior superior iliac spine to the upper border of the great trochanter (Fig. 249). Divide the skin, fascia, gluteus maximus muscle, and the fascia over the gluteus medius muscle, and retract the gluteus medius upward. Feel for the great sacro-sciatic foramen, and at this point the artery is found above the pyriformis

muscle. Clear the vessel and pass the needle from below upward (Kocher's "Operative Surgery"). There is practically no mortality from this operation.

**The Sciatic Artery.**—This artery is the larger of the terminal branches of the anterior division of the internal iliac artery. It passes to the lower portion of the great sacrosciatic foramen, lying back of the internal pudic artery, and resting upon the sacral plexus of nerves and pyriformis muscle (Gray). It leaves the pelvis between the pyriformis and coccygeus muscles, and passes downward between the ischial tuberosity and great trochanter. It is covered by the glutæus maximus muscle, rests upon the gemelli, internal obturator, and quadratus femoris muscles, has the great sciatic nerve external to it, and the small sciatic nerve external and posterior (Fig. 248).

*Ligation.*—The patient lies prone. The surgeon stands to the outer side. The incision "corresponds to the middle two-thirds of a line extending from the posterior inferior iliac spine to the base of the great trochanter."[1] MacCormac advises the incision shown in Fig. 249. Divide the skin, fat, fascia, and the glutæus maximus muscle. Find the artery at the lower border of the pyriformis muscle and trace it to its point of emergence from the pelvis. Pass the ligature from without inward. There is practically no mortality from this operation.

**Internal Pudic Artery.**—This artery is one of the terminal branches of the anterior trunk of the internal iliac. It passes to the lower margin of the great sacrosciatic foramen, and leaves the pelvis between the pyriformis and coccygeus muscles, crosses the ischial spine, and again enters the pelvis by the lesser sacrosciatic foramen. The vessel is accompanied by the internal pudic nerve (Fig. 248).

*Ligation.*—The position of the patient and the incision are the same as for ligation of the sciatic artery (Fig. 249). The artery is found below the ischial spine. Pass the needle from below upward to avoid the nerve. There is practically no mortality from this operation.

**Ligation of the Abdominal Aorta.**—This operation was first performed by Sir Astley Cooper in 1817. The patient lived but a few hours. The aorta has been ligated twenty times and there have been twenty deaths. Nine of these cases were aseptic operations. The patient of Monteiro, of Rio Janeiro, lived for ten days. The circulation was entirely restored in the limbs, and the man died from hemorrhage resulting from the ulceration produced by a septic ligature. Keen's case lived for forty-eight days after ligation just below the diaphragm. The urinary secretion was plentiful and the circulation in the lower extremities was restored, death resulting from cutting through of the ligature. Robert T. Morris performed distal ligation below an aneurysm. He encircled the aorta with a soft-rubber catheter and clamped it with forceps. Twenty-two hours after operation the aneurysm began to shrink, and in three hours more had apparently disappeared. Twenty-seven hours after operation the clamp and catheter were removed. The patient died of septicemia fifty-three hours after operation. The necropsy disclosed gangrene of a bit of intestine which had been in contact with the forceps, but the dissecting aneurysm was filled with solid clot, the aorta was patent, and the circulation in the extremities was re-established ("Amer. Jour. of Med. Sciences," Sept., 1900). These cases prove that under certain circumstances the operation is feasible, and in desperate cases it must be considered as a possible means of treatment. Halsted and Matas prefer a metal band to the ligature (see page 429). Gradual occlusion and partial occlusion of the aorta are much safer than sudden closure.

*Murray Operation.*—This procedure aims to avoid opening the peritoneum. An incision is made from just below the tip of the tenth rib to a point 1 inch internal to the anterior superior iliac spine. The peritoneum is separated

[1] Kocher's "Operative Surgery," by Stiles.

from the abdominal wall until the vessel is reached. Cooper's operation by abdominal section is the preferable procedure.

*Operation by Abdominal Section (Cooper's Operation); Instruments Required.*—Those used in any ligation, with the addition of an aneurysm needle with a large curve and a very long handle. With an ordinary instrument it is extremely difficult to pass the ligature. It would be a great advantage to use an instrument which, after being passed under the vessel, could have a central eyed shaft projected, as is the center shaft of a Bellocq cannula. Floss silk is probably the best ligature material.

If the patient is much exhausted, an assistant should infuse salt solution in a vein during the operation. In Keen's case there was profound shock, but the moment the ligature was tightened it passed away.

The patient should be placed upon his back. The surgeon stands to the right of the patient and opens the abdomen in the median line, a little above the level of the aneurysm. The intestines are packed aside, the posterior layer of the peritoneum is divided, the surface of the aorta over a small area is cleared of nerves, the plexuses being separated with a blunt dissector.

The needle is passed from right to left. A double ligature of floss silk should be passed and the ends should be tied with a stay-knot. The wound is closed and dressed.

It has been suggested—I think by Wyeth—that it might be wise to only partially tighten the ligature at first, completing the occlusion of the artery after a day or two. Such a procedure would certainly give a better chance for the collaterals to dilate and restore circulation in the legs (see page 429).

Unfortunately, in an aneurysm, the vessel will usually be extensively diseased, and ligation will be out of the question. If, however, a normal region is found, the chance of success in a case of aneurysm will be greater than in a case of hemorrhage from a branch of the aorta, because, in a case of aneurysm, the probabilities are that the collaterals are somewhat distended before a ligature is applied.

## XX. DISEASES AND INJURIES OF BONES AND JOINTS

### Diseases of the Bones

**Atrophy of bone** is a diminution in the amount of bony matter without change in osseous structure. It arises from want of use (as seen in the wasting of the bone of a stump) or from pressure (as seen in the destruction of the sternum by an aneurysm of the aorta). *Eccentric* atrophy is the thinning of a long bone from within, the outer surface being perhaps unchanged. It is usually a senile change. *Concentric* atrophy means a thinning of the outer surface of the shaft, causing a lessened diameter. It is usually linked with eccentric atrophy.

**Hypertrophy of bone** may be due to increased blood-supply (as seen in chronic epiphyseal inflammation), the bone growing much more than does its fellow. It may arise from excessive use or from strain (as seen in the increased size of the fibula when the tibia is congenitally absent).

**Tumors of Bone.**—Bones give origin to both innocent and malignant tumors. Myeloid sarcoma takes origin in the endosteum and expands the bone. The fasciculated sarcoma is a periosteal growth. Besides these growths there may develop an osteoma, a chondroma, and secondary deposits of cancer and sarcoma. A bone may become cystic, and occasionally the cysts are due to hydatids. Gummata are frequently met with.

**Cysts and Cystomata of Bone.**—One variety of cyst is found in the jaws (dentigerous cysts, see page 361). The other variety occurs usually in the medullary canal of long bones and very seldom in short bones and flat bones. "It differs from the dentigerous cyst in the absence of a connective-tissue capsule. The fluid is usually hemorrhagic. Islands of cartilage may be found in the bone capsule" (Bloodgood, in "Progressive Medicine," Dec. 1, 1907).

A bone-cyst slowly expands and thins the shaft of the bone, and in some cases fracture of the bone is the first evidence of the trouble. Union takes place after fracture, but the enlargement remains. The *x*-ray picture does not enable us to make the diagnosis, because it exactly resembles the picture of any medullary growth possessed of a bony capsule and producing osseous absorption.

The diagnosis is proved by exploratory incision and the condition is treated by curettement and drainage.

Many bone-cysts are produced by softening of solid neoplasms (sarcoma, myxoma, medullary fibroma, chondroma). Occasionally cysts form in osteomalacia and osteitis deformans, the condition arising from softening. Hydatid cysts and dermoid cysts are sometimes encountered. A true cystoma of bone, except in one of the jaws, is a surgical rarity. In the jaws cystomata are not very uncommon.

**Syphilis of Bone.**—Secondary syphilis may attack the bones (see page 326). Tertiary syphilitic lesions are considered on page 330.

**Actinomycosis of bone** is most usual in the jaw, but may attack the orbit, ribs, sternum, or limbs (see page 309). Actinomycosis of bone may arise secondarily after infection of superficial parts by the streptothrix. In the jaw the fungus obtains entrance to the interior of the bone through a tooth socket. In some cases of bone actinomycosis the fungus reaches the bone by the blood. Actinomycosis leads to the production of granulation tissue, the bone is expanded and becomes carious, and a quantity of new bone is sometimes produced. In vertebral actinomycosis, although the condition resembles tuberculosis, angular deformity does not occur.

**Tuberculosis of bone** (*tuberculous osteomyelitis*) tends especially to appear in the cancellous ends of long bones. In about one-fifth of the cases it is primary, that is, only one focus can be found. In such cases the point of entry shows no lesion, or the lesion at that point may have healed. In some cases the bacilli enter through the tissue. Trauma may be an exciting cause. Long after apparent healing the disease process may awaken into activity and trauma is often the cause of the awakening. In one of König's cases trouble began anew after sixty years. The disease is especially apt to attack the epiphysis and spread to the joint, although in some cases it spreads to the shaft. Primary tuberculosis is rare in the shafts of long bones, but is not uncommon in the shafts of short bones. A bone focus leads to the formation of a bone cavity which may contain tuberculous granulations or bone sequestra. The sequestra in tuberculous osteomyelitis are not completely loose, but are still attached at some point. The bone may sclerose or may undergo alterations of an osteoporotic nature, making it soft. Sclerotic bone means a healing process. Softened bone means a spreading process. A sequestrum in tuberculous osteomyelitis is usually wedge shaped, the base being toward the joint. A sequestrum is due, König thinks, to the obstruction of a terminal artery by a tuberculous embolus or by the intra-arterial growth of bacilli ("Die Tuberculose der Menschlichen Gelenke, swiel der Brustwand und des Schaedels"). In a certain number of cases tuberculosis infiltrates a spongy bone with great rapidity because of rapid caseation. This condition is known as *infiltrating progressive bone tuberculosis.*

**Osteitis, Periostitis, and Osteoperiostitis.**—Osteitis, or **inflammation of bone,** may be due to traumatism, to a constitutional malady or diathesis, to the extension of inflammation from some other structure, to certain fevers, to cold, to phosphorus or mercury, to infection, or to working in pearl button factories. In inflammation of bone the exudate and leukocytes pass into the Haversian canals, spaces, and canaliculi. The bone corpuscles proliferate and the bone undergoes thinning (rarefaction), not because of pressure, but because of absorption by voracious leukocytes and osteoclasts. This process of rarefaction enlarges all the bony spaces, and by destroying septa throws many of the spaces into one. If the surface of a bone inflames, the periosteum will be separated more or less by exudation, and the bone will be covered with little pits or erosions made by the leukocytes. Inflamed bone is so soft that it can readily be cut with a knife.

*Pearl workers' osteitis* occurs particularly in youths before fusion of the epiphyses. It arises in the diaphysis by the epiphysis. The bones of the limbs are most apt to suffer, but the bones of the face or chest may be attacked. The attack begins with pain and moderately elevated temperature and the fever may persist for several weeks. The condition may apparently get well and yet begin again when the patient returns to work. The lesions are often symmetrical and always multiple (Broca). It is a condensing osteitis and undergoes spontaneous cure if the patient gives up the occupation (Deturk, in "Archives Générales de Chirurgie," Nov., 1908).

Osteitis may terminate in *resolution* or it may terminate in *sclerosis*, the mass of proliferating cells being converted first into fibrous tissue and next into dense bone which contains a very few small cancellous spaces. If the exudation is under the periosteum, the bone will be thickened at this point, bone stalactites marking the points of passage of the vessels. Osteitis may terminate in *suppuration*, this condition being often called *caries*. In tuberculous osteitis caseation of the inflammatory products is very apt to arise (*tuberculous caries*, the *strumous caries* of our predecessors). Acute osteitis may terminate in *necrosis*, the inflammatory exudate compressing the vessels in their bony canals, a portion of the bone being, in consequence, deprived of nutritive material. The portion cut off from nutritive fluid dies *en masse* (necrosis). Osteitis is usually associated with more or less periostitis. A simple acute periostitis without involvement of the bone may arise from traumatism or strain; but in all severe cases of periostitis, in all chronic cases, in all cases due to syphilis, rheumatism, measles, scarlatina, or enteric fever the bone is involved at the same time or subsequently. In syphilitic states gummatous degeneration frequently ensues.

**Symptoms of Osteitis and Osteoperiostitis.**—As a chronic process, *osteitis* is most commonly found in the femur. The history may exhibit a record of an antecedent injury or chilling of the body. Pain is severe, boring or aching in character, deep seated, worse at night, and aggravated by a dependent position of the part. The symptoms closely resemble those of periostitis, with which disease it is almost sure to be linked. Tenderness exists on percussion, and sometimes on pressure. Subperiosteal swelling, fusiform in shape, is noted; cutaneous edema and discoloration are observed if a superficial bone is inflamed. In syphilis atrophic osteitis may attack the cranial bones and produce softening or even perforation, or osteophytic osteitis may arise, exostoses being formed. *Osteoperiostitis* may be acute or chronic, circumscribed or diffused, and may terminate in resolution, organization, or suppuration. It arises from cold, blows, wounds, strains, the spread of adjacent inflammation, specific febrile maladies, pyogenic infection, syphilis, rheumatism, or tuberculosis. The symptoms are pain (which is worse at night and which is aggravated by motion, pressure, or a dependent position), swelling, edema, and

discoloration of the soft parts. Pain in the syphilitic form is not so severe as in other varieties. *Acute necrosis* or *diffuse osteoperiostitis*, a pyogenic inflammation of bone and periosteum, is commonest in boys about the age of puberty. It is usually awakened by cold, a specific fever, or injury, and most often affects the tibia or femur; the symptoms locally are redness, swelling, and severe pain; constitutionally there are rigors, fever, and sometimes convulsions. Necrosis is apt to result. Pyemia is common. In *simple acute periostitis* a swelling is felt upon the osseous surface. The swelling is firmly fixed and is very tender, but the bone itself is not enlarged. There is some local heat, discoloration, often fever, and the patient complains of an aching pain, which is worse at night.

Periostitis due to strain demands some special attention. Sir James Paget, years ago, pointed out that muscular exertion might cause periostitis. C. T. Dent has written a valuable article upon this subject.[1]

It is common to hear football players complain of some swelling of the knee-joint. Examination finds tenderness over the tubercle of the tibia with slight swelling of the joint. Dent points out that pain is felt on straightening the leg, not on rotating it. The same observer states that omnibus drivers suffer from periostitis of the fibula, due to pressing forcibly against the footboard; those who ride may develop periostitis of the adductor insertion (riders' bone); the victims of flat-foot may labor under periostitis of the inner tuberosity of the os calcis; bar-keepers, from working a beer-pump, may get periostitis of the scapula, pain being marked on contracting the biceps; a housemaid may develop periostitis at the points of bony origin of the great pectoral from the chest, the condition being due to sweeping and scrubbing.[2]

**Treatment of Osteitis and Osteoperiostitis.**—In syphilitic forms the local treatment consists in rest, elevation of the part, the application of iodin and mercurial ointment, and bandaging. Specific treatment is given by the stomach or hypodermatically. Operation is rarely justifiable. In other forms, if the case be recent and severe, put the patient to bed, place the limb in a splint and elevate it, employ cold, apply a bandage, and give salines and iodid of potassium internally. Later use ichthyol inunctions locally and apply a hot-water bag. Morphin is administered for pain. If these means fail, order counterirritation by iodin and blue ointment or blisters, and apply heat locally. In severe cases take a tenotome and slit the periosteum subcutaneously to relieve tension; this procedure often quickly relieves the pain. Some cases demand longitudinal osteotomy, which is performed by taking Hey's saw and dividing the bone longitudinally into the medullary canal. If pus forms, drain at once.

*Diffuse osteoperiostitis* requires early and free incisions through the periosteum, antiseptic irrigation, drainage, rest and elevation of the limb, and strong supporting and stimulating treatment. Amputation is sometimes demanded, as when the patient grows weaker and weaker even after incision, and when a joint is seriously involved. If the necrosis affects the entire shaft, which separates from its epiphyses, and new bone has not yet formed from the periosteum, make a subperiosteal resection of the shaft.

**Chronic periostitis** is usually syphilitic. A *node* is a chronic inflammation of the deep periosteal layers. Nodes occurring early in the secondary stage remain soft and soon pass away under treatment, but those occurring two years or more after infection are apt to cause a bony deposit. A node may soften, leaving a sinus, at the bottom of which is a piece of dead bone. Gumma of the periosteum is one form of node which is apt to produce caries or necrosis.

**Osteoplastic periostitis** accompanies chronic osteitis and causes the deposit of new bone, which undergoes sclerosis. The chief *symptom* is aching

[1] "Practitioner," Oct., 1897. [2] Ibid.

pain, which is worse when the patient is warm in bed, and is aggravated by damp and wet. A hard swelling is found at the seat of pain (often over the tibia, ulna, clavicle, or sternum). The soft parts are uninflamed and move freely unless softening or suppuration has occurred. Tenderness is manifest.

*Treatment of Chronic Periostitis and Osteoplastic Periostitis.*—For the nodes of early syphilis administer mercury by the plan usually followed in secondary syphilis; for the nodes of late syphilis give mercury and large advancing doses of iodid of potassium. Blisters, blue ointment, and iodin are applied to the skin over the area of periostitis in both forms, and subcutaneous division of the periosteum is of value. If suppuration occurs, incise antiseptically and drain.

**Chronic Abscess of Bone, or Brodie's Abscess.**—This condition is sometimes due primarily to tuberculous infection, symptoms being absent for a longer or shorter time and arising because of secondary infection with staphylococci. It is always chronic, never acute. A very acute inflammation, such as is induced by virulent pyogenic organisms, causes acute necrosis rather than an acute abscess. After typhoid fever an area of suppuration may slowly form in the head of a long bone, due to action of typhoid bacilli. Nonvirulent staphylococci may be responsible, and the condition may follow long after a staphylococcus osteomyelitis. In 84 per cent. of cases of Brodie's abscess this is the history (Alexis Thomson). The same author says the latest period between the osteomyelitis and the abscess varies from one to fifty-seven years. Chronic abscess of bone was first described by Sir Benjamin Brodie, and is often called *Brodie's abscess*. It occurs in the cancellous structure of the ends of bones—usually in the head of the tibia, sometimes in the femur (Fig. 250) or humerus. It seldom occurs in the shaft of a long bone. A tuberculous abscess of bone may follow a slight injury, which constitutes a point of least resistance. Bacteria lodge and multiply; bone rarefaction leads to the formation of a cavity, the inflammatory products caseate, suppuration arises, and the surrounding bone thickens and hardens because of growth from the periosteum. The abscess is apt to break and often breaks into a joint, as the joint surface is not covered by periosteum and no barrier of bone is there formed. Brodie's abscess may induce necrosis.

Fig. 250.—Chronic abscess in the great trochanter ("American Text-Book of Surgery").

Alexis Thomson thus describes Brodie's abscess ("Edinburg Med, Jour.," April, 1906):

In the first or quiescent stage there is a cavity filled with serum and lined with a membrane like the periosteum of young bones. The outer layer of the membrane is forming new bone of a spongy nature, "further away the old bone is sclerosed and the medullary canal obliterated."

When the mature stage or abscess stage arises the lining membrane is converted into granulation tissue, and the cavity becomes filled with staphylococcus pus. The outer layer of granulations erodes the bone and the abscess progressively enlarges. As the bone is eroded within, new bone is formed by the periosteum and the bone enlarges. If pus formation is more rapid than bone erosion there is tension and pain, but if bone erosion is suffi-

ciently rapid to prevent tension there is little or no pain. Finally, the abscess perforates the bony shell "on the periosteal surface or into an adjacent joint."

**Symptoms.**—There are attacks of boring pain, worse at night and aggravated by motion and pressure and a dependent position. The pain is intermittent, and may be absent for many days at a time. These pains are frequently thought to be rheumatic. The tenderness is marked, even when pain is absent, and is not in the joint, as the patient believed the pain was, but is over the site of infection. If the head of the tibia or the great trochanter is the seat of disease, percussion over that region develops pain most certainly. At times pain in the bone becomes excruciating and tenderness acute. There is more or less loss of function in the limb and in far advanced cases the bone is enlarged. There may be thickening of the bone and soft parts, edema and discoloration of the skin over the seat of trouble, and attack after attack of synovitis in the nearest joint. Irregular fever and sweats are usually noted, but there may be no fever. The harrassing pain causes sleeplessness, exhaustion, and emaciation. When the pus breaks through the bone abscess develops in the soft part, and if this bursts or is opened pain ceases (Thomson). The *x*-rays aid greatly in making the diagnosis.

**Treatment.**—In treating bone-abscess, trephine the bone at the point of greatest tenderness, and if the abscess is missed, follow the advice of Holmes and perforate the wall of bone with the trephine, opening in several directions, to discover the pus. It is often easy to open into the abscess with a chisel or gouge. After opening the cavity scrape its walls, remove dead bone, thoroughly dry with gauze, touch with pure carbolic acid, and pack with iodoform gauze. If the abscess opens into a joint, trephine the bone, and also open, irrigate, and drain the joint.

**Caries** was a term once used universally to signify suppuration or molecular death of bone. In some cases caries means suppurative osteitis; in others, tuberculous osteitis; in still others, gummatous osteitis. Typhoid fever is occasionally followed by a carious condition of bone. Osteitis is apt to become purulent when the bone is exposed to the air, when rest is not secured, when the health of the individual is below normal, when a foreign body such as a bullet is in the bone, or when tubercle or syphilis exists. The term is seldom used to-day except loosely, and then usually to signify tuberculous disease of bone. When caries arises the softened and granulating bone breaks down and is eventually discharged through a sinus. After drainage is secured, organization, sclerosis, and healing may result. In these cases new bone may form and a cure follow.

*Tuberculous* or *strumous caries* (*caseous osteitis*), a condition produced by the caseation of the products of a tuberculous osteitis, seldom shows any tendency to self-cure, neither organization nor sclerosis takes place, and new bone seldom forms unless an operation is performed. The interior of bones, especially of the carpus and tarsus, is entirely softened and destroyed and thin shells only are left.

*Caries necrotica* is a condition in which small but visible portions of soft and dead bone are cast off; *caries sicca* is molecular death of bone without liquefaction or suppuration.

The caseating masses in tuberculous caries contain tubercle bacilli. If a tuberculous collection is evacuated and infection with pus organisms occurs, genuine suppuration takes place, and constitutional infection causes septic fever and may cause death. Pyogenic osteitis may affect any part of any bone; but caseous osteitis (tuberculous caries) tends to arise, especially in cancellous structure (heads of long bones, vertebral bodies, ribs and sternum, and bones of the carpus and tarsus). Tuberculous osteitis of the shaft

of a long bone occasionally, but rarely, arises. Tuberculous osteitis is apt to cause tuberculous disease in an adjacent joint. Tuberculous osteitis may be followed by the formation of a cold abscess.

**Symptoms.**—In the beginning the evidences of caries are usually those of osteitis, but the first sign noted may be a fluctuating swelling due to pus or to caseated tubercle. After a time, at any rate, a fluctuating swelling is discovered. If not opened, the softened mass breaks externally, voids its contents, and leaves a sinus from which flows caseated matter which after a time becomes thin, reddish, and irritating to the skin, contains small portions of gritty bone, and has a foul smell. The opening of the sinus fills up with edematous granulations. A probe carried to the bottom of the sinus finds bone which is sieve-like (worm-eaten), and which on being struck gives a muffled note rather than the clear, sharp note of necrosis; the bone is rough, is bared, and is so soft that the probe can usually be stuck into it. In old cases of caries amyloid disease may arise.

**Treatment.**—If syphilis exists, give iodid of potassium in advancing doses and a mild mercurial course. If tuberculosis exists, give iodid of iron, arsenic, cod-liver oil, and nourishing foods, and recommend ocean air and living in the open air. Locally, in all cases, insist on rest and at once secure drainage, enlarging the opening, if necessary, and inserting a tube, and even making additional openings; syringe often with antiseptic fluids and dress antiseptically. If the case is seen before spontaneous evacuation has occurred, open under strict antiseptic precautions. When a chronic sinus exists there arises the question of operation. Incomplete operations are worse than useless, for they may be followed by diffuse tuberculosis or pyemia. If the gouge is used, try to remova *all* carious bone. The diseased bone is white, crumbles, and does not bleed; the non-carious bone is pink and vascular. Scrape away all granulations, swab the cavity with pure carbolic acid, and pack it with iodoform gauze. Instead of gouging away bone, there may be used the actual cautery, sulphuric acid, or hydrochloric acid. In severe cases excision is required, and in some rare cases amputation may be necessary. Caries of the spine is considered under Diseases of the Spine.

**Necrosis** is the death of visible portions of bone from circulatory impediment or the direct action of bacterial toxins. It is analogous to gangrene. One cause of necrosis is traumatism (such as the tearing off of periosteum) which deprives the bone of blood. Inflammation of the periosteum further lessens the nutrition. Acute inflammation in bone causes necrosis, the excessive exudation in the canals and spaces occluding the blood-vessels by pressure. The occlusion of vessels by bacterial thrombi or emboli may lead to necrosis, or the direct action of toxins may first inflame and finally destroy a portion of the bone. A thin shell of bone only may necrose from periosteal separation, or an entire shaft may die from acute pyogenic osteomyelitis or diffuse infective periostitis. Osteomyelitis is the most usual cause of necrosis. Necrosis is most frequently met with in the diaphyses of the long bones, caries in the cancellous tissue of bones. The ribs may become carious, but very rarely become necrotic. A sequestrum may, but does not often, form in a vertebral body, in the cancellous head of a long bone, in the carpus, or in the tarsus. If a sequestrum arises from tuberculous osteomyelitis it is seldom found completely detached, but still retains some vascular connection. In tuberculous osteomyelitis of a long bone the sequestrum is wedge shaped with its base toward the joint, and is due to infarction of terminal arteries. A fragment of dead bone is a foreign body; the healthy bone adjacent to it inflames and softens; granulations form, and this line of granulation, like the line of demarcation of gangrene, tends to separate the dead part from the living, the white dead bone being surrounded by the red zone of granulation tissue. A bit of dead bone is

called a *sequestrum*, and Nature tries to cast it off. A superficial sequestrum is known as an *exfoliation*.

Nature's method of casting off a sequestrum is as follows: Suppuration takes place at the line of demarcation, osteitis extends for a considerable distance around this line, the periosteum shares in the inflammation, and new bone forms. A cavity is made within by suppuration, and a box or case is formed without by ossification, the now entirely loosened sequestrum being so encased that it cannot escape. The pus finds its way through the new bone, and there is presented the condition so often seen by the surgeon—namely, a case of new bone known as the *involucrum*, a *cavity* containing pus and the dead fragment or *sequestrum*, and a discharging sinus or *cloaca* (Fig. 251). Nature may eventually cast off the fragment, but the surgeon should not wait for the completion of this slow process.

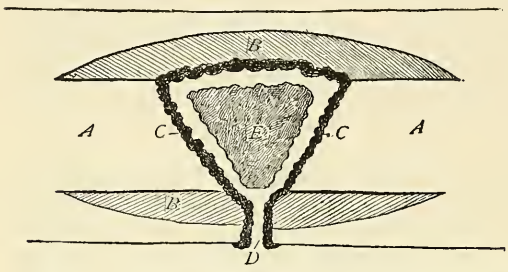



Fig. 251.—Diagram illustrating the formation of a sequestrum: *A, A*, Sound bone; *B, B*, new bone; *C, C*, granulations lining involucrum; *D*, cloaca; *E*, sequestrum.

When a portion of the bone surrounding the medullary canal dies, the condition is called *central necrosis*. In some rare cases necrosis occurs without apparent suppuration, a painless swelling of bone simulating sarcoma. This condition is known as *quiet necrosis*, and has been described by Sir James Paget and Mr. Morrant Baker. Mercury is an occasional cause of necrosis. The fumes of phosphorus may cause necrosis of the lower jaw in those with decayed teeth. Necrosis may be produced also by frost-bites and burns. Many fevers (measles, typhoid, scarlet fever, etc.) are occasionally followed by necrosis. Syphilis and tuberculosis are occasional causes.

The **symptoms** of necrosis are at first those of osteitis or osteomyelitis. The abscess, when formed, opens itself or is opened by the surgeon, and a sinus or sinuses form in the soft parts, as happens in caries. As a matter of fact, were cases of acute osteomyelitis operated on early, extensive necrosis would be rare. If surgeons followed the rule of removing hopelessly damaged bone at the primary operation for osteomyelitis it would seldom be necessary to do extensive operations for dead bone at a later period. When a sequestrum exists a probe introduced into the sinus strikes upon hard bone with a clear, ringing note, and often finds a sinus or sinuses in the bone. In superficial necrosis the discharge is slight and the probe shows the limitations of the disease. In extensive necrosis the discharge is profuse, much new bone forms, several sinuses appear far apart, and the probe must pass through a considerable thickness of new bone before it finds the bit of dead bone. In a chronic case in which there is an involucrum the surgeon may not operate until the dead bone is separated from the living by a line of demarcation unless there is general sepsis. He may wait until the sequestrum is loose unless the patient is being poisoned during the wait. The tendency of thought is against such long delay as was formerly the custom. In youth dead bone loosens quickly, but in old age slowly. An exfoliation becomes loose sooner than the sequestrum of central necrosis. In diffuse periostitis the necrosed shaft loosens quickly. Necrosed portions of the upper extremity loosen more rapidly than those of the lower. In a young adult two or three months will be required to loosen a necrosed fragment in the lower extremity and from six weeks to two months in the upper. A loose sequestrum may be moved by the probe,

and when struck gives a hollow note. In protracted cases of necrosis there is always danger that amyloid disease may arise.

*Quiet necrosis* is a rare condition which has led to some deplorable but pardonable mistakes, because it resembles ossifying sarcoma. It follows injury, particularly fracture. The bone enlarges greatly. There is little or no pain and no fever. The diagnosis can only be made by exploratory incision, and it may even be necessary to remove portions for microscopic study before a conclusion can be reached.

*Postfebrile necrosis* is most usually caused by typhoid fever. The bacilli of typhoid cause chronic osteomyelitis, and this may be followed by necrosis. Scarlet fever, measles, and other febrile processes may also induce necrosis. It is certain that bacilli accumulate in the bones during typhoid fever. They may promptly induce disease; they may remain for long periods apparently inactive and finally pass away; or after a slight strain or injury these organisms may induce bone disease months or even years after the primary infection. *Typhoid bone disease* is often multiple, many bones being involved successively.[1] Not unusually after typhoid fever muscle strain causes periostitis and osteitis, and at such a point necrosis may occur. Either exfoliation or central necrosis may follow typhoid fever. The tibia is involved more often than other bones.

**Treatment.**—An exfoliation should be removed as soon as it becomes loose, the seat of trouble should be touched with pure carbolic acid, and packing of iodoform gauze should be inserted. The treatment of central necrosis comprises free incisions for drainage, removal of the sequestrum and disinfection, antiseptic dressing, frequent cleansing, rest, nourishing food, stimulants, and tonics. When a sequestrum exists the operation of *sequestrectomy* should be performed. The extremity is drained of blood, an Esmarch band is applied, the bone is exposed by a longitudinal incision, the periosteum is reflected on each side, the involucrum is broken through, and the opening is enlarged with the chisel, gouge, and rongeur. The dead bone should be removed by sequestrum forceps, the cavity scraped by a sharp spoon, the lateral edges of the involucrum cut down until the cavity which formerly contained the sequestrum is very shallow, the wound is irrigated with hot salt solution, dried, painted with pure carbolic acid and then with alcohol, again irrigated with salt solution, and firmly packed with iodoform gauze. Remove the Esmarch band, tie the vessels in the soft parts, suture the wound, and apply dressings. It was long the rule of surgery to remove a sequestrum only when loose, and wait if necessary for it to become loose. This rule has been largely abandoned in favor of early operation. The simple removal of a sequestrum—*i. e.*, the operation of *sequestrectomy*—often fails to effect a cure, and even in the most satisfactory cases healing requires a very long time. "The involucrum always contains pyogenic germs that may live in its small foramina and crevices almost indefinitely. For this reason, and on account of the denseness of bony structure, it is well-nigh impossible to disinfect it" (Dr. J. Shelton Horsley, in the "Medical Record," Oct. 20, 1900). Because of the difficulty of curing a case when an involucrum has formed, Dr. Cushing has warmly advocated early operation in osteomyelitis; that is, operation before an involucrum has formed, and when the osteoblasts of the periosteum are extremely active. He points out that if an involucrum has formed, the sequestrum and involucrum should be removed after stripping the periosteum from this region. If the periosteum is found not to be infected, it may be stitched together at the gap where the bone has been removed, so that a periosteal cord exists between the two ends of the bone; and the soft parts above this may be closed. If the periosteum is found to be infected, we agree with Cushing that the cavity should

[1] Keen's "Surgical Complications of Typhoid Fever."

be packed with gauze. The cavity that is left by the removal of a sequestrum and the chiseling of the walls of the involucrum, if large, may be filled by various methods more or less satisfactory. In some cases of widespread necrosis due to diffuse infective osteoperiostitis or to osteomyelitis, extensive resection, or even amputation, may be necessary. If the entire shaft of the tibia requires removal the length of the limb may be maintained by implanting the head of the fibula into the head of the tibia. Otherwise extension must be used during repair.

*Treatment of Bone-cavities.*[1]—Before filling a bone-cavity try to disinfect it. This can be done only relatively. It may be swabbed with pure carbolic acid, followed in one minute by alcohol, or can be mopped with pieces of gauze wet with boiling water. Schede does not pack the bone-cavity, but allows it to fill up with blood-clot after the wound in the soft parts has been closed by sutures. The blood-clot obliterates the dead space in the bone, acts as a support for granulations from the margins, and is slowly eaten by phagocytes. Unfortunately, it is an excellent culture-medium and it often fails of its purpose. Sherman arrests hemorrhage, fills the gap with normal salt solution, and sutures the soft parts without drainage. The surgeon may try to fill the cavity by taking flaps of skin from the sides of the wound, separating them freely from the fascia beneath, and holding them within the bone-cavity by inversion sutures or fastening them to the bottom with tacks (*Neuber's operation*). Another operation consists in breaking the edges of the involucrum and turning them in. Some surgeons insert decalcified bone-chips. The cavity in the bone is made as sterile as possible and is well dusted with iodoform, the bone-chips are dried and inserted into the cavity, a capillary drain is employed, the periosteum is stitched over the opening, and the soft parts are sutured; but if this cannot be done, iodoform packing is used to keep the chips in place. This method we owe to the genius of Senn. Senn's method often fails because of the impossibility of completely sterilizing the walls of the bone-cavity. Attempts have been made to fill bone-cavities as a dentist fills teeth—with gutta-percha, plaster-of-Paris, copper amalgam, etc., but each of these materials acts as a foreign body in the bone (James E. Moore, on "The Treatment of Bone-cavities," "Jour. Amer. Med. Assoc.," May 20, 1905). Schleich uses formalin-gelatin to fill bone-cavities. The difficulty in every case is the impossibility of completely sterilizing the walls of the cavity. Dressman has advised for this purpose the use of boiling oil, but it is apt to cause superficial necrosis. In some cases the cavity has been healed by the insertion of a Thiersch skin-graft. This method has been advocated by J. P. Lord ("Jour. Amer. Med. Assoc.," May 31, 1902). Von Mosetig-Moorhof's method is one of the best ("Zeitschrift für Chirurgie," lxxi, No. 5). He pours into the cavity a melted material which completely fills the cavity, which will not act as a culture-medium or as a foreign body, which is gradually absorbed, and which "possesses the inhibitory and medicinal properties of iodoform without causing iodoform intoxication" (James E. Moore, Loc. cit.). Mosetig-Moorhof's material consists of 60 parts of iodoform, 40 parts of spermaceti, and 40 parts of oil of sesame. These materials are mixed by heating gradually up to 100° C. On cooling, a solid mass is formed. When the surgeon wishes to use it he heats it up to 50° C. and stirs it while heating (Moore), and pours it into the cavity in the bone. On entering the cavity it at once solidifies. A capillary drain is introduced, the periosteum is sutured with catgut, and the other soft parts are sutured with silkworm-gut. Usually union by first intention is secured. Even if the wound gaps the wax is apt to hold. Mosetig-Moorhof has used this material in 4000 cases without ill effect. Cases of poisoning and at least 1 case of death have been reported

[1] The views of Macewen and Murphy on repair of bone will be found on pages 126 and 127.

(Durvergey, in "Presse Médicale," July 1, 1911). Many attempts have been made to fill the defect by *bone-grafting*. The first case of satisfactory transplantation from one of the lower animals with the retention of a vascular attachment was reported by A. W. Morton, in "American Medicine," July 12, 1902. The patient suffered from a compound comminuted fracture of both bones of the right leg. The fracture in the fibula united, but the tibia underwent necrosis, and it was necessary to remove 5 inches of the lower end of the bone. Some days later the periosteum was raised from the ends of the bone and these ends were freshened. The left leg of a dog was amputated just above the tarsus, the bones being sawed so that the ulna was 1 inch longer than the radius. The lower end was partly bared of periosteum, and the ulna of the dog was forced into the cavity of the tibia of the man, and wired to that bone with silver wire. The incision in the man's leg was then sutured, and powerful tendons in each leg of the dog were divided. Each of the dog's other legs was wrapped separately in a plaster-of-Paris bandage, and the entire animal and the leg of the man were then put up in a plaster-of-Paris dressing. Five weeks later the cast was removed, and the bones were sawed and placed in contact with the astragalus. Union took place, and the man was fortunate enough to obtain a useful leg. In some cases a bone defect may be supplied by transference of another bone. Nichols reported 11 cases and insisted on the necessity of preserving the periosteum ("Jour. Amer. Med. Assoc.," Feb. 3, 1904). Huntington has reported a case similar to case No. 2 in Nichols's list. The patient was a boy of seven. A large piece of the entire thickness of the tibia was lost as a result of acute osteomyelitis. There was a gap of 5 inches between the ends of the bone, and the leg was a mere flail. Eight months after the beginning of the osteomyelitis the fibula was sawed opposite the lower end of the upper fragment of the tibia and the upper end of the lower fragment of the fibula was fixed in a cup-shaped depression in the lower end of the upper fragment of the tibia. Six months later union was solid, but in order to improve the weight-bearing power of the limb, nine months after the first operation, the lower end of the upper fragment of fibula was fastened to the upper end of the lower fragment of tibia. The result was excellent. The shortening is only ¾ inch ("Annals of Surgery," Feb., 1905).

**Osteomyelitis.**—By this term we mean inflammation arising in and about the blood-vessels and attacking the marrow of bone. It may attack the soft tissues and cells in the Haversian canals, in the cancellous spaces, or in the medullary cavity. It may be acute or chronic, localized or diffused. Simple osteomyelitis is not due to bacteria. If localized it usually depends upon a traumatism (fracture, contusion, wrench of an epiphysis). Simple diffuse osteomyelitis may arise in a victim of rickets or osteitis deformans. An acute simple inflammation may cause softening and permit bending. A chronic inflammation causes sclerosis.

**Acute infective** or **pyogenic osteomyelitis** is an acute and diffuse inflammation of the bone-marrow due to pyogenic organisms. Infection from staphylococci may be limited to a portion of one bone. Streptococcus infection causes widespread involvement of a bone or of several bones. Acute osteomyelitis may be due to mixed infection with bacilli of typhoid and pyogenic organisms, or bacilli of tubercle and pyogenic organisms, a typhoid process or a tuberculous process serving to establish a point of least resistance. The gonococcus and the pneumococcus occasionally produce acute osteomyelitis. In a case of gonorrheal arthritis in which I resected the wrist-joint cultures of gonococci were obtained from the interior of the bone removed.

It was at one time believed that osteomyelitis was due to a specific organism, but Pasteur proved that micrococci are the cause, and Ogston demonstrated pyogenic bacteria in pus obtained from cases of osteomyelitis. In some cases

there is pure staphylococcus infection (aureus or albus), both aureus and albus may be present, there may be mixed infection with streptococci and staphylococci, streptococci and several sorts of bacilli, or staphylococci and bacilli. Mixed infections with streptococci are more malignant than staphylococcus infections. Most cases of osteomyelitis are due to staphylococci. Trauma is a common predisposing cause. It creates an area of tissue damage which captures bacteria from the blood. Ullman was unable to experimentally induce osteomyelitis without first creating by bone injury a point of least resistance. When he applied a ligature to a rabbit's leg for fourteen hours distinct changes were found to occur in the marrow of the bones. These changes consisted chiefly in extravasation and localized hemorrhages. When the marrow was in this condition, if virus were injected into the animal, osteomyelitis resulted, because the bones presented points of least resistance, vulnerable points in which pus cocci lodged and multiplied.

The pyogenic organisms may gain entrance directly by way of a wound (a gunshot-wound, a compound fracture, an amputation). The causative organisms may reach the bone by way of the blood, having entered the blood originally through the lymphatic system or from a focus of suppuration in the skin, the subcutaneous tissue, or a deeper part. Staphylococcus infection commonly depends on cutaneous suppuration.

Pus organisms may pass into the blood from the tonsils or respiratory organs (Kraske); the intestinal canal (Kocher); the genito-urinary tract; or from excoriations, bruises, small wounds, or suppurations in the skin. Certain fevers strongly predispose to the disease by preparing the soil, as it were, for the growth of pyogenic bacteria. Typhus fever, small-pox, malarial fever, scarlet fever, measles, and diphtheria lessen the vital resistance of bone-marrow. Typhoid fever is not unusually followed by chronic osteomyelitis, due solely to typhoid bacilli. If mixed infection with pus organisms occurs, acute osteomyelitis arises. Vital resistance of marrow is lessened by exhausting diseases, overexertion, unhealthy and, especially, putrid food. We know that various infections produce various reactions in marrow, and in this changed marrow vital resistance is probably lessened or even seriously impaired. Longcope made a study of the marrow in 26 fatal cases of enteric fever, and he invariably found numerous lymphoid cells, phagocytes of large size, and multiple foci of distinct necrosis. The cells whose function is to form blood were noted to be undergoing hyperplasia. In persons dead of perforation and general peritonitis there were numerous foci of necrosis, and also widespread degenerative changes in the blood-making cells and pronounced edema and congestion of the marrow ("A Text-Book of Pathology," by Alfred Stengel). When organisms gain entrance directly by a wound (as in a compound fracture), the endosteum, the medulla, and the cancellous tissue inflame and suppurate, and the entire length and thickness of the bone may be involved. The periosteum becomes infiltrated, detached from the bone, and retracted from the edges of the wound in the bone. The soft tissues around the bone may inflame, suppurate, or slough. More or less necrosis inevitably occurs.

Acute pyogenic osteomyelitis without a wound is often called *acute epiphysitis*. This condition is most common in infants or children of one or two years of age, but is not uncommon in older children (from ten to seventeen years), and even occurs in adults. It is vastly more common among males than females. The tibia is the bone most prone to attack. It is most common during the period of active growth of bone. It is frequently preceded by one of the predisposing causes before mentioned. In some cases a strain or bruise is followed by pyogenic infection, because the damaged tissue extends a hospitable welcome to micro-organisms which are traveling in the body-fluids and pass through the injured area. The most usual antecedent

injury is a twist. As Ollier showed, a twist damages the weakest structure, which is the soft, new bone. In at least half of the cases a history of trauma can be obtained. In some cases there doubtless was trauma, even though we can obtain no history of it. In some cases chilling of the surface of the body is a predisposing cause. In others no predisposing cause is discoverable.

The compact bone suffers secondarily, but is never attacked primarily. New tissue is more susceptible to infection than old tissue, and the disease, as a rule, begins near the epiphyseal line, where new bone is being formed. This point was spoken of by Ollier as "the zone of election of pathological processes." Warren points out that in a growing bone near the epiphyseal cartilage there exists a newly formed spongy tissue, very vascular and connected with the cartilage by a spongy layer of tissue, which is not yet bone, but which does not possess a cartilaginous structure. It is in this portion of the skeleton that the most active changes take place during the period of growth. The medullary substance is very vascular at this point; it is red and

Fig. 252.—Fracture of femur after acute osteomyelitis.

Fig. 253.—Osteomyelitis, showing sequestrum formation.

without fatty tissue. It communicates with the medullary canal and with the periosteum by a number of vascular channels. The epiphyseal cartilage itself is intimately blended with the periosteum. The diaphyseal side of the cartilage produces much more bone than is found in the epiphyseal margin. There is also an active growth of bone in the periosteum, and it is in these regions and in the medullary canal that the inflammatory process originates.[1] The end of the diaphysis is very vascular, but the blood-stream is sluggish because of the large size of the capillary loops ("Practice of Surgery" by Spencer and Gask). The lower end of the femur and the upper end of the tibia are the regions most commonly attacked; but the upper end of the femur and the lower end of the tibia may suffer, and other bones may be attacked, especially

[1] Warren's "Surgical Pathology."

the humerus, radius, ulna, and inferior maxilla. The adjacent joint not unusually becomes involved. Though the inflammation begins in the spongy tissue or medulla, it passes to the canals and spaces of the compact bone. The inflammatory exudate in the canals compresses the vessels and cuts off nutrition from certain areas. Suppuration begins, clots form in the medulla from thrombophlebitis, and the clots in the vessels of the Haversian canals become septic. A small sequestrum forms at the seat of origin of the disease, and the pus about the sequestrum is apt to empty into the medullary canal, causing diffuse osteomyelitis, or into the adjacent joint, causing suppurative inflammation of the articulation.

Marked constitutional symptoms arise from absorption of toxins (suppurative fever), and sometimes true septic infection or even pyemia arises.

Very extensive necrosis may follow osteomyelitis if the patient recovers. Fracture of the bone may occur (Fig. 252). An acute pyogenic osteomyelitis may involve the medulla, may break into the adjacent joint, or may remain localized in the head of a bone and cause an abscess containing fragments of bone or a distinct sequestrum (Fig. 253). The walls of such an abscess are composed of sclerosed bone.

When the medullary canal is involved in a pyogenic inflammation, a part or the whole of the medulla may suffer. The condition may result in central necrosis or in suppurative periostitis and death of the shaft of the bone. The mortality of acute osteomyelitis is high. In the 309 cases collected by Kennedy the mortality was 34 per cent. ("Brit. Med. Jour.," July 20, 1912). The earlier the operation, the less the mortality. Operation within forty-eight hours of the initial chill has a mortality of less than 10 per cent.

**Symptoms.**—Osteomyelitis secondary to a wound of the bone may occur in a person of any age. If a wound exists,—for instance, a compound fracture,—the diagnosis is evident. The constitutional symptoms of septic absorption are positive: there is a profuse, offensive, purulent discharge containing bone-fragments and tissue-sloughs; the periosteum is red, thick, and separated; there are swelling over the bone, great tenderness, and violent boring, gnawing, or aching pain. Osteomyelitis occurring without a wound, the condition often known as acute epiphysitis, occurs in the young, and particularly in children under three years of age.

The symptoms of acute osteomyelitis without a wound of the bone usually come on suddenly and especially at night, and the attack may be so acute as to cause death by systemic poisoning before a diagnosis is arrived at. The disease is generally ushered in by a chill, which is followed by septic febrile temperature. The history will sometimes contain the statement that a blow had been received, that a febrile process had existed, or that the patient had been suddenly chilled after having been overheated (sitting in a draft or in a cellar on a hot day, possibly swimming when very warm, etc.). There is violent aching pain in the bone and acute tenderness near the joint. Within seventy-two hours of the initial chill there will usually be pus in the medullary cavity and a quantity of hopelessly damaged bone if operation has not been performed. The soft parts, which at first are healthy in appearance, after a time discolor, swell, and present distended veins, and may become glossy and edematous because pus is gathered below. The medullary cavity becomes filled with pus. An abscess sometimes reaches the surface and may break spontaneously. The neighboring joint swells, and may become filled with pus; the periosteum and the shaft are involved for a considerable distance; each epiphysis may become affected, the shaft between being comparatively uninvolved, and the epiphyses may separate, displacement and shortening taking place. Extensive necrosis may occur. This disease is often mistaken for rheumatism because of the joint swelling, occasionally for typhoid fever because of

the fever, and in some cases for erysipelas because of the redness of the skin. It gives a very grave prognosis. Sometimes an epiphysitis shows milder symptoms and is slower in progress (subacute). These cases are very often mistaken for rheumatism. But in rheumatism the joint is the part involved from the beginning, while in epiphysitis the joint is involved secondarily after obvious evidence of inflammation well clear of the articulation. Further, the symptoms of rheumatism will be rapidly improved by the use of the alkalis or the salicylates.

**Treatment.**—If osteomyelitis arises secondarily to a wound, apply a tourniquet, sterilize the parts, enlarge the wound, expose and curet the medullary cavity, remove loose fragments of bone, irrigate the medullary cavity with a hot solution of corrosive sublimate or hot salt solution, scrape it with bits of gauze held in the bite of a forceps, paint with pure carbolic acid, pack lightly with iodoform gauze, dress with hot antiseptic fomentations, and secure rest for the parts by splints and bandages. The constitutional treatment is the same as that for septicemia. In some cases amputation is necessary. Acute osteomyelitis without a wound is a most serious condition, rapidly progressive, apt to be quickly fatal, and requiring prompt and radical treatment. Operation should be done as soon as possible after the initial chill. Murphy has insisted upon this for years. Cushing, Le Conte, and others warmly advocate it. I always practice it when possible. Within seventy-two hours, perhaps within forty-eight hours of the initial chill, there will be pus in the medullary cavity and a quantity of bone will be hopelessly damaged. Operation consists in opening the medullary cavity by means of a burr, trephine, or chisel. At such an early stage drainage is all that is necessary, as dead bone has not as yet formed. Very early operation anticipates pus formation. It is not desirable to curet the cavity. A delay of a very few hours will be responsible for pus and dead bone. The medullary cavity must then be freely opened, curetted, and disinfected with pure carbolic acid. The former custom was to pack with iodoform gauze and wait for the formation and loosening of a sequestrum. It is safer and wiser to freely remove dead bone at the primary operation (Le Conte, "Boston Med. and Surg. Jour.," June 1, 1911). In any case if the joint is involved it must be drained. In all cases employ rest, anodynes, strong supporting treatment, and other remedies advised in septicemia. Amputation may be required. In a neglected or prolonged case very extensive necrosis occurs and a formidable operation may be required. Even amputation may be necessary. The entire shaft may have to be removed, a bloody and dangerous operation.

Fig. 254.—Chronic osteomyelitis of the tibia.

**Chronic osteomyelitis** is usually linked with osteitis. Pus may or may not form. There may be only thickening of and pain in the bone. Such

a condition can be caused by attenuated bacteria or by bacteria of ordinary power acting on tissue possessed of a very high vital resistance. It may eventuate in osteosclerosis with filling up of the medullary canal, in limited suppuration, in chronic abscess of the cancellous tissue (Brodie's abscess), or in necrosis. A tuberculous inflammation is one form of chronic osteomyelitis (see page 249). Syphilis, typhoid fever, etc., may cause it, and it can be caused by glanders, leprosy, and actinomycosis.

The typhoid bacillus under certain conditions is pyogenic. Fränkel taught this some years ago, and Keen proves it in his work on "The Surgery of Typhoid Fever." Osteomyelitis due purely to typhoid bacilli is chronic. When the medulla contains typhoid bacilli pus infection is apt to take place, and if such a mixed infection arises acute osteomyelitis develops.

Fig. 255.—Chronic osteomyelitis of the femur.

In chronic osteomyelitis there are pain, tenderness, and swelling, but no marked constitutional symptoms. In some cases the real trouble is not identified until an abscess forms (see Necrosis). There is a form of chronic osteomyelitis which is nearly always mistaken for rheumatism. J. C. Stewart describes it fully ("N. Y. Med. Jour.," March 25, 1911). It is sometimes preceded by a fever, sometimes by a trivial traumatism, but usually comes on insidiously and endures indefinitely as a chronic condition. Stewart shows that the bone enlarges because of subperiosteal production of new spongy bone. The marrow cavity fills with tissue resembling red marrow. The thick bone is tender at points, there is aching pain, and a trivial rise of temperature toward night.

**Treatment.**—If an abscess exists, at once evacuate it by incising the soft parts and chiseling the bone. Do not wait for an involucrum to form, but promptly incise, disinfect, and drain. If dead bone is present it must be removed. In the insidious form, so often mistaken for rheumatism, expose and cut through the new bone formation and open the marrow cavity.

**Osteomalacia, or Mollities Ossium.**—In this disease the bones are partly decalcified, and consequently soften and bend. Masses of new uncalcified bone-tissue are formed. Many bones are usually involved, but the bones of the head are not obviously affected. It is commoner beyond than before middle age, though it may occur in infancy, and a case has been reported in which the disease arose at the age of seventy. It is more frequently met with in women than in men, and pregnancy seems to bear more than a casual relation to its production. The disease is particularly apt to arise when pregnancies are rapidly repeated (Marquis, "L'Obstetrique," June, 1910). In osteomalacia the medulla increases in bulk and becomes more fatty, and the

osseous matter is absorbed gradually, first from the cancellous tissue and then from the compact tissue. Some observers believe that this curious condition is due to lactic acid in the blood, an abnormal amount of acid having been produced and absorbed because of disorder of the primary assimilation. Volkmann maintained that some inflammatory condition disturbs the blood-supply of the medulla, and von Recklinghausen asserted that arterial hyperemia is responsible. Hönnicke suggests that the disease is due to hypersecretion of the thyroid gland.

Fehling, influenced by finding that improvement may follow removal of the ovaries, set forth the view that the disease is due to overaction of the ovaries, causing reflex dilatation of the blood-vessels of bone. The answer to this theory is the fact that the disease can occur in the male. Some have thought that the disease is of bacterial origin. It is most common in those who dwell in damp or dark habitations. It may arise after a soaking and "taking cold." It is a rare disease in England and America, but is much more common in Germany.

The **symptoms** of osteomalacia are as follows: many points of pain which are often thought to be due to rheumatism; deformities from twisting and bending of bone; sometimes lactic acid and occasionally an excess of calcium salts in the urine. There is no fever early in the case, but later there may be a hectic fever. When the disease comes on after childbirth the iliac bones suffer first. Severe pain arises in the pelvis and back and the pain radiates into the thighs, the pain is worse at night, and is greatly aggravated by pressure or movements. Finally, standing and walking become unbearably painful. The earliest pain may be in the sacrum and be felt only when supine. Pains eventually become generalized throughout the skeleton. Pain in the bony walls of the thorax makes respiration difficult. Great deformity occurs because the partly decalcified bones bend. Many of these patients become fat. Fractures occur from very slight force. In the majority of cases the disease is not cured, but grows progressively worse until the patient dies, after many years, from exhaustion. In some cases the process is arrested and the osteoid tissue is calcified.

**Treatment.**—In treating osteomalacia in women insist that pregnancy must not occur. In all cases put braces and supports upon distorted limbs to prevent further bending and fracture. Advise hygienic surroundings and nourishing food, and insist on the value of fresh air. Among the medicines that can be used may be mentioned cod-liver oil, lime salts, extract of the pituitary body, preparations of phosphorus, and bone-marrow. In women the removal of the ovaries sometimes produces great improvement. It has been asserted that the production of anesthesia by means of chloroform may be of benefit.

**Acromegaly.**—In 1886 Marie reported 2 cases with acquired and symmetrical enlargement of the face, hands, and feet. He named the condition acromegaly. This curious disease is, in all probability, due to hypersecretion or perverted secretion of the anterior portion of the pituitary body. There is a hypertrophy or a hyperplasia of this body. As a result of the perverted secretion or accentuated secretory activity there is accelerated growth of the skeleton. It was once thought that tumor was always the cause of acromegaly, but it is now known that whereas tumor may be a cause, many cases occur without tumor. Cases of adiposis dolorosa may show hypophyseal symptoms. Gigantism is probably acromegalic. (For a masterly discussion of this subject see "The Pituitary Body and its Disorders," by Harvey Cushing.) Acromegaly is a disease which causes progressive and often great enlargement of both the bones and soft parts of the extremities, which enlargement is symmetrical. The cranium becomes triangular in shape, with the base below, at the lower jaw.

The lower jaw projects in advance of the upper jaw, the nose becomes prominent and thick, the supra-orbital ridges are accentuated (Fig. 256), and the costal cartilages and inner ends of the clavicles become protuberant. Later the larynx, ribs, shoulder-blades, and vertebræ become involved, and the back becomes markedly humped (cervicodorsal hump). The hands and feet are affected in advanced cases. As a rule, the thyroid gland is enlarged, and a postmortem examination may detect a diseased pituitary gland. Severe and uncontrollable headache is sometimes a distressing feature of the disease. In some cases there is marked somnolence. A fireman who suffered from acromegaly would go to sleep almost the moment he sat down. Early there is low assimilation of carbohydrates, often with glycosuria. The disease is not regularly progressive, but exhibits periods of rapid increase, a stationary condition, or perhaps retrogression. In a prolonged case signs of pituitary insufficiency are noted, viz.: such high assimilation of carbohydrates that it is difficult or impossible to cause alimentary glycosuria, adiposity, subnormal temperature, and atrophy of the sexual organs. In all suspected cases an *x*-ray picture should be taken. If a tumor is present or if the gland is hypertophied the plate will show an enlarged sella turcica. A tumor causes rapid blindness. The disease slowly but surely causes death. Medical treatment is of no avail in cases of pituitary overactivity (*hyperpituitarism*). If there is evidence of hypophyseal insufficiency (*hypopituitarism*) administer hypophyseal extract.

Fig. 256.—Face in acromegaly. Note enlarged superciliary ridge, thickened lips, massive jaw, and general grossness. (Church and Peterson).

If there is hyperpituitarism it is justifiable to partially extirpate the gland, on the same principle as we partially extirpate the thyroid in a case of exophthalmic goiter. In Cushing's case the operation of partial extirpation of the pituitary body relieved subjective discomforts and modified acromegalic conditions. If there are evidences of tumor of the hypophysis it is justifiable to undertake its removal by operation. This has been accomplished successfully by Hochenegg, Von Eiselsberg, Brochardt, Cushing, Sir Victor Horsley, and others. In the cases of Hochenegg and Cushing not only were pressure symptoms relieved, but acromegalic conditions were greatly benefited.

**Leontiasis Ossea or Hyperostosis Cranii** (*Virchow's Disease*).—This is a symmetrical hypertrophy limited to the facial and cranial bones, and which begins, as a rule, in the superior maxillæ. The hypertrophy progressively increases, causes difficulty of mastication, and is accompanied by headache. It produces distinct deformity of the jaw like a tumor, whereas acromegaly enlarges all of the proportions of a bone (Fig. 257). It may produce blindness, new bone pressing upon the optic nerves. Treatment is very unsatisfactory. Horsley has obtained some degree of amelioration by operating and removing masses of bone.

**Osteitis Deformans** (*Paget's Disease*) (Fig. 258).—This disease was first described by Paget in 1877. Higbee and Ellis state that up to January, 1911, 158 cases have been reported ("Jour. Med. Research," vol. xxiv, No. 1). Packard and Steel were of the opinion that many reported cases were not genuine instances of the disease, some being ordinary osseous tumors, others being cases of enlargement after fracture, and still others being instances of mollities ossium ("Amer. Jour. Med. Sciences," Nov., 1901). Many of the reports are purely

clinical. The disease does not appear to be hereditary. It is usually associated with widespread arterial sclerosis. There is no evidence that gout, rheumatism, or disease of the nervous system play any part in causation. Some consider the condition parasyphilitic, but there is no real evidence of such origin. It has been asserted that heat or cold may be causal and that the condition has begun after injury of a long bone. A possible cause is disturbance of some internal secretion. The enlarged thyroid in Askanazy's case led him to suggest the theory. No parathyroids were found at autopsy in the patient of Higbee and Ellis. These two observers suggest that Paget's disease may be due to absence of parathyroid secretion and the consequent formation of substances which abstract calcium from the bones. In this disease great quantities of new bone are formed, but calcification does not occur. The material undergoes absorption, and the medullary substance of the bone becomes extremely vascular and filled with white blood-cells, and also with giant-cells. The bones lengthen and thicken. The long bones bend

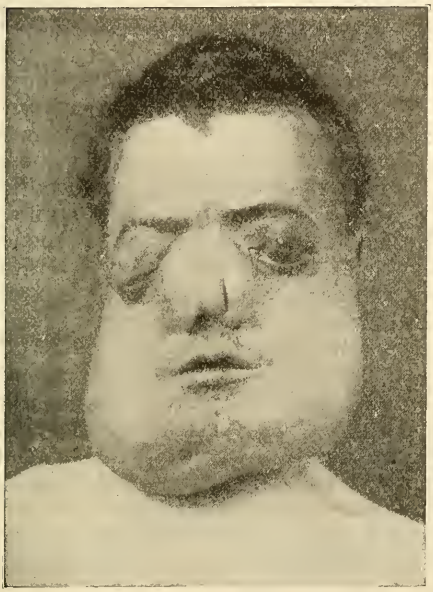


Fig. 257.—Leontiasis ossium.

Fig. 258.—Paget's disease.

and the bones of the skull bulge. The bending of the long bones has been believed to depend upon the weight of the body acting on uncalcified new bone, but fracture is not particularly apt to occur. It is now maintained that the bending and bulging are active processes, "since the bending is always well marked in those bones which are fixed at both ends by muscles and ligamentous attachments, *e. g.*, the clavicle, radius, and tibia, the accompanying bone (fibula or ulna) acting as the string to the bow" (Gruner, Scrimger, and Foster, in "Archives of Internal Medicine," June, 1912). It is extremely rare before the age of forty, and usually begins between forty and fifty or later. The enlargement of the bones is usually accompanied by pain which may be severe. Enlargement may be first detected in the cranium, but is more often first seen in some other bone—for instance, the clavicle, the tibia, the spine, or the radius. The tibia in most cases suffers first, and other bones become involved later. In

fact, in some cases the bones of the head do not enlarge at all; but, taking all the reported cases, the skull is affected more frequently than any of the other bones. In some cases the enlargement of the bones seems to be symmetrical; in others it is not. In the disease known as leontiasis ossea the chief enlargement is manifested in the face; in Paget's disease there is no enlargement of the bones of the face, or else these bones are trivially involved. Packard and Steele point out that the diagnosis is extremely difficult when but a single bone is involved; but that if two or more bones are involved, we should think of Paget's disease as the condition, especially if we are able to exclude syphilis, cancer, and sarcoma. In mollities ossium the head is not involved at all, and there is not nearly so much thickening of the bone. The two authors before quoted show that in acromegaly the cranium is a triangle with its base below at the lower jaw, the orbital arches being distinctly involved, but that in Paget's disease the involvement is chiefly of the calvarium. The patient actually diminishes in height. The chest becomes deformed. There is angular curvature in the dorsocervical region. The lower extremities are usually bent, and the pelvis, as a general thing, is broadened. Paget says: "The most characteristic features are the loss in height, indicated by the low position of the hands, the stooping with round shoulders, the head held forward with the chin raised and the chest sunken toward the pelvis, the abdomen pendulous, the curved lower limbs held apart and usually with one advanced in front of the other and both with knees slightly bent, the ankles overhung by the legs, and the toes turned out. The enlarged cranium, square looking and bossed, may add distinctness to these characteristics, and they are completed in the slow and awkward gait of the patients." In some of the cases there is a tendency to tumor formation. In the 67 cases collected by Packard and Steele, 3 suffered from cancer and 5 from sarcoma. In the 158 cases collected by Higbee and Ellis ("Jour. Med. Research," vol. xxiv, No. 1) there were 14 instances of tumor growth. In a case reported by Gruner, Scrimger, and Foster ("Archives of Internal Medicine," June, 1912) sarcoma appeared in the radius, head of the humerus, and other places. Some cases are associated with goiter. It has been suggested that there is a bacterial cause for Paget's disease. An Italian investigator claimed to have discovered a bacterium and made a serum for use in treatment. In 2 cases I exposed areas of new bone, removed portions, and took cultures. One set of cultures remained sterile. A tube of the other was found contaminated by the skin staphylococcus.

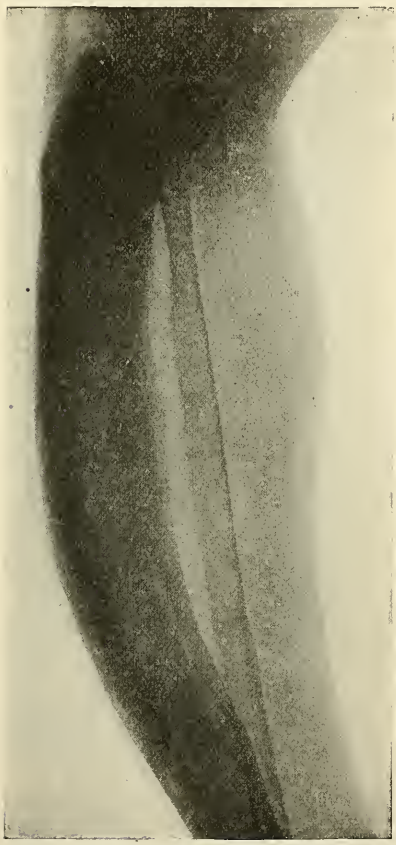


Fig. 259.—Paget's disease.

Treatment is practically useless. No known remedy diminishes the size of the bones, although iodid of potassium is said occasionally to mitigate pain.

**Osteo-arthropathie Hypertrophiante Pneumique** (*Marie's Disease*).—(See page 644.)

**Multiple Myeloma.**—By this term we mean a new growth in the bone-marrow which occurs particularly in the ribs, sternum, and vertebræ. It may occur also in the bones of the cranium and in other bones. The multiplicity is not due to metastasis, but the growths start as separate foci. The nature of the cells in the growths is uncertain. They almost certainly spring from marrow cells. Some consider them myelocytes, some bone-marrow plasma cells. The effect of the growth is to thin the bones and make them very brittle. This condition was first described by Rustitzky in 1873. Stumm, who reports 2 cases, estimates that about 50 cases have been reported.

The condition begins insidiously, in either sex, during middle age, sometimes in advanced life. It begins usually with attacks of aching in the limbs and weakness. Such attacks may come and go over a considerable period of time. Eventually the back, chest, and ribs become the seats of pain and tenderness. Finally, the pain becomes constant and the patient is confined to bed. In many cases a sternal tumor can be palpated. Spontaneous fractures are apt to occur. Usually the Bence-Jones body is found in the urine. Death is due usually to exhaustion. Treatment is futile.

## Fractures

**Definition.**—A fracture is a solution, by sudden force, of the continuity of a bone or of a cartilage. Clinically, under this head are placed epiphyseal separations and the tearing apart of ribs and their cartilages.

**Varieties of Fractures.**—The varieties of fractures are as follows:

*Simple fracture* is a subcutaneous fracture, or one in which there is no wound extending from the surface to the seat of bone injury. This corresponds to a contusion of the soft parts.

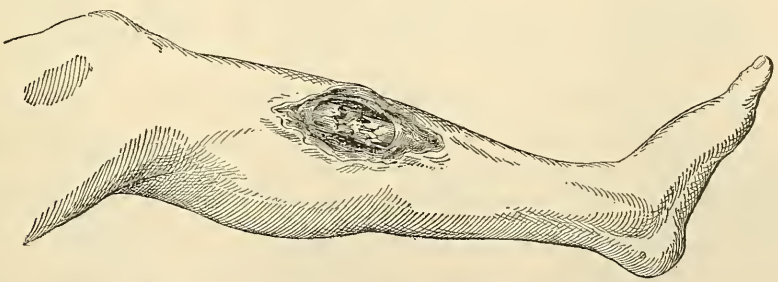


Fig. 260.—Compound comminuted fracture of the tibia.

*Compound fracture* (Figs. 260 and 270) is an open fracture, or one in which an open wound extends from the surface to the seat of bone injury or in which a wound opens up a passage from the fracture to the surface. This corresponds to a contused or lacerated wound of the soft parts. The opening may be through the skin; through a mucous membrane, as in some fractures of the base of the skull and pelvis; through the drum of the ear, as in some fractures of the middle fossa of the base of the skull; through the lung, as when a broken rib penetrates that organ; or through the bowel or bladder, as in some fractures of the pelvis.

A *primary compound fracture* is one in which the breach in the soft parts is produced at the time of the accident, either by the direct violence of the injury or by the forcing of a bone or bones through the tissues.

A *secondary compound fracture* is one in which the breach in the soft parts occurs after the accident, either from sloughing of damaged tissues, from ulceration because of the pressure of ill-adjusted fragments, or from the forcing of a bone or bones through the soft parts because of rough handling, neglect, or the tossing of delirium.

*Complicated fracture* is a fracture plus the complication of a joint injury, arterial or venous damage, or injury to the nerves or soft parts. When a fractured rib injures the lung or when a broken vertebra damages the cord a complicated fracture exists. The term is unfortunate, as it conveys no definite meaning, and its use is no more justifiable than it would be to speak of "complicated pneumonia" or "complicated typhoid," for the complication should be named in any case. It must be remembered that damage to the soft parts not sufficiently severe to produce a wound reaching from the surface to the seat of fracture does not make the case a compound fracture, but rather complicates a simple fracture. Remember also that even superficial areas of tissue destruction must be treated antiseptically, otherwise absorption of pyogenic bacteria and their deposition at the seat of injury may cause diffuse osteomyelitis.

*Complete fracture* is that which extends through the whole thickness of a bone or entirely across it (Fig. 261).

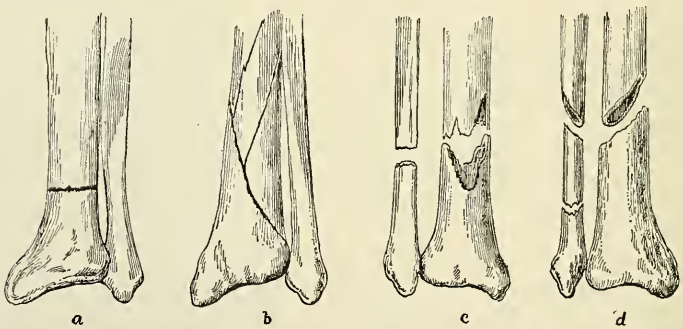



Fig. 261.—Complete fractures: *a*, Transverse; *b*, spiral; *c*, dentated; *d*, oblique or multiple.

*Incomplete fracture* is that which extends only partially through the thickness of a bone or only partially across it (Fig. 262).

A *linear, hair, capillary*, or *fissured fracture*, or a *fissure*, is a crack in a bone with very little separation of the edges. This is an incomplete fracture, but may be associated with a complete break (Fig. 261, *b*).

A *green-stick, hickory-stick, willow*, or *bent fracture* is a true incomplete break (Fig. 262). The bones most frequently so broken are the radius, ulna, clavicle, and ribs. It arises from indirect force, and it is very rare after the age of sixteen. In rickets green-stick fractures are very common. It is called "green-stick" because the bone breaks like a green stick when forced across the knee, first bending and then breaking on its convex surface. The bone, being compressed between two forces, bends, and the fibers on the outer side of the curve are pulled apart, while those on the concavity are not broken, but are compressed. In correcting the deformity such fractures are often made complete. The permanent bending of a bone without a break may possibly occur in youth. In children a portion of a bone of the skull may be bent inward, causing depression. In some cases such a depression is permanent; in others it is temporary, the bone returning to its proper level.

*Depression-fracture* occurs when a portion of the thickness of a bone is driven in by crushing. Fracture by depression is a result of the bending in

of a bone (as the parietal), a fragment breaking off from the side toward which the bone is bending. A *depressed fracture* is complete, not incomplete, and by this term is meant an injury in which a fragment of the entire thickness of the bone is driven below the level of the surrounding surface.

*Splinter- and Strain-fracture.*—The breaking off of a splinter of bone (splinter-fracture) or of an apophysis constitutes a form of incomplete fracture. A strain upon a ligament or a tendon may tear off a shell of bone, and this injury is the "strain-fracture" or "sprain-fracture" of Callender.

*Longitudinal fracture* is a fracture whose line is for a considerable distance parallel, or nearly so, with the long axis of the bone. Such fractures are common in gunshot injuries (Fig. 263).

*Oblique fracture* is a fracture the direction of which is positively oblique to the long axis of the bone. Most fractures from indirect force are oblique (Fig. 261, *d*).

*Transverse fracture* is a fracture the direction of which is nearly transverse to the long axis of the bone (no fracture is mathematically transverse) (Fig. 261, *a*). The cause is often, but not invariably, direct force. The *fracture en rave* (radish-fracture, so called because the bone breaks as does a radish) is transverse at the surface, but not within.

Fig. 262.—Green-stick fracture.

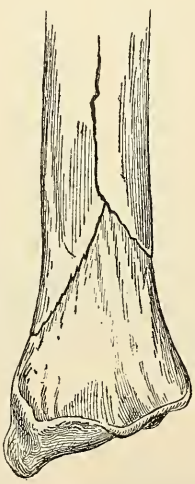


Fig. 263.—Longitudinal and oblique fracture.

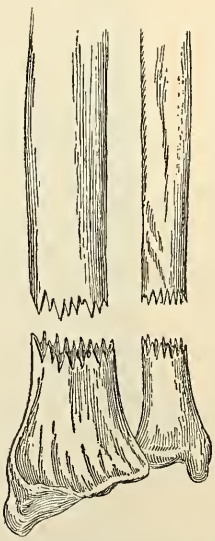


Fig. 264.—Appearances of the ends of fragments.

*Toothed* or *dentate fracture* is a form of fracture in which the end of each fragment is irregularly serrated and the fragments are commonly locked together; hence it is difficult to correct the deformity (Fig. 261, *c*, and Fig. 264). Most simple fractures from direct force are serrated.

*Wedge-shaped, V-shaped, cuneated,* or *cuneiform fracture* ("fracture oblique spiroïde," "fracture en V" of Gosselin, "fracture en coin") is one the lines of which take the shape of a V, which may be entire or may lack the point. It occurs at the articular extremity of a long bone, and a fissure usually arises from its point and enters the joint. If complete, it is a "comminuted fracture."

T-*shaped fracture* is a fracture which presents a transverse or oblique line and also a longitudinal or vertical line. It occurs at the lower end of either the

humerus or femur, the transverse line being above, and the vertical line (intercondyloid) between, the condyles. If complete, it is, in reality, a form of comminuted fracture.

*Multiple* or *composite fracture* is a condition in which a bone is broken into more than two pieces, the lines of fracture not intercommunicating, or a condition in which two or more bones are broken. Multiple fractures of one bone are divided into double, treble, quadruple, etc. Multiple fractures involving more than one bone are seldom seen, and represent less than 2 per cent. of fracture cases. The reason of their rarity in hospitals is that they result from severe force and many of the victims die before they can be brought to an institution. The mortality in cases which reach the hospital is large, over 27 per cent. (Astley P. C. Ashhurst, in "Annals of Surgery," August, 1907).

*Comminuted fracture* is a condition in which a bone is broken into more than two pieces, the lines of fracture intercommunicating (Figs. 265 and 266). The bone may be broken into many small fragments, there may be much splintering, or the osseous matter may actually be ground up.

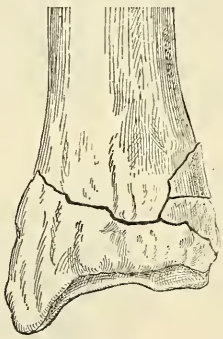


Fig. 265.—Comminuted fracture of the lower extremity of radius.

Fig. 266.—Comminuted fracture of the upper part of femur.

*Impacted fracture* is one in which one fragment is driven into the other and solidly wedged (Figs. 267, 268, and 269).

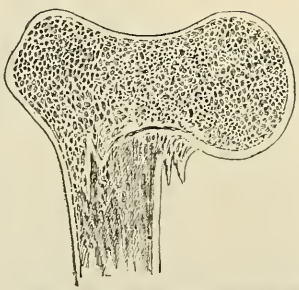


Fig. 267.—Impacted fracture of the neck of the femur.

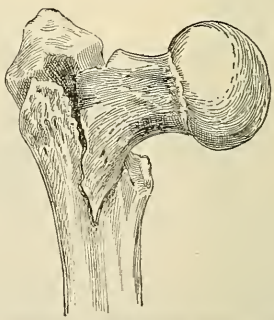


Fig. 268.—Impacted fracture of the neck of the femur.

*Fracture with crushing* or *penetration* is a fracture in which one bone is driven into the other, the encasing bone being so splintered that the impacting bone is not firmly held.

*Pathological, spontaneous,* or *secondary fracture* is one occurring from a very insignificant force acting on a bone rendered brittle by disease.

*Ununited fracture* is a fracture in which bony union is absent long after the passage of the period normally necessary for its occurrence.

*Direct fracture* is one occurring at the point at which the force was primarily applied.

*Indirect fracture* is one occurring at a point distant from the area of primary application of force.

*Stellate* or *starred fracture* (fracture par irradiation) is one in which several fissures radiate from a center. If the fracture be complete, the condition is, in reality, a form of comminuted fracture.

*Helicoid, spiral,* or *torsion fracture* is a fracture resulting in a long bone from twisting.

*Fracture by contrecoup* is a fracture of the skull which is on the opposite side of the head to that which was the recipient of the force.

*Epiphyseal Separation or Diastasis.*—This injury occurs only before the age of twenty-five. In order of frequency, the bones chiefly subject to epiphyseal separation are: the upper end of the humerus, the lower end of the radius, the lower end of the femur, and the lower end of the tibia (John Poland, in the "Practitioner," Sept., 1901). This injury induces deformity, which is often

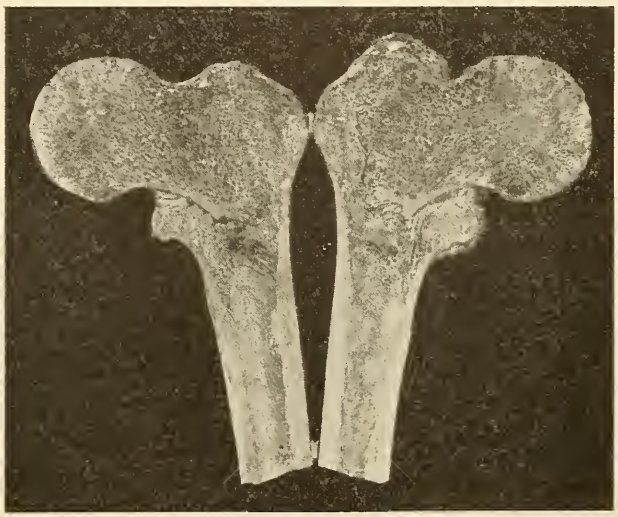


Fig. 269.—Impacted fracture of neck of femur (Conner).

difficult to reduce, and by damaging the cartilage may retard or inhibit a further lengthening of the limb by growth. Occasionally after damage to an epiphysis suppuration will occur, sometimes thickening takes place. Non-union is very rare. After a sprain of an epiphysis tuberculous disease sometimes develops, but very rarely after a separation.

*Intra-uterine fractures* are usually due to injuries of the mother's abdomen sustained toward the end of pregnancy. Some hold that they can arise as a consequence of the force of violent uterine contractions. Many so-called "intra-uterine" fractures are wrongly named, as they result from injury during delivery. In sporadic cretinism the bones are fragile and ill-ossified, and many fractures may occur *in utero.*

*Designation According to Seat of Fracture.*—A fracture may be designated according to its anatomical seat; for instance, fracture of the upper third of the shaft of the femur, fracture of the olecranon process of the ulna, fracture of the middle third of the clavicle, and fracture of the body of the lower jaw. *Intra-articular* fracture is one within or extending into a joint; *intracapsular* fracture

is one within the capsule of either the shoulder- or hip-joint; and *extracapsular* fracture is one just without the capsule of either the shoulder- or hip-joint.

**Causes of Fracture.**—The causes of fracture are: (1) exciting, immediate or direct, and (2) predisposing or indirect.

**Exciting causes** are: (*a*) external violence and (*b*) muscular action.

*External violence* is the most usual exciting cause. Two forms are noted: (1) direct violence and (2) indirect force.

*Fractures from direct violence* occur at the point struck, as when the nasal bones are broken with the fist. In such fractures the soft parts are injured; they may be destroyed at once in part, they may be damaged so severely that a portion sloughs, or they may be damaged so slightly that they do not lose vitality; hence fractures by direct violence may be compound from the start, may become so, or may remain simple. In fractures by direct force discoloration, due to effused blood, usually appears at the point struck soon after the accident. In compound fractures by direct violence the soft-part injury is so great that primary tissue union cannot occur.

*Fractures from indirect force* do not occur at the point of application of the force, but at a distance from it, the force being transmitted through a bone or a chain of bones, as when the clavicle is broken by a fall upon the extended hand. Such fractures tend to occur in regions of special predilection. If they are not compound, there is no injury of the surface over the fracture. If they become compound by projection of fragments, primary union may still occur. Discoloration over the seat of fracture is usually not present soon after the accident, but may occur later. Discoloration rapidly appears in soft parts at the point where the force was first applied.

*Muscular action* is rather an unusual cause except in the patella. Fractures thus produced result from sudden or violent muscular contraction. Bones so broken are usually diseased. Violent coughing may fracture the ribs; attempting to kick may fracture the femur; saving one's self from falling backward may fracture the patella; throwing a stone may fracture the humerus; and sudden extension of the forearm may fracture the olecranon process of the ulna.

**Predisposing Causes.**—There are two classes of predisposing causes, namely: (1) physiological, natural or normal, and (2) pathological or abnormal.

*Natural Predisposing Causes.*—Under this head is considered the liability to fracture possessed by individual bones because of their shape, structure, function, or position. Those predispositions occasioned by special ages are also considered. In youth epiphyseal separation is commoner than fracture and a fracture is apt to be incomplete. Fractures are commonest between the ages of twenty-five and sixty. From two to four years of age a child is more liable to fracture than later, because he is then learning to walk (Malgaigne). The bones of the old are easily broken, but the normal lack of activity of the aged saves them from more frequent injury. Thus, the predispositions of age are in part due to habits and in part to bony structure. The bones of the young, being elastic, bend considerably before they break; the bones of the old, being brittle and inelastic, break easily, but do not bend. In old age the bones become lighter and more porous, though they do not diminish in size. Absorption takes place from the interior of a bone, particularly at its articular head, the medullary canal increases in size, the cancellous spaces become notably larger, and portions of the remaining bone of the interior show a fatty change. There is no increase in the amount of mineral salts present, as was long taught. These alterations occur earlier in women than in men.[1] The change of age is a diminution in the amount of bone present, and sometimes a fatty change in a portion of what remains. If the atrophy of bone is other than that normal to senility, it constitutes a pathological predisposing

[1] Humphrey on "Old Age."

cause of fracture. Normal predisposing causes include the person's weight (which determines the force of a fall), muscular development, habits, sex, occupation, and the season of the year.

*Pathological Predisposing Causes.*—*Hereditary fragility*, a form of *fragilitas ossium*, is a condition commonest among women, often existing in generation after generation, and in this condition fractures occur from a very slight force. There exists in these cases bony rarefaction—in fact, a premature senility. *Fragilitas ossium* (*osteopsathyrosis*) may be congenital or may come on later in life. It may result from senility, wasting diseases, scurvy, scarlatina, bone-cyst, malignant disease of the bone, certain nervous disorders, rickets, osteomalacia, and atrophy due to disuse.

*Nervous Diseases.*—Bony nutrition is dependent on the spinal cord, and the trophic influence is probably exerted through the posterior nerve-roots. In diseases of the anterior cornua bony growth is much interfered with; in diseases of the posterior columns, as in locomotor ataxia, a true bony atrophy bespeaks trophic disorder. Syringomyelia causes brittleness of the osseous structures, and in paralysis agitans bones are thought to break easily. Trophic changes may occur in the bones of the insane, most commonly when insanity is linked to organic disease. About one-quarter of paretic dements show undue brittleness or unnatural softness of bones.[1] The bones of maniacs are frequently fragile. Fractures among the insane are not necessarily an indication of abuse.

*Rickets* predisposes to fracture because of altered bone structure and the great liability to falls.

*Osteomalacia* predisposes to fracture of the long bones, sternum, and ribs.

*Atrophy of Bone.*—This condition, as has been stated (see page 518), is normal in senility. It may arise from want of use, as is observed in the bedfast, in the wasted femur of hip-joint disease, and in the bones of a stump. It may arise from pressure, as when an aneurysm compresses the ribs, sternum, or vertebræ. Among other of the pathological predisposing causes are to be mentioned cancer, sarcoma, hydatid and solitary cysts of bone, caries, necrosis, gout, scrofula, syphilis, mollities ossium, and scurvy.

**Symptoms of Fractures.**—*History of An Injury.*—In spontaneous fracture there may be no record of violence; for instance, a bone may break while an individual is turning in bed. In investigating the history, not only seek for a record or for evidences of violence, but try to determine exactly how the accident happened.

A *sound of cracking* is occasionally audible to a bystander at the time of the injury. The patient may have heard it, but very rarely does. A rupture of a tendon or a ligament produces a similar sound.

*Pain* is usually, but not invariably, present (absent often in rickets). In some fractures the pain is slight, in some there is no pain, in others pain is torturing, and in most it is severe for a time after the injury, but gradually abates unless reinduced by movement. Pain developed at the time of the accident is far less important as a symptom than that which can subsequently be produced by movement. In indirect fracture there is an area of pain at the point of application of the force, and another at the seat of fracture. Pain at the seat of fracture can be greatly aggravated by pressure or movement and is rather narrowly localized.

*Deformity* or *alteration in length or outline* is due in part to swelling and in part to a change in the mutual relation of the fragments (displacement). The deformity due to swelling is no aid to diagnosis, as the same condition occurs in contusion, and often hides some positive symptomatic distortion. The swelling is due first to blood and next to inflammatory products and

[1] "Manual of Insanity," by Spitzka.

pressure-edema, and is very great in joint fractures. Swelling due to bleeding is early and rapid. Swelling due to inflammatory exudation is later and gradual. Swelling due to pressure-edema may be rapid. The greater part of the swelling is due to hemorrhage and exudation from the damaged soft parts, a portion of it is due to hemorrhage and exudation from the bone. The swelling is usually in direct ratio to the mobility, the greater the mobility, the greater the swelling. "The swelling in fractures of the skull is inconsiderable, notwithstanding that the total area of bone surface involved is commonly more than in fractures of the leg or arm, and the vascularity greater. The reason for this is the natural immobility of fractures of the skull" (James P. Warbasse, in "Jour. Amer. Med. Assoc.," March 13, 1909). The deformity of displacement may be produced by the violence of the injury (as is the depression in a skull fracture), by the weight of an extremity (as is the falling of the shoulder in a fracture of the clavicle), or by muscular action (as is the pulling upward of the fragment of a fractured olecranon process).

The **varieties of displacement** are: (1) *transverse* or *lateral*, where one fragment goes to the side, front, or back, but does not overlap the other; (2) *angular*, the bony axis at the point of fracture being altered and the fragments forming with each other an angle; (3) *rotary*, one fragment rotating in the bony circumference, the other remaining stationary. As a rule, it is the lower fragment which turns on its long axis, the limb below the level of the break rotating with it; (4) *overlapping* or *overriding*, when the upper level of one fragment is above the lower level of the other fragment. It is usually the lower fragment which is drawn by the muscles above the upper, but in a fracture of the lower extremity the body-weight and sliding down in bed may push the upper below the lower fragment. In overriding the ends are near together and the bones are usually in contact at their periphery. It is obvious that overlapping is associated with transverse displacement, as one fragment must go front, back, or to the side; (5) *penetration* or *impaction* when one fragment is driven into the other, thus producing shortening; (6) *separation of the two fragments* occurs in fracture of the patella, olecranon, os calcis, certain articulations, and in some breaks of the humerus when the arm is not supported.

It is important to remember that a dislocation as well as a fracture may produce displacement, but these two conditions may be differentiated by the observation that the displacement of fracture tends to reappear even after complete reduction, while the displacement of dislocation does not reappear after correction. A displacement is difficult of detection in a flat bone and when one of two parallel bones is broken.

*Loss of function* may be shown by inability to move the limb because of the break, but it is not always markedly present, though some degree invariably exists. It is slight in "green-stick" and impacted fractures (unless the loss of power arises from pain or nerve injury). A person can walk when the fibula alone is broken, and likewise in some cases of intracapsular fracture of the femur, and can often put the hand on the head in fractured clavicle (Malgaigne). The pain of an injury or the loss of power from nerve traumatism may cause loss of movement in the limb. This symptom is of slight diagnostic value in most fractures.

*Extravasation of Blood.*—A contusion of the surface accompanied by skin abrasion indicates merely the point of application of direct external violence. If contusion is extensive over a superficial bone, as the tibia or parietal, after a few hours it often stimulates fracture by presenting a soft, compressible center surrounded by a ring of hard, condensed tissues and coagulated blood. Direct external violence may merely occasion ecchymosis, and in fracture from indirect force ecchymosis may occur throughout a considerable area.

In regard to this symptom, note that even great external violence may occasion no evident contusion or ecchymosis, and in any fracture this symptom may be present or absent. In old people, anemic subjects, fat individuals, alcoholics, and opium-eaters extravasation of blood is frequently marked and persistent. By suggillation is meant an extravasation of blood which slowly invades wide areas of tissue and which appears at the surface only after some time, and then usually as a yellowish discoloration, red hemoglobin having been changed to yellow hematoidin. Linear ecchymosis has been esteemed by some as a sign of fissure, and it is often noted after fracture of the fibula. Linear ecchymosis over the line of the posterior auricular artery was shown by Battle to be a valuable sign of fracture of the posterior fossa of the base of the cranium.

*Preternatural mobility* is a most important symptom, which is pathognomonic when surely found. The unbroken bone is nowhere mobile in continuity. By preternatural mobility is meant that a bone is mobile in continuity or that there is abnormality in the direction or extent of joint mobility. In some fractures this symptom does not exist (impacted, green-stick, and locked serrated fractures); in others it cannot be found (fractures of tarsus, carpus, vertebral bodies); in others it is difficult to obtain, but at times can be developed (fractures near or into many joints). To develop this symptom try, when the case admits, to grasp the fragments and to move them in opposite directions. In a fracture of the shaft of the femur or humerus fix the upper fragment and carry the knee or elbow in various directions to develop bending at the point of fracture. In fracture of the clavicle push the shoulder downward and inward. In fractures of either bone of the forearm grasp the parallel bone with four fingers of each hand and make pressure on the suspected bone alternately with either thumb, and the same procedure can be used in fractures of the leg. In fracture of the neck of the femur the altered rotation-arc of the great trochanter demonstrates preternatural mobility (Desault). In fracture of the lower end of the radius bend the hand back, and in a break of the lower end of the fibula evert the foot (Maisonneuve). In seeking preternatural mobility remember that the elastic ribs when forced in give a sense of bending, and that the fibula at its middle is "normally flexible" (Dupuytren). Some rachitic bones may be bent.

*Crepitus* or *crepitation* is both a sensation and a sound, which indicates the grating together of the two rough surfaces of a broken bone. This symptom is of great value, but it is not always present. It is absent in locked serrated fractures, in impacted fractures, in cases where the broken ends cannot be approximated (as in overlapping), is rare when a fractured surface is against the side, and not the broken face, of the other fragment, and is unusual in incomplete fractures. Crepitus is often absent in epiphyseal separation, in softened bones, and in fractures in or near joints, and it may be prevented from occurring by blood-clot, fascia, synovial membrane, periosteum, or muscle between the broken surfaces. The grating found in tenosynovitis must not be mistaken for the crepitus of fracture; the former is diffuse, large, soft, and moist; the latter is limited, small, harsh, and dry. The clicking of an inflamed or eroded joint and the crackling of emphysema must also be separated from bony crepitus. Crepitus of fracture may be present at one moment, but absent the next. It is often not detected during the time swelling is marked, and cannot be discovered after organization of the callus begins. In but few fractures is it needful to try to hear crepitus with the unaided ear or with a stethoscope upon the part, but in doubtful cases of fractures of ribs and joints this evidence should be sought for.

The above-named symptoms are known as "direct." There are other symptoms known as "circumstantial," such as the flow of blood and cere-

brospinal fluid from the ear after some fractures of the middle fossa of the base of the skull; emphysema of the face and epistaxis after fracture of the nasal bones; hemoptysis and emphysema after crushes of the chest; discoloration following the line of the posterior auricular artery after fracture of the posterior fossa of the skull, and subconjunctival ecchymosis after fracture of the anterior fossa of the base of the skull.

**Diagnosis.**—Examine as soon as practicable after the injury—before the onset of swelling, if possible. Expose the part completely, taking off the clothing, if necessary, by clipping it along the seams. Attentively scrutinize the part and compare it with the corresponding part on the opposite side. If any deformity be present, it must be ascertained that it did not exist before the accident. If the nature of the injury be uncertain, if the patient be very nervous, or if the part be acutely painful, it is better to give ether to diagnosticate, set, and dress. In injuries of the elbow-joint anesthetize before examination, unless an *x*-ray apparatus is accessible to settle the diagnosis, and even then it is usually well to anesthetize in order to facilitate reduction and dressing. In every case of suspected fracture get an *x*-ray picture if possible. A correct diagnosis is of the first importance and on a correct diagnosis proper treatment primarily depends.

A fracture is distinguished from a dislocation by its preternatural mobility, its easily reduced but recurring displacement, and its crepitus, as contrasted with the preternatural rigidity, the deformity, difficulty to reduce but remaining reduced, and the absence of crepitus of a dislocation. Further, in dislocation the bone, when rotated, moves as one piece, whereas in fracture it does not so move; in dislocation the bony processes are felt occupying their proper relations to the rest of the same bone, while in fracture some of them present altered relations. In dislocation the head of the bone is found out of its socket, but in fracture it is felt in place. It is important to remember, moreover, that a fracture and a dislocation may occur together, and that the rubbing of a dislocated bone against an articular edge, when the joint has been roughened by inflammation, simulates crepitus.

Great contusion, by inducing extreme tumefaction, may mask characteristic deformity and obscure crepitus. When only a contusion exists, pain is apt to be widespread; but if a fracture has occurred, the pain is accentuated at some narrow spot. In many cases, before he can give a certain opinion, the surgeon must wait some days until the swelling has largely subsided. In such a case it is best to assume in our treatment that a fracture exists until the contrary is known. Combat swelling by rest, the use of evaporating lotions, and moderate compression.

In impaction the diagnosis is difficult. The moderate deformity is concealed by swelling; crepitus and preternatural mobility do not exist unless the fragments are pulled apart, and there is not necessarily much loss of function. A conclusion is reached largely by considering the nature, direction, and extent of the violence, the seat of the pain, and by a careful study of the most minute deformity. It is difficult to recognize fissures. They rarely present any evidence of their existence except a localized pain, and possibly a linear ecchymosis appearing after a few days.

In green-stick fractures the age, the deformity, and possibly crepitus during reduction help in the diagnosis, although in many cases no crepitus is obtained. Epiphyseal separations are diagnosticated by the age, the preternatural mobility, the pain, the swelling, the ecchymosis, the deformity, the situation of the injury, and the absence of crepitus or the presence only of a soft crepitus. It is important, however, to remember that an epiphyseal separation is sometimes incomplete, and even when it is complete there may be no displacement. In cases without displacement the *x*-rays will not enable

us to make a diagnosis. In many cases of complete separation soft crepitus is obtainable, but in not a few cases it is not to be found. In incomplete separation crepitus is absent. If absent in complete separation, probably some tissue is caught in the opened area between the fragments. Fractures are often difficult to recognize when occurring in a group of bones (which are firmly joined by dense ligaments) like those of the carpus and tarsus, or in one of two parallel bones. There is not always a certainty that a fracture exists (see below), and when, after a careful examination, there is still uncertainty, do not prolong the efforts or use great force, but treat the case as a fracture until a cure ensues or the diagnosis becomes apparent.

In a child the diagnosis of fracture is sometimes difficult. Pain may be trivial. Children are liable to a form of fracture in which the periosteum is but slightly torn or is not torn at all, the disability and pain are often slight, and the fracture may be easily overlooked (Cotton and Vose).

We have recently had added to our resources a method of incalculable value in diagnosticating fracture; that is, the use of the force known as the *x*-ray or the Röntgen ray. We can look through a part with a fluoroscope and see the bones as shadows, or we can take a negative of the shadows and print skiagraphs from it. This method is applicable even when the parts are swollen, and even when a limb is clothed or wrapped in dressings. It is possible to obtain a picture of a fractured skull; fractured ribs and vertebræ can be detected; and the process is of the greatest use in detecting fractures of the limbs. It is not infallible. An epiphyseal separation may not be detected, and a slight angling of the plate may give a deceptive apperance of distortion. An *x*-ray picture, to be useful, must be taken by an expert and should be interpreted by a surgeon in association with the *x*-ray expert. It is imperative to employ this method in doubtful cases if an *x*-ray apparatus is accessible. It is advisable to use it in all recognized cases and in all suspected cases.

**Complications and Consequences.**—Some of the consequences and complications of fractures are—sloughing of the soft parts, thus making the fracture compound; extravasation of blood, causing swelling or even gangrene; rupture of the main artery or vein of the limb; dislocation; edema from pressure of extravasated blood, from inflammatory exudation, from tight bandaging, from thrombosis, or, later, from the pressure of callus; stiffness of joints from synovitis with adhesion, from displaced fragments, or from intra-articular callus; stiffness of tendons from adhesive thecitis or from the pressure of callus; paralysis from traumatic neuritis, the pressure of callus upon nerve-trunks, or from division of a nerve; muscular spasm; painful callus; exuberant callus; embolism; fat-embolism; pulmonary congestion; pulmonary embolism; gangrene; shock; septicemia; pyemia; tetanus; delirium tremens; urinary retention; extensive laceration of the soft parts; rupture of large nerves, and involvement of joints. A fracture may fail to unite, fibrous union or cartilaginous union only being obtained. An epiphyseal separation may arrest the future growth of the limb.

**Repair of Fractures.—Simple Fracture.**—In a simple fracture the bone is broken, the medullary contents are lacerated, the periosteum is torn, and the overlying soft parts are damaged to a considerable degree. The periosteum is stripped more or less from each fragment, but it is rarely completely torn through, an untorn portion known as the *periosteal bridge* remaining. The amount of blood effused is usually considerable, and it forms a decided prominence at the seat of fracture; it gradually gathers because of oozing, and soon clots. This clot lies in the medullary canal, between the fragments, under the periosteum at the ends of the fragments, and in the tissues outside of the periosteum. Very rapidly after the accident the dam-

aged parts inflame (bone, endosteum, periosteum, and the torn periosseous structures). The inflammatory exudate enters into the blood-clot and the leukocytes eat up and destroy the clot. The clot is simply dead material and in no way contributes to repair. The cells of the damaged tissue proliferate and the young proliferating cells (fibroblasts) enter into the spaces in the clot which were eaten out by the leukocytes. Finally, the entire clot is replaced by fibroblasts and much of this cellular mass quickly becomes vascularized (granulation tissue).

The osteoblasts which exist in the deeper layers of the periosteum and, in the tissue of the medulla itself, begin to proliferate actively soon after the fracture has taken place. The fibroblasts have been formed by the proliferation of the ordinary connective-tissue cells, and the proliferating osteoblasts soon enter into and become widely distributed through this mass of fibroblasts. Some observers maintain that the fibroblasts themselves are directly transformed into bone; others deny this, and think that all bone formation comes from the osteoblasts. Osteoblasts may form bone directly, or may form cartilage first and then bone. When a fracture takes place, a bridge of periosteum is usually left untorn, and this bridge holds the fragments in contact at some point, just as a strap nailed to a trunk and also to its lid holds these two objects in contact at some point. The new tissue about the periosteal bridge always

Fig. 270.—Compound fracture.

becomes cartilaginous for a time, but the rest of the callus rarely shows the development of cartilage, and passes directly into bone. If, however, osteoblasts fail to proliferate with sufficient activity, the mass of granulation tissue becomes fibrous tissue; bone is not formed at all, or is very scantily formed, and fibrous union occurs. If the osteoblasts lack activity, but are more active than in the case just cited, they form cartilage extensively—but cartilage only; consequently, cartilaginous union occurs. During the process of the repair of a fracture the ends of the bony fragments are always softened, and some of the bone is absorbed by the osteoclasts. The osteoclasts are really large osteoblasts that have lost the power of producing bone and that furnish a secretion to absorb bone (the elder Senn). After bony union has been accomplished the osteoclasts absorb the superfluous callus. The mass of new tissue around and between the bone-ends is called *callus*. It will be observed that the name is applied successively to fibroblastic tissue, granulation tissue, fibrous tissue, and bone. Warren tells us that callus has no well-defined outline, and "involves not only the bone and periosteum, but also the connective tissue and some of the surrounding muscular tissue." Within a few days after the injury the inflammatory mass is much firmer than follows inflammation involving other structures, and the bone ends have become deeply embedded in a dense mass.

During the second week the callus is greatly strengthened by the formation of dense fibrous tissue in and below the periosteum, of less dense fibrous tissue outside the periosteum, and of cartilage from the periosteal bridge. The newly formed tissue contracts decidedly. During the third week ossification begins at the points farthest from the fracture, and in the course of a short time (from three to six weeks) is complete. The mass of ossified callus, or new bone, is spindle shaped and spongy.

The terms *intermediate*, *definitive*, or *permanent* callus are used to describe the material which forms between the ends of the broken bone. The names *provisional* or *temporary* callus are given to the material within the canal (central callus) and external to the bone (ensheathing callus). The amount of provisional callus depends directly on the extent of separation and the amount of motion between the fragments. It is Nature's splint, and when the break is not well immobilized a large amount is formed. The greater the amount of motion, short of a degree sufficient to cause non-union, the larger the amount of provisional callus.

Fig. 271.—Ununited fracture of humerus; unsuccessful wiring.

The ensheathing callus is after a time largely absorbed, and the central callus in the course of a long time may also be absorbed, with the restoration of the medullary canal, although this latter result is rare. An excessive amount of provisional callus may ossify nearby tendons, may unite parallel bones (radius to ulna, tibia to fibula, a rib to its neighbors), may block a joint just as a stone in the crack of a door will block a door, or may absolutely abolish a joint. Fragments, even if entirely detached, often unite, but they may be surrounded by provisional callus; sometimes they do not cause trouble, but sometimes suppuration takes place. It takes about one year for Nature to remove the temporary callus. The definitive or permanent callus after a time ceases to be porous and becomes very dense bone.

**Compound fractures** (Fig. 270) without much destruction or bruising of soft parts, if treated antiseptically, soon become simple fractures and unite as such. If the wound is not drained and asepticized and septic inflammation occurs, pus forms, and union by granulation is the best that can be obtained. Compound fractures by direct violence will not heal by first intention because of the loss of vitality of a large area of the soft parts.

**Delayed union** is usually due to imperfect approximation or unstable fixation of the fragments. Imperfect approximation may result from failure to reduce the fracture (muscle, ligament, or synovial membrane being caught between the bone-fragments); the use of unsuitable splints; too tight application of bandages; pregnancy; and general causes of ill health, for instance, anemia, scurvy, Bright's disease, rickets, and syphilis. In delayed union there is pain on passive motion; in non-union there seldom is. In delayed union there

is loss of voluntary motion; in non-union there is power of voluntary motion (A. H. Tubby, in "Brit. Med. Jour.," Dec. 7, 1901). In delayed union there is apt to be tenderness on pressure and often a quantity of callus can be palpated. Delayed union is not non-union, but may eventuate in non-union. The exact time requisite for the solidification of a particular fracture cannot be predicted. The average, taken from a large majority of patients, is not true in a minority. For no apparent reason consolidation may require two or three weeks more than the average, but be accomplished at last. Mr. Jones, of Liverpool, well says that ununited fracture is often the result of "surgical impatience," the surgeon frequently examining a part in which union is slow, and that "non-union would rarely occur if delayed union obtained proper attention" ("Brit. Med. Jour.," Dec. 7, 1912).

Fig. 272.—Vicious union of fractured bones of the leg. View from inner side of limb.

Fig. 273.—Ununited fracture of the femur.

**Bending of Callus.**—Sometimes apparently sound callus will bend. This is particularly apt to occur in the leg if the bones are not in correct alignment. Failure of correct alignment means great deflection of the weight of the body. I have seen shortening of the femur increase during the third month after a fracture. Mr. Jones, of Liverpool (Ibid), points out that the surgeon can refracture bones by manipulation up to four months after the break. It is obvious that fractures are not soundly united as early as we used to believe, and that a patient must not be allowed to walk too early.

**Vicious** or **faulty union** is union with great deformity (Fig. 272). This occurs when no treatment has been employed, or when immobilization has been imperfect, or when deformity has not been reduced. It may arise because retentive dressings have been removed by the patient at too early a period, the

callus yielding. In many cases it is slight and produces little or no pain or impairment of usfulness. In other cases it is pronounced and produces functional impairment or disastrous pressure on nerves or vessels. Vicious union near a joint always impairs function. If there is pronounced vicious union the bone should be rebroken and set as a fresh fracture. In some recent cases the bone is broken by manual force, and for a number of weeks after a fracture this can be easily accomplished. In older cases osteotomy should be performed.

**Non-union of Fractures.**—An ununited fracture is a fracture in which union is not effected at all or in which it is not brought about by bone (Figs. 271 and 273). Non-union is especially common in fractures of the upper third of the femur and of the middle third of the humerus. The causes are local and constitutional. The *local causes* are: (1) Want of approximation of fragments; a frequent cause of want of approximation is interposition of soft tissues—viz., muscle, fascia, or periosteum; this is a common cause of non-union, a cause responsible for a decided majority of the cases; if soft tissues are interposed between bone-fragments non-union is almost inevitable; (2) want of rest. As pointed out above, delayed union may result in non-union because of frequent meddlesome examinations. As Jones says, if there is no union at the end of the fifth week do not examine daily, but leave the parts alone for a fortnight at least; (3) want of blood-supply (as seen in the heads of the humerus and femur, or when a nutrient artery is torn, or when a thrombus forms in a vein near the fracture); (4) defective innervation; (5) bone disease; (6) the use of unsuitable splints; (7) tight bandaging. The *constitutional causes* are debility, scurvy, Bright's disease, syphilis, etc. Sometimes union fails without appreciable reason. In an ununited fracture the broken ends of the bone round off and the medullary canal of each fragment becomes closed by bone. The fragments may not be held together by any material, or they may be held by very thin and much-stretched fibrous tissue (*membranous* union), or by strong, thick, fibrous tissue (*ligamentous* or *fibrous* union). When the ends of the bones come together, are held by a fibrous capsule, and move on each other, there exists a *false joint* or *pseudo-arthrosis*. Such a joint may after a time secrete serous fluid for lubrication. In very rare cases a fracture once apparently soundly united may at a later period be obviously ununited, callus having been absorbed or broken. Pain on active motion in the region of a fracture a number of weeks old suggests non-union. If there is also tenderness non-union is highly probable. If, with pain and tenderness, there is marked thickening from callus, non-union is certain ("Brit. Med. Jour.," Dec. 7, 1912).

**Treatment of Fractures.**—If a man is found in the street with a fracture, further injury must be prevented by applying, after cutting off the clothing over the fracture, some temporary support. If an ambulance or patrol-wagon cannot be obtained, move the patient by hand. If the lower extremity be involved, an improvised stretcher (a board or a shutter) is placed on the ground beside the patient, who is laid on the stretcher, the surgeon lifting the injured limb, and the patient is then carried to the hospital and carefully transferred to a fracture-bed, or, if taken home, to a small ordinary bed, several boards being placed transversely beneath a rather hard but even mattress. The temporary appliances are now removed and a diagnosis is made by the methods before given. Whenever possible have *x*-ray pictures taken (see page 523). After determining the nature of the injury the fragments must be adjusted. This should, if possible, be done at once, because a fracture remaining unreduced may become compound, the fragments may injure important structures, and they are apt to cause intense pain. Reduction is easily effected during shock, as the muscles are in a state of relaxation. Early reduction and fixation largely prevent swelling. If there is very great swelling, reduction may be impossible, and the part must then be supported, moderate

cold, sorbefacients, and gentle pressure being used, ice and tight bandaging, which predispose to gangrene, not being employed. In most cases we can reduce displacement in spite of swelling and cure swelling by the reduction. Set the fracture at the first possible moment. Velpeau's axiom was to reduce fractures at once, regardless of pain, spasm, or inflammation, as reduction is their cure. The longer we wait to reduce a fracture, the greater the amount of force necessary to accomplish it because of progressive infiltration of the soft parts with inflammatory exudate and blood, a process which lessens and finally destroys tissue elasticity. In reduction try to get broken ends in even apposition. In this we may fail, but we must at least strive to obtain a correct alignment. In order to obtain apposition or alignment it may be necessary to make traction by pulleys, and if this is done the patient must be anesthetized (Jones, "Brit. Med. Jour.," Dec. 7, 1912). He says that "end-to-end apposition with an angle of deflection is less satisfactory than slight overlapping in the presence of correct alignment" (Ibid.).

If the patient is very nervous, if the pain is severe, or if rigid muscles antagonize the efforts of the surgeon, reduce the fracture under anesthesia. In some fractures (as those of the clavicle) adjustment is effected by altering the position, and in others (as those of the femur) by extension and counterextension, aided perhaps by pulleys; in some by tenotomy, and in some by kneading, bending, and coaptation. When extension is employed, always endeavor to get a point of counterextension. The extension is to be made on the broken bone (if possible, in the axis of the bone), is to be steady, and neither jerky nor violent. In some cases complete reduction is impossible. This may be due to spasm, to swelling, to the catching of soft parts between the fragments, to the existence of a loose fragment, to locking, or to impaction. An impaction by rotation can generally be released, but it is sometimes undesirable to unlock it. If the fragments cannot be adjusted without violence, retain them in the best attainable position, combat the antagonistic cause, and set them properly as soon as possible or else operate.

After adjusting the fragments maintain them in position by some apparatus. Do not use set splints for each variety of injury. The splints we describe as commonly used are suited to many cases, but in each case a surgeon uses the plan of treatment which in his opinion is suitable to that case. In a given case the routine plan may prove unsuitable. The treatment is to be adjusted to the individual case. The case is never to be forced to an unsuitable routine treatment. All sorts of materials are used for splints, among them may be mentioned wood, felt, pasteboard, plaster of Paris, silicate of sodium, tin (Levis), and aluminum (Elsberg). Avoid pressure over joints or bony prominences and particularly guard against tight or improper bandaging. In fracture of a bone of a limb the circulation in the fingers or toes must be observed as an index of circulation in the limb; hence leave those digits exposed. A retentive apparatus should prevent the redevelopment of deformity, and not be itself productive of pain or harm. For the first few days of treatment of a simple fracture the dressing is removed every day, to make sure that deformity has not recurred, and if it does recur the fragments must at once be reset. The splints should be padded thoroughly, especially when over joints or bony prominences, and they should, if possible, fix the joints immediately above and below the break. A primary roller should not be used unless plaster is to be employed. By a primary roller is meant a bandage applied to the extremity before splints are placed upon it.

Some surgeons at once apply an immovable dressing. This proceeding is safe in simple fractures without much displacement or soft-part injury. This dressing is valuable in military practice, for the old and feeble whom we fear to put to bed, for the young who are very restless, and for the insane or the

delirious. If, however, there is great deformity, much soft-part injury, or marked swelling, immovable dressings may induce sloughing, edema, gangrene, or faulty union. In the above-named cases use ordinary splints for the first few days; then, if it is desirable, the immovable dressing can be applied. Plaster-of-Paris bandages are used with great care in very young children, as gangrene might result from careless application. It is dangerous to keep old or feeble persons long in bed, as they are prone to develop bed-sores and hypostatic pulmonary congestion. The period for the artificial retention of the fracture varies with the seat of the fracture and the age and condition of the patient. Passive motion is to be made in most fractures in from two to three weeks, though it is sometimes made earlier to prevent ankylosis, and sometimes later because of risk of non-union. Landerer strongly advocates massage, believing that it hastens union and prevents wasting. He applies it as soon as there is no danger of the callus bending (in from eight to fourteen days). Massage should not be used when great edema points to the possibility of venous thrombosis. The movements might break up a clot and cause fatal embolism.[1] Very early massage may cause fat-embolism. In fracture of the patella wiring is frequently performed, and wiring or plating is frequently practised in fracture of the clavicle, fracture of the tibia, fracture of the upper third of the femur, and other regions. If fragments cannot be approximated or retained by ordinary methods, an incision should be made, approximation effected, and the fragments retained by wire, a clamp, a plate, or a bone ferrule.

The plan known as the **ambulatory treatment** of fractures of the lower extremities has had warm advocates. The ambulatory splint is an apparatus which enables a man to walk about a few days after receiving a fracture of the leg or thigh. It was devised by Hessing, a carpenter dwelling in a village near Augsburg. Its aim is not only to get the patient about on crutches, but also to cause him to use the limb. It is held that this plan of treatment greatly lessens the patient's sufferings and actually favors union by the stimulation of walking. Bardeleben, in his report to the German Surgical Congress, gave the records of 111 fractures of the lower extremity thus treated (77 simple and 12 compound fractures of the leg, 17 simple and 5 compound fractures of the thigh). The patients were gotten about a few days after the accident, were able to attend to business, had excellent appetites, digested their food perfectly, slept well, and were saved from muscular atrophy. Pilcher has warmly advocated the method. It can be used in fractures as high up as the middle of the femur. The apparatus which we should employ in the ambulatory treatment reaches below the sole of the foot, and is supported firmly above the seat of fracture, the weight of the body being transferred from above the fracture to the firm pad below the sole of the foot on which the patient walks (Figs. 274 and 275). This appliance in a fractured thigh is put on about one week after the infliction of the injury. While the patient sits on the ischial tuberosities extension is made upon the leg. The seat of fracture is encircled by a thin plaster cast. The sole of the other foot is raised by a cork sole. Albers, when treating a fractured thigh, uses plaster of Paris strengthened by bits of wood, running from *below* the sole of the foot to the iliac crest. Krause says in fracture of the ankle carry the dressing to the head of the tibia; in fracture of the leg carry it to the middle of the thigh; in fracture of the lower end of the femur carry it to the pelvis.[2] Bradford warmly advocates the use of Thomas's splint often combined with plaster of Paris. During the last few years surgeons have come to recognize that ambulatory treatment must not be used for all fractures of the lower extremity and is only suited to selected cases.

[1] Cerne's case, in "Normandie méd."; "Bull. med.," 1895, No. 44.
[2] "Centralbl. f. Chir.," vol. xxii, 1895.

**Prevention and Treatment of Complications.**—In every case of fracture of an extremity feel for the pulse between the periphery and the seat of injury in order to be sure the artery is not ruptured. If the soft parts are badly contused, try to prevent sloughing by employing rest and relaxation and by applying heat. If superficial sloughing occurs, treat antiseptically, remembering that even a superficial excoriation can admit bacteria which, carried by the blood or lymph, may infect the bones. If a slough leads down to the fracture, treat the case as a compound fracture. If there be great blood extravasation the danger is gangrene, and after fracture of the lower extremity the foot of the bed may be elevated, or, better, after fracture of the upper or lower limb the extremity, to which splints and bandages are to be loosely applied, is to be raised and surrounded with hot bottles. If a bleb forms, it is to be opened

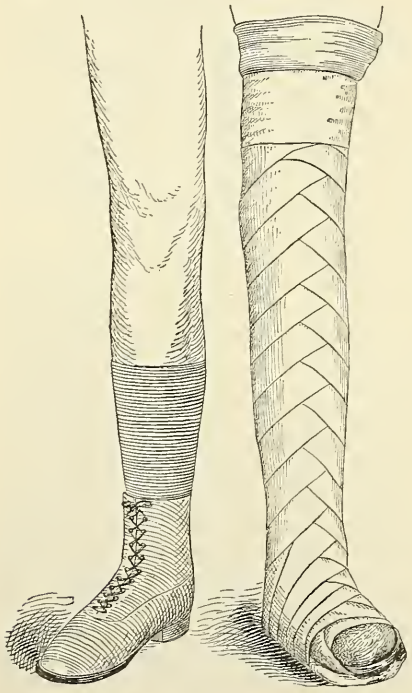


Fig. 274.—Ambulatory dressing of plaster of Paris for fracture of the bones of the leg (Pilcher).

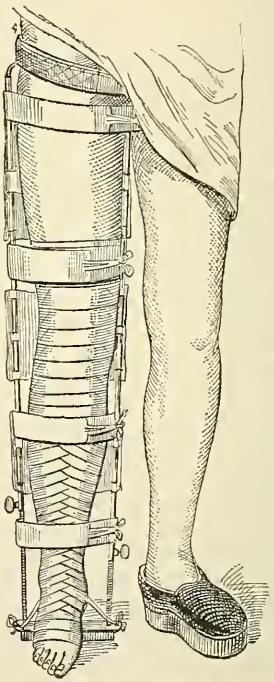


Fig. 275.—Ambulatory dressing apparatus for fracture of thigh (Harting).

with a clean needle and dressed antiseptically. If gangrene occurs, treat by the usual rules. Frequently after fracture of a bone blebs containing reddish serum form on the skin. The appearance of *blebs* when the circulation is good does not mean gangrene, and is not of any particular consequence. If blebs are distinct symptoms of circulatory impairment, gangrene impends or already exists.

*Edema* may be due to tight bandaging. If it is due to phlebitis, there is danger of pulmonary or cerebral embolism. In phlebitis elevate the limb, remove all constriction, and employ locally ichthyol ointment; do not use massage, and give stimulants by the mouth. In edema due to weak circulation or venous relaxation use daily frictions and firm bandaging. If the fracture involves a joint, carefully adjust the fragments, make passive motion early, and inform the patient that he is in danger of a permanently stiff joint.

A *dislocation occurring with a fracture* is reduced at once if possible. To do this, splint the limb and give ether, and try to reduce while the limb is massaged, using the splint as a handle. Allis is often able to reduce a dislocation accompanied by a fracture. He uses the untorn portion of periosteum as a hinge, pulls upon the lower fragment, and thus draws down the upper fragment and pushes it in place by manipulation. If this fails, it is best to incise and pull the separated end in place by the hook of McBurney and Dowd (Figs. 276–278); but some surgeons say, get the bones in the best possible position, set them, await union, and then treat the unreduced dislocation. A *rupture of the main artery* of the limb presents the symptoms of absent pulse below the rupture, a tumor which may pulsate, and possibly a whirring sound or an aneurysmal thrill and bruit. This condition demands that the surgeon should apply an Esmarch bandage, cut down upon the tumor, turn out the clot, and ligate each end of the vessel. *Rupture of the main vein* of a limb causes intense edema and calls for sutures, lateral ligature, or complete ligation. If these measures fail after injury of vein or artery it is seldom successful to ligate higher up. Such a course might succeed in the upper extremity. It would almost certainly fail in the lower. Amputation will probably be necessary. If gangrene appears, amputate at once above the seat of the fracture.

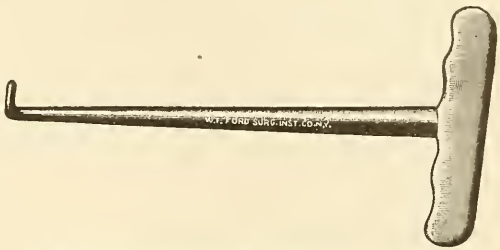


Fig. 276.—Fracture-hook (McBurney and Dowd).

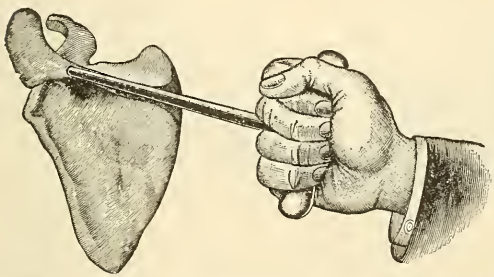


Fig. 277.—Fracture-hook applied at base of acromion process (McBurney and Dowd).

*Inflammation* is to be treated by compression, rest, moderate cold, and later by 50 per cent. ichthyol ointment. *Muscular spasm* requires morphin internally, firm bandaging, or even tenotomy. Fat-embolism is treated by stimulants and inhalation of oxygen, and possibly artificial respiration. Shock, delirium tremens, urinary retention, etc., are treated according to the ordinary rules of surgery.

**Functional Result of Non-operative Treatment.**—Union with a good anatomical result means a good functional result in over 90 per cent. of the cases. We used to suppose that a good functional result is usual even with a poor anatomical result, but Jones ("Brit. Med. Jour.," Dec. 7, 1912) shows that we get it in less than 30 per cent. of cases in which the anatomical result is bad.

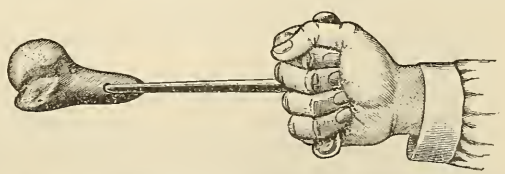


Fig. 278.—Fracture-hook inserted in displaced fragment (McBurney and Dowd).

**Treatment of Compound Fractures.**—It must first be decided, in a case of compound fracture of a limb, if amputation is necessary, and the *x*-rays are of great value in determining the condition of the bones in a crushed part.

Amputation is demanded when the limb is completely crushed or pulpified through its entire thickness; when extensive pieces of skin are torn off; when the main artery, vein, and nerve are torn through; and sometimes when there is violent hemorrhage from a deep-seated vessel or when an important joint is splintered. What is to be done is to some extent determined by the patient's age and general health. In a healthy young person, if in doubt, give the limb the benefit of the doubt and try to save it; if the artery alone is ruptured, cut down upon it and tie both ends; if the vein alone is torn, suture it, apply a lateral ligature, or tie both ends; if the nerve is severed, suture it; if a joint is opened, drain and asepticize. If an attempt is made to save the limb, be ready at any time to amputate for gangrene, secondary hemorrhage (if re-ligation at original point and compression high up fail), extensive cellulitis, and profuse and prolonged suppuration.[1] When it is determined to try to save the limb, the part must be cleansed thoroughly by the antiseptic method (in no injuries is this more important). If a small portion of bone protrudes, cleanse the skin of the extremity and the protruding bone, push the spicule out a little more, and cut it off. If a large piece of bone is protruded it must not be cut

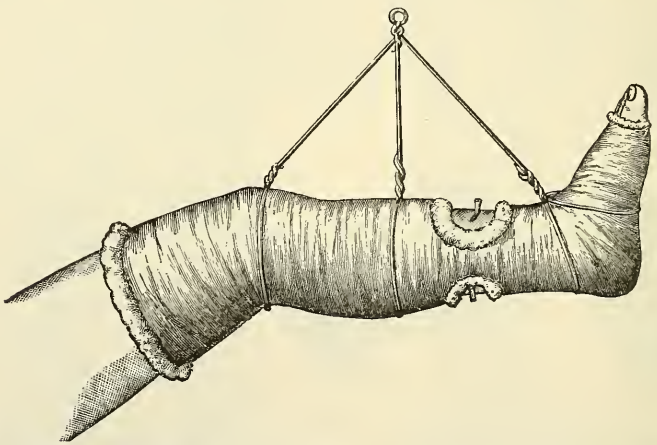


Fig. 279.—Fenestrated plaster-of-Paris dressing. Drainage-tube pulled through limb.

away, but should be thoroughly disinfected, and after the skin wound has been enlarged should be returned into place. Hemorrhage requires a free incision to permit of ligation of bleeding points. In most comminuted fractures splinters should not be removed. To remove them favors non-union. In all cases a drainage-tube must be carried down to the seat of fracture, and in some cases a counteropening must be made and the tube be pulled through the limb (Fig. 279).

After inserting the tube the wound is sutured, a plentiful antiseptic dressing is applied, and the extremity is dressed with plaster. The plaster can be applied over a narrow strip of wood, trap-doors or fenestra being cut in the plaster before it sets (the *fenestrated splint*) (Fig. 279). The wound is then covered with gauze and a bandage.

The bracketed splint is a better dressing than the one just described. After the wound has been dressed with gauze, plaster is at once applied over the ends of brackets (Fig. 280). The above methods not only immobilize the fractured bones, but keep the parts aseptic and afford easy access to the wound. The drainage-tubes are usually removed, if suppuration does not occur, in from

[1] See Howard Marsh, on "Fractures," in Heath's "Dictionary of Practical Surgery."

forty-eight to seventy-two hours. The wound is treated as any other wound. In some compound fractures there is difficulty in retaining the fragments in apposition (lower end of femur, upper third of femur). In such cases the ends of the bone should be resected and the bones should be fastened together as in a case of ununited fracture, with silver wire, aluminum wire, chromicized catgut, kangaroo-tendon, or the bones should be plated. In a *compound fracture of the patella*, after free incision and disinfection, investigate to determine the gravity of the injury. In an ordinary case in which there are two or three fragments, open the joint, irrigate with saline fluid, drill the fragments, and fasten them with silver wire. Very small fragments should be removed. A tube is carried into the joint, the wound is sutured and dressed, and the limb is immobilized in extension. In a case of severe compound comminuted fracture of the patella, after disinfection, any completely loose piece should be removed and "the remaining portions made smooth with bone forceps and the sharp spoon."[1] The wound is only partially sutured, is drained and dressed, and the limb is placed on a straight posterior splint. If a fracture of a rib is compound internally, resect the rib; if it is compound externally, dress antiseptically.

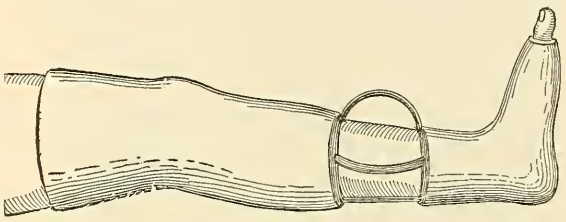


Fig. 280.—Bracketed plaster-of-Paris dressing.

Compound fractures may be followed by gangrene, sloughing, periostitis, septicemia, pyemia, osteomyelitis, necrosis, etc.

*Operative Treatment of Recent Fractures.*—Many cases are now operated upon primarily. Others are operated upon because non-operative methods fail to obtain or to maintain good position. In some fractures reduction and fixation are only possible by operation.

Skiagraphs have demonstrated that the ordinary non-operative treatment is often followed by permanent displacement. In many cases this does not seriously impair function, in not a few it does. There is much impairment of function after fracture of the patella with wide gaping of the fragments, and after fracture of the femur with repair in a position of marked angulation or decided overlapping.

The most perfect results are obtained by operation, which exposes the fracture and enables the surgeon to correct the deformity and solidly fix the fragments. Practically all surgeons agree that for fracture of the patella, fracture of the olecranon, and fracture of a long bone with incomplete reduction, or in which deformity recurs in spite of splintering, operation gives the best chance for a good functional result. In most fractures of long bones treated conservatively perfect apposition of the fragments is not obtained, although we may think it has been. Bloodgood points out that in fractures near joints there is great difficulty in reduction and little evidence of deformity.

The Special Committee of Inquiry of the British Medical Association in a recent report warmly advocates operation in many cases. Jones, of Liverpool, although believing as a general rule in non-operative treatment, says that if in any case there is sound reason to doubt the successful outcome of non-operative treatment, operation should be performed. He opposes waiting to see what happens because the delay may lose the chance of obtaining a good functional result ("Brit. Med. Jour.," Dec. 7, 1912).

[1] Lilienthal's "Imperative Surgery."

Personally, I follow Mr. Jones's rule and operate when I fear that conservative treatment may fail. I operate primarily for:

Fracture of the patella.

Fracture of both bones of the leg in the lower third.

Most fractures of the os calcis.

Some cases of Pott's fracture.

Most cases of fracture of the upper third of the femur.

Some fractures of the neck of the femur in the young and middle aged.

Some fractures of the surgical neck of the humerus.

Fractures of the olecranon, especially those in which the upper fragment has rotated.

Some fractures of the elbow-joint.

Some fractures of both bones of the forearm (in order to preserve pronation and supination).

Some fractures of the metacarpus.

Fractures of the zygoma.

Some fractures of the mandible.

Fractures of the clavicle when complete reduction is impossible or when sharp-pointed fragments threaten to pierce the skin or damage important structures.

In compound fractures, in many comminuted fractures, if an important nerve or blood-vessel has been divided.

Most children are managable by conservative methods and do not do as well as adults after operation. Hence in children I am more conservative than in adults.

Again, do not forget that operation may not give a good functional result. It often fails to do so. If this is not well understood both the surgeon and patient may be disappointed after operation. There is, of course, some risk in the treatment by incision, and it is only justifiable in competent hands and amid proper surroundings. The occasional operator should look to it with less confidence than the daily operator. If infection occurs it will be a catastrophe, and may cause death, hence perfect asepsis is imperatively required. It can never be routine treatment and will never be extensively employed outside of a hospital.

If operation is determined upon, an incision is made, the bone is inspected, tissue intervening between the fragments is removed, and the fragments are coaptated and fixed by screws and a perforated plate, by silver wire sutures, bone-ferrules, chromic catgut, nails, or some form of clamp.

Plates and screws give the best results. Wire used for fracture of a long bone acts as a hinge, and in a wired fracture the alignment is apt to be disturbed. Wire was first used for this purpose in 1775 by Lapeyode and Sicre, of Toulouse (Geo. W. Guthrie, "Amer. Med.," March 7, 1903). It is now seldom used except for the patella, the olecranon, the clavicle, the zygoma, and the anatomical neck of the humerus. I prefer the steel plates of Arbuthnot Lane (Fig. 281) or the silver plates of Halsted. These plates are perforated for screws. The plate is not removed unless it loosens or gives trouble. After operation the extremity must be carefully fixed by splints or plaster of Paris; the circulation must be watched, guarded, and maintained; massage should be used and passive motion be employed as in a case treated by the non-operative method. If rigid fixation of the fragments is obtained and maintained repair will usually occur with very little callous formation.

*Treatment of Delayed Union and Ununited Fracture* (see page 692).—When delayed union exists, seek for a cause and remove it, treating constitutionally if required, and thoroughly immobilizing the parts by plaster. Orthopedic splints may be of value. Use of the limb while splinted, percussion over the fracture,

and rubbing the fragments together, thus in each case producing irritation, have all been recommended. Blistering the skin with iodin or firing it has been employed. If the union be very long delayed, forcibly separate the fragments and put up the limb in plaster as we would a fresh break. If these means fail, irritate by subcutaneous drilling or scraping, or, better, by laying open the parts and then drilling and scraping at many places. Buechner advocates the induction of hyperemia by a constricting band, just as Bier induces congestive hyperemia in treating tuberculous areas. At first the constriction is permitted to remain but a short time, but the period is lengthened every day, until in a few days it remains almost continuously day and night. It is to cause a pinkish-blue flush of skin, but not pain. The limb must be warm to the touch. During the two or three hours daily that the band is off raise the limb to relieve edema. Ten days of this treatment will inaugurate union in many cases. Helferich devised this method in 1887. In several cases I have thought that it did good. I also administered thyroid extract to these patients. Lannelongue and Menard inject a 1:10 solution of zinc chlorid between the fragments. I have had several successes with this plan. Leaving acupuncture

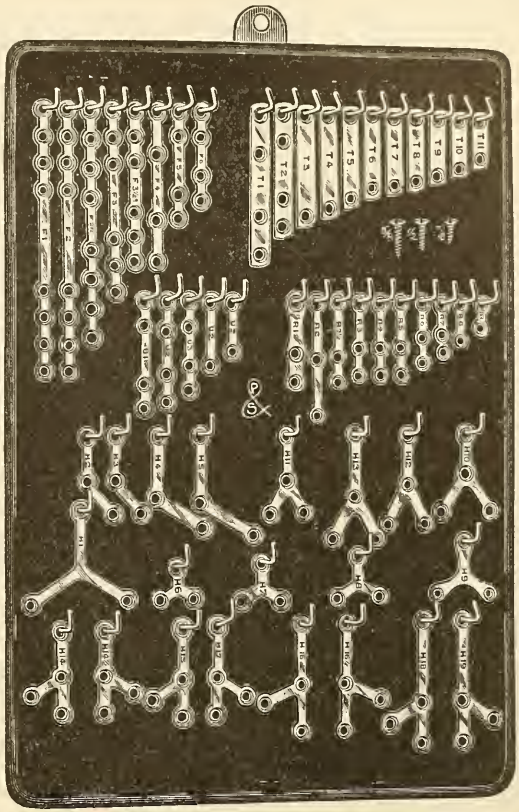


Fig. 281.—Lane's vanadium steel plates.

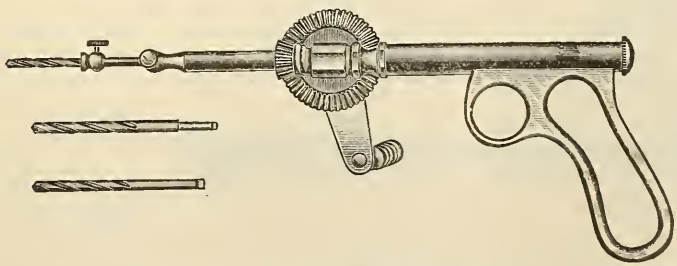


Fig. 282.—Richter's bone-drill (pistol grip).

needles in for days is approved by some, and electropuncture is advocated by others. Cases of ununited fracture must be treated by excision of the bony ends and fibrous tissue, securing the fragments together by periosteal sutures, by pins, by screws and plates, by ivory pegs, by screws, by silver or aluminum

bronze wire, by kangaroo-tendon, by Senn's bone-rings or bone-ferrules, or by chromicized catgut. Personally, I use Lane's plates, made of vanadium steel. Delorme makes an incision, removes bone-splinters and fibrous tissue, smooths

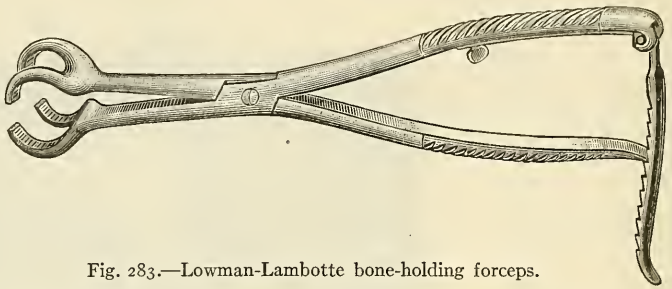


Fig. 283.—Lowman-Lambotte bone-holding forceps.

off one end, forces this into the bored-out medullary canal of the other fragment, and sutures the periosteum. Gussenbauer's clamp will often give a good result, and was used for years by Billroth. This is a metal bar with two nails set at right angles to the bar. One nail is driven into each fragment. Langenbeck fixed a screw into each fragment and connected the screws by a piece of iron. Parkhill's clamp, which is an improvement on Langenbeck's instrument, secures absolute immobility and is a very useful instrument.

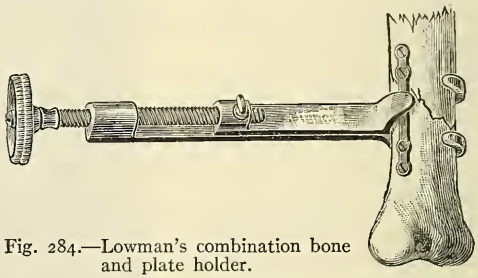


Fig. 284.—Lowman's combination bone and plate holder.

Sometimes union fails in spite of a formidable operation. In such cases there is no tendency to bone production. A bone-graft may be partially separated from one of the fragments and interposed between the freshened ends, a bone-graft may be taken from the sound tibia and be interposed, fresh bone-splinters may be interposed, or a portion of a rib may be used with screws or nails as a clamp. It is always desirable to take the bone-graft from the individual. Such a graft is more powerfully stimulating to bone growth than a graft from another individual or from one of the lower animals. Transplantation from the lower animals has, however, been successfully practised (see page 503).

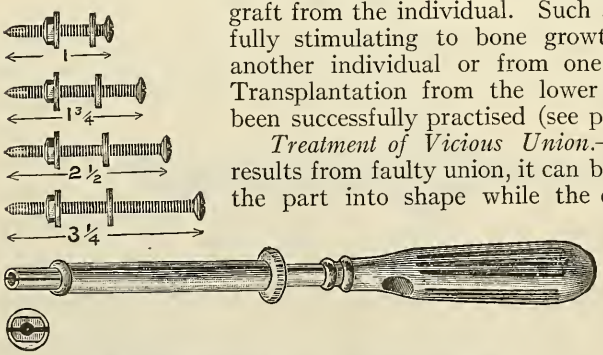



Fig. 285.—Sherman's bone bolts and nut-driver.

*Treatment of Vicious Union.*—If angular deformity results from faulty union, it can be corrected by molding the part into shape while the callus is soft. If the callus has become hard, the bone can be refractured. If faulty union occurs with overriding, an osteotomy can be performed.

**Special Fractures.—Nasal bones,** because of their situation, are often broken. The commonest seat of fracture is through the lower third, where the bones are thin and lack support. The fracture is usually compound externally or through the mucous membrane internally. The *cause* is direct violence. Displacement may not occur at all, but when present it arises purely from

force, and never from muscular action, no muscle being attached to these bones. If the force is from the front, the nose is flattened; if from the side, it is deflected. Displacement is soon masked by swelling. Crepitus can sometimes be elicited by lightly grasping the upper part of the nose with the fingers of one hand and moving it gently below from side to side with the fingers of the other hand. Preternatural mobility is valueless as a sign, because of the natural mobility of the cartilages. Nose-breathing is difficult because of blocking of the nostrils by blood-clot. Diagnosis may be almost impossible when deformity is absent.

The complications that may be noted are cerebral concussion, brain symptoms from implication of the frontal bone or cribriform plate of the ethmoid bone, and extension of the fracture to the superior maxillary or lachrymal bones. Emphysema of the root of the nose, the eyelids, and the cheeks is common, and means either a rent in the mucous membrane of Schneider or a crack in the frontal sinus. There may be much discoloration because of subcutaneous hemorrhage. Epistaxis is usual, and is recognized from the epistaxis produced by fracture of the base of the skull by the facts that the bleeding in the first condition, although profuse, is, as a rule, soon checked, and is not followed by oozing of cerebrospinal fluid, whereas in the second condition it is profuse, continued, and is perhaps followed by a flow of cerebrospinal fluid. Fracture of the bony septum occasionally complicates nasal fractures, and deviation of the cartilaginous septum often takes place. Suppuration may occur and necrosis of bone or cartilage may follow. The prognosis is usually good.

*Treatment.*—Whenever possible nasal fractures should be treated by a rhinologist. After cocainizing the nares a careful inspection should be made by means of a mirror and a light to determine if there is any injury of the septum. This point must be determined in order that the deformity of the septum may be corrected at the same time as the deformity of the nasal bones. When there is no displacement, or when a displacement does not tend to be reproduced after reduction, employ no retentive apparatus of any kind. Order the patient not to blow his nose for ten days and syringe it daily with a solution of bicarbonate of sodium. If deformity be noted, correct it at once, as the bones soon unite in deformity. If the attempts at reduction are very painful, or if the subject be a child, a woman, or a nervous man, give ether to obtain primary anesthesia. Reduction is effected by a grooved director or steel knitting-needle wrapped in iodoform gauze and passed into the nostril; the fragments are lifted with this instrument, and the fingers externally mold them into place. A rubber dilator can be used in reduction. This is pushed into the nose and inflated by air or water. If the septum is deviated and cannot be pushed in place by a metal sound, it must be twisted into place by means of septum forceps. If bleeding is moderate, check it with cold; if severe, by plugging. "For fractures high up with displacement, gauze packing carried well up will be required to retain the elevated bones. For lower deviations the Asch tube will be needed" (Scudder, on "The Treatment of Fractures"). A hollow vulcanite plug is inserted in each nostril and the nose is molded into correct shape over the plug. The patient breathes through the hollow plug. A thread runs from each plug and is fastened to the cheek by adhesive plaster. Once or twice a day the plugs are removed, cleaned, and greased with iodoform ointment. The nose is cleared and the plugs are reinserted. If flattening tends to recur, pass a Mason pin (Fig. 286) just

Fig. 286.—Mason's pin.

beneath the fragments, through the line of fracture and out the opposite side. Steady the fragments by a piece of rubber externally caught on each end of the pin, or with figure-of-8 turns around the ends with silk. Leave the pin in place for five days. The instrument of Mason is a sharp, strong, nickel-plated pin, with a triangular point.

If lateral deformity tends to recur, hold a compress over the fracture or fix a molded-rubber splint over the nose by a piece of rubber plaster 1½ inches broad and long enough to reach well across the face, and use compression for ten days. In neither of the above cases is the nose to be blown, and in both cases it is to be syringed once or twice a day. In fractures rendered compound by tears in the mucous membrane, irrigate with normal salt solution or boric acid solution, holding the head so that the solution will not run into the mouth; plug with iodoform gauze around a small rubber catheter, which instrument permits nose-breathing; carefully remove the gauze daily and syringe. In fractures compound externally cleanse antiseptically externally, and dress with a film of cotton soaked in iodoform collodion or compound tincture of benzoin, or apply sterile gauze. Fractures of the bony septum, if showing a tendency to reproduction of deformity, require packing as above explained, or the use of a special splint within the nostrils (Fig. 287), or the application of vulcanite plugs, so made that the patient can breathe through them and threads can be attached to them. Fractures of the nasal cartilages are to be pinned in place. Fractures of the nose are entirely united in from ten to twelve days.

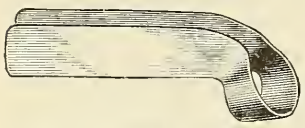


Fig. 287.—Jones's nasal splint.

**Fractures of the Lachrymal Bone.**—The lachrymal bone may be broken when the nasal bones, a superior maxillary bone, or the lateral plate of the ethmoid are fractured, and union is solid in from three to four weeks. The question of how much deformity is to be expected is always uncertain, and in not a few cases obstruction of the nose follows fracture because of damage to the septum.

*Treatment.*—Treat the chief injury, which is the fracture of the other bone or bones. Maintain the patency of the lachrymal duct by frequently passing a clean probe.

**Fractures of the Superior Maxillary Bone.**—Although a fragile bone, the superior maxillary is rarely broken except through the alveolar border. It may be broken by transmitted force from blows on the chin, or on the head when the chin is fixed; but direct violence is the usual cause. The walls of the antrum may be crushed in. Comminution is the rule, and the injury is often compound. These fractures induce great swelling, pain, and inability to chew. Mobility and crepitus may be detected. Deformity is due to the breaking force, and not to the action of any muscle. When a portion of the alveolar arch is fractured, as may occur in pulling teeth, the small fragment is depressed backward, and there exist irregularity of the teeth (some of which may be loosened) and inability to chew food. Fracture of the nasal process is apt to injure the lachrymal duct. When the antrum is broken in there are great sinking over the fracture, depression of the malar bone, and emphysema. Transverse fracture of the upper part of the body of the bone may cause no deformity. The force required to break the superior maxillary bone is so great that fractures of other bones almost certainly occur, and concussion of the brain not infrequently exists. Injury of the infra-orbital nerve is not unusual, causing pain, numbness, or an area of anesthesia involving one-half of the upper lip, the ala of the nose, and a triangle whose base is one-half the upper lip and whose apex is the infra-orbital foramen. There is

also loss of sensation in the gums and upper teeth of the injured side. Fractures of the superior maxillary bone occasionally induce fierce hemorrhage from branches of the internal maxillary artery; and if this has happened, be on the watch for secondary hemorrhage (these vessels being in firm canals).

Fig. 288.—Hard-rubber splint; wire arms and chin-piece held together by metal rods and nuts.

*Treatment.*—If the fracture does not implicate the alveolus, or if no deformity exists, apply no apparatus, but feed the patient on liquid food for four weeks. Reduce deformity, if it exists, by inserting a finger in the mouth. If the antrum is broken in, put the thumb in the mouth and push the malar bone up and back. In certain cases of deformity make an incision at the anterior border of the masseter muscle, insert a tenaculum or aneurysm needle, and pull the bone into place (Hamilton). If the malar bone or malar process is driven into the antrum, Weir tells us to incise the mucous membrane above and external to the canine tooth of the upper jaw, break into the antrum with a bone-gouge, insert a steel sound, lift out the malar bone, and pack the antrum with gauze. Loose teeth are not to be removed; they are pushed back into place and held by wiring them to their firmer neighbors. Hemorrhage is arrested by cold and pressure. If hemorrhage is dangerously profuse or prolonged, tie the external carotid artery.

Fig. 289.—Front view of splint (Fig. 288), with mouth closed (Moriarty).

If the line of the teeth, notwithstanding the wiring, is not regular, mold on an interdental splint. The usual splint for the upper jaw is the lower jaw held firmly against it by the Gibson, the Barton, or the four-tailed bandage. There is a great amount of dribbling of saliva during the treatment, and a dressing must be used to catch this fluid. Every day remove the bandage and dressing,

and wash the face with ethereal soap. The patient, who is ordered not to talk, is to live on liquid food administered by a nasal tube or by pouring it into the mouth back of the last molar tooth by means of a tube or a feeding-cup. Never pull a tooth to obtain a space; but if a tooth is lost, utilize the vacant space for this purpose. After every meal wash out the mouth with peroxid of hydrogen, followed by chlorate of potassium, boric acid, or normal salt solution, and thus prevent foulness and the digestive disorders it may induce. Dispense with the dressings in six weeks, and let the patient gradually return to ordinary diet.

In fractures compound externally do not remove fragments, antisepticize, arrest bleeding as far as possible by ligature, by pressure, or by plugging, wire the fragments if feasible, dress with gauze, and wash the mouth with great frequency. Fractures compound internally are treated as simple fractures, except that the mouth is washed more frequently.

The **malar bone** is rarely broken alone. Hamilton says no uncomplicated case is on record. The malar is a strong bone resting on a fragile support, and hence it may become a wedge to break other bones and yet itself be unfractured. The *cause* of fracture is violent direct force. A fracture of the orbital surface of this bone causes subconjunctival hemorrhage like that encountered in fracture at the base of the skull, and may produce irritation of the infra-orbital nerve. Protrusion of the eye may result either from hemorrhage or from crushing in of the malar bone. There is a hollow below and to the outer side of the orbit. Occasionally the line of fracture is detectable, but mobility and crepitus are very rarely discoverable. Chewing is apt to cause pain, and often the motions of the lower jaw are limited, the coronoid process being pressed upon by a depressed malar bone, an associated fracture of the zygoma, a blood-clot, or swollen tissue (see Scudder, on "The Treatment of Fractures").

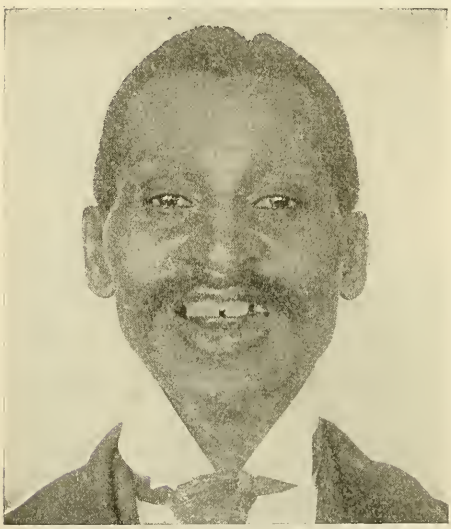


Fig. 290.—Hard-rubber splint in position, upper teeth resting upon it (Moriarty).

*Treatment.*—If no deformity exists, there is practically nothing to be done. If deformity exists, try to correct it as in fractures of the superior maxillary bone. If correction is impossible by ordinary methods and the movements of the lower jaw are impeded by the displaced bone, make a small incision and through this insert an instrument and endeavor to lift the bone into place. As these cases are almost invariably complicated by fracture of the upper jaw, they are treated in the same manner as the latter injury. The union is complete in three weeks.

**Fractures of the zygomatic arch** are very rare. The *causes* are: (1) direct violence; (2) indirect force (from depression of the malar) and (3) forcing foreign bodies through the mouth. Direct violence is the usual cause. Direct violence causes inward displacement, and indirect force may cause outward displacement. The usual seat of fracture is at the smallest portion of the

process—that is, on the temporal side of the temporomalar suture (Matas). The symptoms are pain, ecchymosis, swelling, displacement, and difficulty in moving the jaw (because of injury to the masseter muscle).

*Treatment.*—In simple fracture give ether and try to push the arch in place. Many surgeons do not make an incision, as depression will do no harm and the functions of the jaw will be restored. Simply dress with a compress, adhesive strips, and the crossed bandage of the angle of the jaw. Union will take place in three weeks. Matas[1] advises operation. An anesthetic is administered and the parts are antisepticized. A long semicircular Hagedorn needle is threaded with silk, is entered 1 inch above the middle of the displaced fragment, is passed well into the temporal fossa, and is made to emerge ½ inch below the arch. The silk is used to pull a silver wire around the fracture, and this wire is employed to pull the bone into position. A firm pad is applied externally and the wire is twisted over the pad. Antiseptic dressings are applied, and on the ninth or tenth day the wire, splint, and dressings are removed permanently. I have employed this plan in 2 cases with perfect satisfaction.

**Fractures of the inferior maxillary bone** may, and usually do, involve the body, although they occasionally occur in the rami. Any part of the body may be fractured, the most usual seat being near the canine tooth or a little external to the symphysis (Pick). A portion of alveolus may be broken off. In fractures of the ramus either the angle, the condyloid neck, or the coronoid process may be broken. In fractures of the body the posterior fragment generally overrides the anterior. Fractures of the lower jaw are often multiple and are almost always compound, because the oral mucous membrane and alveolar periosteum are torn. The *cause* is usually direct violence. Indirect violence (lateral pressure) may fracture the body anteriorly. Fractures near the angle are always due to direct violence. Indirect violence may fracture the condyle (falls on the chin), and so may direct violence. Fractures of the coronoid process are very rare, and they arise from great direct violence (usually a gunshot-wound or some other penetrating force).

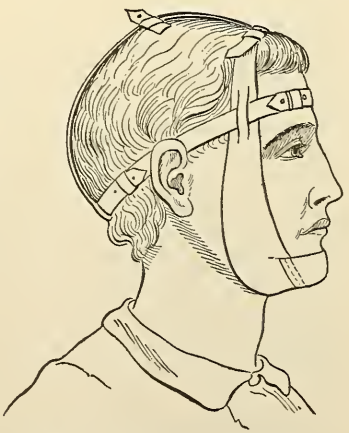


Fig. 291.—Hamilton's bandage.

*Symptoms.*—In fracture of the body preternatural mobility and crepitus generally exist. The gum over the fracture swells rapidly and decidedly. There is bleeding because of laceration of the gum; saliva dribbles constantly; the patient supports the jaw with the hand; great pain exists (possibly from injury of the nerve), and deformity is present, shown by inequality of the teeth if the fracture is anterior to the masseter, the anterior fragment going downward and backward and the posterior fragment going upward and forward. The downward displacement is due to muscular action (action of the digastric, geniohyoid, and geniohyoglossus). The backward displacement is due to the violence. The temporal, internal pterygoid, and masseter muscles draw the posterior fragment upward and to the front. Two or three days after fracturing the jaw some of the cervical lymph-glands enlarge. When a fracture of the lower jaw is compound internally, suppuration usually takes place and the odor of decomposition becomes marked. In fracture of the neck of the condyle the jaw

[1] "New Orleans Med. and Surg. Jour.," Sept., 1896.

is drawn toward the injured side, and the condyle is pulled inward and forward by the action of the external pterygoid muscle. In fracture of the coronoid process the temporal muscle pulls the small fragment upward.

The *complications* are: digestive disorders and dirrahea from swallowing foul discharges; loosening of the teeth; lodgment of loosened teeth between the fragments; bleeding (usually only oozing from the gum, but there may be hemorrhage from the inferior dental artery), and suppuration. Necrosis may follow these fractures, an abscess of the neck may develop, or a sinus may form.

*Treatment.*—Correct deformity with great care and be sure to bring the teeth into normal alignment. As a rule, push loose teeth into place and put back detached ones; but occasionally a tooth obstinately prevents perfect approximation, and if it does it must be removed. Remove a tooth if it lies between the fragments, but replace it in its socket after reducing the fracture. Wash the mouth with hot water to clean it and to check bleeding. If bleeding is very severe, compress the carotid artery for a time. The fracture can be dressed with a pad of lint over the chin and Hamilton's four-tailed bandage (Fig. 291). A common plan is to take a splint of pasteboard, felt, or gutta-percha; pad it lightly with cotton, mold it to the part, and hold it in place with a Barton or a Gibson bandage. If apposition of the fragments cannot be maintained by the above methods, fasten the teeth together with wire, wire the fragments together, or have a dentist apply an interdental splint (Figs. 292, 293). Fracture of the lower jaw can often be satisfactorily treated by Angle's bands. These bands are of great value in complicated cases, in which two or more fractures exist. Each band consists of thin metal and a screw and a nut to fit the screw. The band is adjusted around a firm tooth and a nut is applied so as to hold the band tightly. Several bands are placed upon teeth in both jaws. Silver wire or silk is thrown around the pins of the bands so as to catch, and the jaws are thus held firmly together. The patient is to be fed on liquid food (see Fracture of the Upper Jaw), the mouth is to be washed frequently with peroxid of hydrogen, followed by boric acid solution or normal salt solution, and if bandages are used they should be changed every second day. The union should be complete in five weeks. The best plan of treatment in all cases not operated upon is to send the patient to a skilful dentist and have him apply an interdental splint and direct the treatment. Though these fractures are usually compound, they do not endanger life.

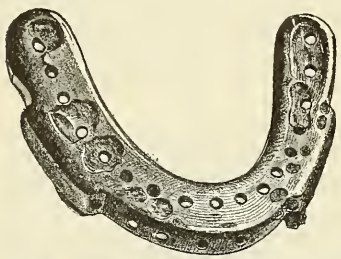


Fig. 292.—Vulcanite splint with boxes vulcanized on each side. If the jaw is fractured in the region of the molars, considerable pressure is required to get the parts in position; therefore it is best to vulcanize on to the sides of the vulcanite splint boxes into which wire arms can be inserted (Pilcher).

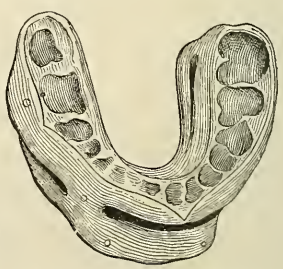


Fig. 293.—Interdental splint.

**Fractures of the Hyoid Bone.**—These fractures are uncommon injuries, and are caused by hanging, by throttling, and by falls in which the neck strikes some obstacle. If the bone breaks by throttling, it is its body which fractures (indirect force). Fractures by muscular action are most unusual.

*Symptoms.*—The symptoms are: A sensation of something breaking;

bleeding from the mouth if the mucous membrane be lacerated; pain, which is worse on opening the jaws or on moving the head or tongue; difficulty in swallowing; muffled, hoarse voice or aphonia; swelling, and frequently ecchymosis, of the neck. There are observed occasionally, though rarely, harsh cough and dyspnea, irregularity of bony contour, and crepitus. Always look into the mouth and see if there can be detected ecchymosis or laceration of the mucous membrane or projection of a bony fragment. The displacement is produced by contraction of the middle constrictor of the pharynx. A fracture of the hyoid bone may destroy life.

*Treatment.*—For dyspnea, be ready to perform intubation or tracheotomy at a moment's notice. Edema of the glottis is a great danger. Try to restore the fragments with one hand externally and with a finger in the mouth. Put the patient to bed and have him lie back upon a firm rest so that his shoulders are elevated. His head is to be placed between extension and flexion, a pasteboard splint or collar is molded on the neck, and a bandage is applied around the forehead, neck, and shoulders to keep the head immobile. The patient must not utter a word for a week; he must at first be fed by enemata, and then for some time on liquid diet, which is given through a tube early in the case. Endeavor to control the cough by opiates. A fractured hyoid bone requires about four weeks to unite.

**Fractures of laryngeal cartilages** are caused by direct violence, as throttling, blows, or kicks. They are rare in young persons, and are commonest when the cartilages have begun to ossify. They are very grave injuries, death tending to occur from obstruction to the entrance of air.

The *symptoms*, which are severe, are pain, aggravated by attempts at swallowing or speaking; swelling, ecchymosis it may be, and emphysema of the neck; cough; aphonia; intense dyspnea; and bloody expectoration if the mucous membrane is ruptured. There can be detected inequality of outline (flattening or projection) and perhaps moist crepitus. The usual seat of the injury is the thyroid cartilage.

*Treatment.*—Cases without dyspnea require quiet, avoidance of all talking, feeding with a stomach-tube, the application of compresses and adhesive strips over the fracture, and the use of remedies to quiet cough. The surgeon must be ready to operate at any moment. In most cases dyspnea exists, due to projection of the fragments or submucous extravasation. When there is dyspnea, emphysema, or spitting of blood, at once practise intubation, or, if unable to do this, open the larynx or trachea below the seat of fracture. If laryngotomy or tracheotomy is performed, try to restore displaced fragments to proper position. If the fragments will not remain reduced, introduce a Trendelenburg cannula or a tracheotomy tube, and pack gauze around it. Take out the packing in four days, and remove the tube as soon as the patient breathes well, when the opening may be allowed to close. In these cases feed with a stomach-tube and keep the patient absolutely quiet. Union takes place in four weeks.

**Fractures of the Ribs.**—The ribs, owing to their shape, elasticity, and mode of attachment, readily bend and as readily recover shape, and thus withstand considerable force without breaking. Notwithstanding these facts, the situation of the ribs so exposes them that in 16 per cent. of all cases of fractures noted by Gurlt these bones were involved. In children fracture of a rib seldom occurs and is usually incomplete; it is common in adults and the aged, and in them is generally complete. It is more frequent among men than among women. The ribs commonly broken are from the fifth to the ninth, the seventh being the one that most frequently suffers. Fracture of the first rib alone is an excessively rare accident. The eleventh and twelfth ribs are seldom broken. A rib may be broken in several places, and several

ribs are often broken at the same time. Fracture of a single rib is not nearly so common as fracture of several ribs. These fractures may be compound, either through the skin or through the pleura, a damaged lung permitting pneumothorax. Fractures compounded by a wound of the skin surface are very rare, except from bullet-wounds.

*Causes.—Direct* force, as buffer accidents, kicks, blows with heavy instruments, or being jumped on while recumbent, may produce these injuries. A fracture from direct violence occurs at the point struck, and the ends, projecting inward, may damage a viscus. *Indirect* force, as great pressure or blows which exaggerate the natural bony curves, tends to produce fractures near the middle of the ribs or in front of their angles and to force the ends outward. A number of ribs are apt to be broken. Muscular action, as in coughing, sneezing, lifting, or parturition, occasionally, but very rarely, is a cause.

*Symptoms.*—In connection with the history of the accident the symptoms are: acute localized pain (a stitch) on breathing, increased by pressure over the seat of pain, pressure backward over the sternum, cough, and forcible inspiration or expiration; respiration is largely diaphragmatic, the patient endeavoring to immobilize the injured side; cough is frequent and is suppressed because of pain. Crepitus is often, but not invariably, found. The surgeon seeks for it, first, by resting the palm of his hand over the seat of pain while the patient takes long breaths; second, by placing a thumb before and one behind the seat of pain and making alternate pressure; third, by auscultation. It should be remembered that incomplete fractures are the rule in children; hence in them do not expect crepitus. Deformity is usually trivial unless several ribs are broken, because shortening cannot occur and the intercostal attachments prevent vertical displacement. Preternatural mobility may occasionally be elicited, when the region is not deeply covered with muscles, by pressing on one side of the supposed break and observing that a part of, and not the entire, rib moves. If air gathers in the subcutaneous tissue and there is no wound of the surface, it is proof of rib fracture with lung damage. In such a case the lung has been penetrated by a fragment, and air has been forced out into the tissues. This condition is recognized by great and growing swelling, which crackles when touched. Such a collection of air is known as *cellular emphysema.* Bloody expectoration suggests lung injury; bloody expectoration and cellular emphysema, without an external wound, prove injury of the lung. A simple, uncomplicated case of fracture of a rib or ribs in a young person gives a good prognosis.

The *complications* are: additional injury, making the fracture externally or internally compound; laceration of the pleura, pericardium, heart, lung, diaphragm, liver, spleen, or colon; rupture of an intercostal artery; hemothorax; cellular emphysema; pulmonary emphysema; pneumothorax; pyothorax; traumatic pleurisy; pneumonia; bronchitis; congestion or edema of the lungs.

*Treatment.*—In an uncomplicated case the patient is not kept in bed, as breathing is easier when erect than when recumbent. Angular displacement outward is corrected by direct pressure. Displacement inward is soon corrected, as a rule, by the expansion of ordinary respiratory action; but if it is not thus corrected, etherize, the deep breathing of the anesthetic state almost always succeeding. If ether fails, and dangerous symptoms ensue, incise under strict antiseptic precautions, elevate, or sometimes resect a portion of the rib.

After correcting any existing deformity immobilize the injured side. If a man, shave the chest. Direct the patient to raise his arms above his head, to empty his chest of air by a forced expiration, and to keep it empty until a piece of rubber plaster (2 inches wide) is forcibly applied a number of inches below the fracture and from the spine to the sternum. The patient is now

allowed to take a breath and is directed to empty the chest again, another piece of plaster being applied, covering the upper two-thirds of the width of the first strip. This process is continued until the side is strapped well above and well below the fracture (Pl. 7, Fig. 13). Over the plaster light turns of a spiral bandage of muslin are carried, or a figure-of-8 bandage of the chest is applied, the turns crossing over the seat of injury. About once a week the plaster is removed and fresh pieces applied after rubbing the chest with soap liniment, drying, and anointing excoriations with an ointment of oxid of zinc. The dressing is worn for three or four weeks. The patient avoids cold, damp, and drafts. The diet must be nutritious but non-stimulating, and any cough should be treated by opiates and expectorants. A person with this injury who has reached the age of sixty must take stimulant expectorants (ammonii carb., 10 gr., in infus. senegæ, ½ oz., *t. i. d.*) or employ a steam-tent several times a day. The old method of treatment, in which the chest was included in a forcibly applied broad rib-roller, is not to be used except as a temporary expedient; it compresses the entire chest, causes pain and dyspnea, and tends to loosen and slip.

Fracture of the ribs complicated by visceral injury is highly dangerous, and requires confinement to bed. The treatment is that of the visceral injury. If there be bloody expectoration, apply adhesive strips as above indicated, put the patient to bed reclining on a bed-rest, keep him quiet, subdue the circulation, and employ opium, diaphoretics, and expectorants (a good mixture consists of squill, ipecac, ammonium acetate, and chloroform; opium is given separately). Inflammations of the lung or the pleura, fortunately, are apt to be localized, and are treated as ordinary inflammations of these parts. If signs of pulmonary injury are severe from the start or become worse under medical treatment, incise, resect a rib, arrest hemorrhage, and drain the pleura. In laceration of an intercostal artery incise and try to ligate; if unable to ligate, resect a rib and apply a ligature. If the signs point to internal bleeding, resect a rib, search for the bleeding point, and ligate. Emphysema usually soon disappears; but if it does not, make many small incisions in the cellular tissue, dress antiseptically, and employ pressure. When there arises a sudden attack of dyspnea, which is prone to happen in these cases, and in which the face becomes blue, the heart labors, and suffocation seems imminent, bleed the patient almost to syncope.

**Fractures of the costal cartilages** are not common, even in the aged. Such fractures occur either through the cartilages or through their points of junction with the ribs. These injuries generally arise from direct violence, the cartilage of the eighth rib being most prone to suffer. Bennett, of Dublin, has seen over 25 specimens of fracture of the first costal cartilage. Indirect force (such as a blow upon the shoulder) is occasionally the cause, but when it is the cause some other injury besides the fracture of the cartilages is apt to be noticed. Muscular action is a possible cause.

*Symptoms.*—Displacement is often absent; but if present, it is forward or backward of either fragment, and is due chiefly to the force of the injury, but partly, it may be, to muscular action. When displacement is absent, crepitus will not often be found; in fact, crepitus is usually absent in these injuries. Localized pain, swelling, and ecchymosis are noted. Preternatural mobility may or may not be detected. Union by bone is to be expected.

*Treatment.*—If displacement exists, try to reduce it. If the fragment is displaced backward, reduce by deep inspirations; if the fragment is displaced forward, reduce by pulling back the shoulders. In this attempt failure is the rule, and the surgeon may then adopt Malgaigne's expedient of applying a truss over the projection for a day or two. Dress and treat the case as if a rib were broken, dispensing with dressings in four weeks.

**Fractures of the Sternum.**—The sternum may be broken, along with the ribs and spine, from great violence. Fractures of the sternum alone are infrequent, because the bone rests on a spring-bed of ribs. Fractures of the sternum may be simple or compound, complete or incomplete, single or multiple. The most usual injury is a simple transverse fracture at or near the gladiomanubrial junction, at which point dislocation may also occur. Both fracture and separation of the ensiform cartilage are very rare. The sternum may be broken along with the ribs or clavicle.

*Causes.*—These are: *direct* force, as by a fall of an embankment or of a wall, by a car-crush, or by the passing of a cart-wheel over the body; *indirect* force, as by a fall upon the head, thus driving the chin against the chest; by a fall upon the feet, the buttocks, or the shoulder; by forced flexion or extension of the body over an edge or angle (as may occur during labor-pains).

*Symptoms.*—In fracture of the sternum displacement is not always present, but when it does occur the lower fragment is apt to pass forward; displacement may, however, be transverse or angular, or there may be overriding. The posterior periosteum, which rarely tears, limits displacement, but some deformity can, as a rule, be detected. The history of the nature of the accident has a valuable bearing upon the question of diagnosis. The position assumed by the patient is with the head and body bent forward, as attempts to straighten up cause much suffering. There is fixed and localized pain, increased by deep respiratory action, by body movements, or by cough. Crepitus is sought for by auscultation and by placing the hand over the injury and directing the patient to make quick respirations. Mobility may become manifest on external pressure, during respiration, or while attempts are being made to bring the body erect. Respiration in these cases is usually much interfered with. It is not important to separate diagnostically diastasis from fracture.

*Complications.*—Other fractures generally complicate fracture of the sternum, and laceration of the pleura or pericardium and hemorrhage into the anterior mediastinum may exist. Abscess of the mediastinum and necrosis of the sternum may appear as late consequences. The *prognosis* is good in uncomplicated cases.

*Treatment.*—The deformity attending fracture of the sternum is to be corrected, if possible, by external pressure. If overriding is found, effect reduction by bending the body back over a firm pillow and ordering the patient to respire deeply; if this method fails, give ether and then bend the body backward. The deformity, after reduction, tends to recur, but the bones unite well even in deformity, and no great harm results. The fragments need not be cut down on or be hooked up unless there be internal injury. After reducing the deformity, cover the front of the chest with adhesive strips extending laterally from one axillary line to the other, and covering a region from above the fracture down to the ensiform cartilage. Place over this covering an anterior figure-of-8 bandage of the chest. In some cases, where deformity recurs after reduction, a circular bandage of the chest is applied and the shoulders are pulled strongly back with a posterior figure-of-8 bandage. The plaster is to be reapplied once a week. Some surgeons treat these cases by means of a large compress held by adhesive plaster and a broad tight roller.

The patient goes promptly to bed, and reposes, erect or semi-erect, on a bed-rest. This position favors easy respiration and antagonizes the tendency to displacement. The diet should be light, nutritious, and non-stimulating. Convalescence is established in four weeks, and the plaster should be permanently removed in five weeks. When the ensiform cartilage is so bent in as to cause intense pain or to injure the stomach, it should be exposed

by incision and resected. Edema of the skin and fever, if they appear, indicate pus, in which case an incision should be made at the edge of the sternum and the pus-cavity should be irrigated and drained.

**Fractures of the Pelvis.**—In some of the indicated fractures serious injury of the pelvic contents is apt to be found.

**Fractures of the False Pelvis.**—Fractures of this region are seldom dangerous unless comminuted. There may be fracture of the iliac crest or of the anterior superior spine, or the line of fracture may traverse the entire length of the flanged-out ilium, or the bone may be comminuted with the association of grave visceral damage. The anterior superior and posterior superior spines may be broken off.

*Causes.*—The cause of fracture of the false pelvis is generally violent *direct* force, as the passage of a wagon-wheel, the fall of a wall, the kick of a horse or mule, or the force of car-crushes. Violent contraction of the rectus femoris muscle may tear off the anterior inferior spine of the ilium.

*Symptoms.*—In fracture of the false pelvis the history of violent force is noted. The patient leans toward the injured side. Pain exists, which is aggravated by movements (particularly by bending forward), by coughing, or by straining to empty the bowels or the bladder. Ecchymosis and swelling are manifest. Crepitus and preternatural mobility are detected by moving the iliac crest. Deformity is very rarely present. Cases uncomplicated by visceral injury make good recoveries.

*Complications.*—The fracture may be, but rarely is, compound, as the parts are well protected by muscles. The colon may be injured when comminution has taken place.

*Treatment.*—If there are symptoms of injury of the colon, perform laparotomy, search for the injured region, and suture it. In treating an ordinary fracture of the false pelvis put the patient on a fracture-bed, raise the shoulders, and apply a canvas binder about the pelvis, or encase the pelvis in broad pieces of rubber plaster, or employ the belt or girdle. The pressure of the binder, girdle, or plaster must not be so great as to force the fragment of ilium inward. Place the knees over two pillows so as to semiflex the legs and thighs, and tie the knees together. To restrain thigh movements it may be necessary to encase a restless patient in splints or bind him to sandbags. If the pelvic binder displaces the fragments or causes pain, abandon it and trust to position. If the fragment cannot be retained in place, wire it. The dressings can be removed in six weeks, and the patient is allowed to get up in eight weeks. In simple, uncomplicated fracture of the false pelvis the prognosis is good. In compound fractures of the false pelvis asepticize, drain and dress, put on a binder, and direct the same position to be maintained as for simple fractures.

**Fractures of the True Pelvis.**—The most usual seat of these fractures is through the obturator foramen, the ascending ischial and horizontal pubic rami being broken. A fracture may occur near the symphysis pubis, the symphysis may be separated, a line of fracture may run near to or into the sacro-iliac joint; the same may involve each side of the body of the pubis, and there may be multiple fractures. Fractures of the acetabulum and of the tuberosity of the ischium may occur. Before the seventeenth year the innominate bone may be broken into its three anatomical segments. Fractures of the true pelvis are highly dangerous because of the damage which is apt to be inflicted on the pelvic contents. There may be laceration of the bladder or membranous urethra, injury of the vagina, the rectum, the uterus, or the small gut. The *cause* of pelvic fracture is violent force, direct or indirect. Front force tends to produce direct, and side force indirect, fracture. The acetabulum may be broken by falls upon the feet.

*Symptoms.*—In pelvic fracture there is a history of violent force. There are great shock, ecchymosis which is possibly linear, swelling, and intense pain increased by attempts at motion, coughing, and straining. There is also inability to sit or to stand. Mobility becomes obvious on grasping an ilium in each hand and moving the hands. Crepitus may be noticed by this maneuver or by moving an ilium with one hand, a finger of the other hand being inserted in the rectum or vagina. In making movements for diagnostic purposes be very gentle, as rough manipulation may cause injury by sharp fragments. There may be doubt as to whether crepitus is to be referred to pelvic fracture or to fracture of the neck of the femur; in this case follow the rule of John Wood: "The surgeon grasps the femur with one hand and places the other firmly upon the anterior superior iliac spine or crest or upon the pubes; then, on moving the femur and abducting it freely, if a crepitus be detected, it will be felt the more distinctly by that hand which rests on or grasps the fractured bone."

Fig. 294.—Rugh's case of fracture of the acetabulum with internal dislocation of femur.

Rupture of the bladder is made manifest by pain in the hypogastric region, intense desire to micturate, inability to pass urine in quantity, although a few drops of bloody urine may be voided, great shock, sometimes dulness on percussion in the loins, and evidences of extravasation in the prevesical space. The condition is proved to exist by practising the maneuvers suggested under Rupture of Bladder. The symptoms of ruptured urethra are set forth later. Bleeding from vagina or rectum points to laceration of the part by a fragment. The vagina may be badly lacerated and the bowels may emerge from the laceration (Maurice H. Richardson's case). Intestinal injury is apt to induce septic peritonitis. Fractures of the acetabulum occur. Fracture of the brim of the acetabulum permits dorsal dislocation of the femur to occur, which dislocation will not remain reduced, and causes shortening, which at once recurs when extension is abandoned; also inversion and adduction, although the power of eversion and abduction is preserved (Stokes). There is crepitus, and the

head of the bone goes with the fragment upward and backward (Stokes). If the head of the femur be driven through the acetabulum into the pelvis, the injury is very grave; there are then found shortening, adduction, and semi-flexion of the thigh, absence of the prominence of the great trochanter, and more capacity for movement than is noted in dislocation. This injury is called *internal dislocation* of the femur (Fig. 294). Fracture of the ischium rarely occurs alone.

*Treatment.*—Examine carefully to determine if the bowel, the bladder, the urethra, or the vagina is injured. If such an injury exists, radical operation is, of course, demanded. Always use a catheter to see if the urine is bloody. Bloody urine suggests, but does not prove, the existence of a torn bladder. It may be due to simple contusion of the bladder or to contusion of the kidney. In treating a pelvic fracture endeavor to restore the parts to a normal position, employing external manipulation and inserting a finger in the rectum or in the vagina. If reduction is difficult, administer ether. The pelvis should be encircled by a canvas binder and the patient should be placed upon a Bradford frame. If this is done he can be cleaned readily and the bed-pan can be easily used. If movements of the thighs distort the pelvic bones, each thigh should be bound to the frame. In fracture with separation of the pubic bones the bones should be wired together. If urinary extravasation from urethral rupture occurs, perform perineal section. If there are signs of bowel injury or intraperitoneal rupture of the bladder, perform laparotomy; and if the bladder is found to be torn, apply sutures. All visceral injuries are treated by general rules. Remove the dressings in six weeks and allow the patient to get about in twelve weeks. In fracture of the acetabulum, if the limb is shortened, give ether and reduce by extension and counterextension. Treat these fractures in the same way as intracapsular fractures of the femur. Fractures of the ischium are best treated by the application of a pad and adhesive plaster, and rest in bed.

**Fractures of the Sacrum.**—This bone may be broken by direct force, such as a kick, but the injury is rare. The sacral plexus is usually injured, and if it is, paralysis is observed in the territory of its branches.

The *symptoms* of fracture of the sacrum are pain, frequently incontinence of feces and retention of urine, irregularity of the sacral spines, ecchymosis, and crepitus. Crepitus may be sought for with one hand externally and a finger of the other hand in the rectum. The lower fragment passes forward and may obstruct or may tear the rectum. Paralysis may be found in the area of distribution of the sacral plexus.

*Treatment.*—In any case of fracture of the sacrum, if there are evidences of pressure upon nerves by displaced bone, expose and elevate the depressed bone. If the rectum is lacerated, sutures must be inserted. In many cases of fracture of the sacrum the older conservative treatment is sufficient. The conservative treatment is as follows: Press the fragments into place with a hand externally and a finger in the rectum. Do not plug the rectum. Put a pad over the upper fragment, hold it by a plaster or a binder, place the patient recumbent on a fracture-bed, and insert a large cushion underneath the pad. Some surgeons give opium to induce constipation, and allow a fecal support to accumulate in the rectum. Use a clean catheter regularly, and guard against bed-sores. Union occurs in about four weeks, when the dressing can be removed. The patient can get about again in six weeks. If urinary retention persists, or if intractable bed-sores form after eight or ten weeks, cut down on the seat of injury and elevate or remove the portion of bone causing pressure.

**Fractures of the Coccyx.**—The coccyx may be broken or be separated from the sacrum by a fall, a blow, a kick, or the straining of parturition. Its mobility is so great, however, that it does not often break.

*Symptoms.*—The chief symptom of fracture of the coccyx is pain, which is much aggravated by sitting, walking, or straining at stool. If the index-finger is inserted into the rectum, the displaced bone is felt; if the thumb of the same hand is also placed externally, a rocking motion will develop crepitus and preternatural mobility.

*Treatment.*—In treating fracture of the coccyx reduce by external pressure and by the manipulations of a finger in the rectum and put the patient to bed. In four weeks the fracture should be united. If union does not take place, defecation and all movements of the coccyx will cause excruciating pain by pressure on the last sacral nerve. This condition is known as *coccygodynia.*

It must not be understood that coccygodynia always results from fracture of the coccyx, or that fracture of the coccyx is the only cause. As a matter of fact, coccygodynia is a rare condition and is seldom a result of fracture of the coccyx. It may arise after confinement, or a fall or blow upon the region of the coccyx, or may be due to caries.

In most cases it is a referred pain due to some central trouble, and is common in various functional disorders of the nervous system. It is especially common in hysterical, neurasthenic, or anemic women. In the traumatic neuroses it is commonly complained of and it is frequently associated with irritable spine. In very rare cases it is a neuralgia. The treatment is aimed at the causative condition. In very obstinate cases it demands a subcutaneous division of the nerve or of the muscles which move the coccyx, or a resection of the bone.

**Fractures of the Vertebræ.**—(See page 852.)

**Fractures of the Skull.**—(See page 789.)

**Fractures of the Clavicle.**—The clavicle is more often fractured than any other bone. The fracture may occur at any age, but is commonest before the sixth year (Hulke says one-half of the recorded cases). It may be simple, multiple, comminuted, oblique, transverse, complete, incomplete, or, very rarely, compound. Both clavicles may be broken. Fractures are most apt to occur just external to the middle, at the point where the inner or large curve meets the outer or small curve, at which junction the bone is at its smallest diameter. Fractures of the acromial end are more frequent than fractures of the sternal end, and less frequent than fractures of the shaft. The *causes* of fracture of the clavicle are direct violence, indirect violence, and, very rarely, the contractions of "the deltoid and clavicular fibers of the great pectoral" (Treves, in "Applied Anatomy," from Polaillon).

**Fractures of the shaft** are usually due to indirect violence, as falls upon the shoulder or upon the outstretched hand. In the latter accident, which is the usual mode of origin, the concusssion of the fall travels up and the body weight travels down, and these two forces compress the bone, which snaps at its weakest point. Fractures from indirect force are oblique, and in children are of the green-stick form. Fractures from direct force are usually transverse, and are occasionally comminuted. Fractures from muscular action have been recorded (Rubini, the tenor, recorded by Melay).

*Symptoms.*—In fracture of the shaft of the clavicle the attitude of the patient is peculiar. He supports the elbow or wrist of the injured side with the hand of the sound side, and also pulls the extremity against the chest; the head is turned down toward the shoulder of the damaged side, as if trying to listen to something in the joint, thus relaxing the pull of the sterno-cleidomastoid muscle upon the inner fragment. The shoulder is nearer the sternum, on a lower level, and farther front than that of the sound side. Loss of function is shown by inability to abduct the arm, and in many cases by inability to place the hand on the top of the erect head. Considerable pain exists, which is increased by motion, by pressure, and by the extremity hanging down without support.

The deformity above noted is described by stating that the shoulder goes downward, inward, and forward (D. I. F.). The *downward* deformity is chiefly due to the weight of the extremity, which pulls down the unsupported outer fragment, and is contributed to by the action of the pectoralis minor muscle. The *inward* deformity is chiefly due to the contraction of the pectoralis minor and subclavius muscles assisted by the action of the pectoralis major. The *forward* deformity is due to rotation of the outer fragment, which is brought about by the serratus magnus muscle carrying the scapula forward. In this deformity the inner end of the outer fragment is below and behind the outer end of the inner fragment, which overrides it. The inner fragment, though pulled on by the sternocleidomastoid muscle and relatively higher than the outer fragment, is really but little, if at all, elevated, marked elevation being prevented by the attachment of the rhomboid ligament. After noting the deformity, detect with the finger the irregularity of bony contour. Examine for preternatural mobility and crepitus by raising and throwing back the shoulder. In looking for these signs in children it is to be remembered that the fracture is probably incomplete. The prognosis is good, the bone uniting, but, if unoperated upon, with some shortening or inequality.

*Complications.*—Fractures of the shaft are rarely compound, because the sharp end of the outer fragment passes backward and because of the free play the skin makes over the bone (Pickering Pick). Both clavicles may be broken. One or more ribs may be fractured at the same time. In fractures from direct force deeper structures may be injured by fragments. Thus, injury of the brachial plexus will induce paralysis. There are 11 recorded cases of simple fracture of the clavicle complicated by laceration of a large vessel. Eight of these cases died. The vessel ruptured may be the subclavian vein, the subclavian artery, or the jugular vein. After a rupture a huge blood-clot forms (Gallois and Poillet, in "Rev. de Chir.," July and Aug., 1901).

Fig. 295.—Fox's apparatus for fractured clavicle.

*Treatment.*—In treating a fracture of the shaft of the clavicle correct the deformity as soon as possible by throwing the shoulder upward, outward, and backward. If the patient is a girl, it is desirable to minimize the deformity. Place her upon her back upon a hard bed, with a small pillow under her head, a firm and narrow cushion between the shoulders, a bag of shot resting over the seat of fracture, and the forearm lying on the front of the chest, the arm being held to the side by a sand-bag. In three weeks there will be union, practically without deformity. In a child with an incomplete fracture a handkerchief sling for the forearm, worn three weeks, is all that is needed. In a fracture of the collar-bone of an adult the Velpeau bandage is usually efficient. Before applying it, place lint around the chest and cotton over the elbow. Change the bandage every day for the first week, and after that period every third day. Each time it is changed rub the skin with alcohol, ethereal soap, or soap liniment, dry carefully, and examine for excoriations; if any are found, they are anointed with zinc ointment before the dressing is reapplied. The dressing is permanently removed at the end of four weeks, the arm being carried in a sling for

another week. The classical apparatus of Desault is now rarely used. The posterior figure-of-8 bandage associated with the second roller of Desault, some turns being made from the elbow of the injured side to the shoulder of the sound side, can be used in cases in which the forward deformity is apt to return. The apparatus of Fox, which is very comfortable and useful, consists of a pad for the axilla, a sling for the forearm, and a ring for the opposite shoulder, to which ring are tied the tapes from both the pad and the sling (Fig. 295).

The dressing of Moore, of Rochester, is valuable in an emergency. It consists of a piece of cotton cloth, 2 yards long, and folded like a cravat until it is 8 inches in width at the middle. The center of the bandage rests upon the elbow, the posterior tail is carried across the front of the shoulder of the injured side. The forearm is at an acute angle with the arm, and the other end of the bandage is carried across the forearm, across the back over the opposite shoulder, and around the axilla, where the extremities are stitched together. The forearm is suspended in a bandage sling (S. D. Gross, in "A System of Surgery"). The four-tailed bandage is preferred by Pick. Sayre's dressing has many advocates (Fig. 296). For this there are required two pieces of rubber plaster, each piece being 3 inches wide and sufficiently long to go around the chest one and a half times. The end of one piece encircles the arm of the injured side just below the arm-pit; the plaster strip is pulled across the back to the other side, to the front of the chest, and returns again to the middle of the back. This procedure pulls the elbow back and throws the shoulder out. The hand of the injured side is placed on the breast of the opposite side, cotton being interposed, and the second strip of plaster runs from the elbow of the injured side to the front of the opposite shoulder, around, and back, pressing the elbow forward, upward, and inward. In children, if it is found difficult to immobilize the parts, the most satisfactory result is obtained by the application of the Velpeau bandage, which is overlaid by a thin plaster-of-Paris bandage. If the fragments cannot be coaptated, sterilize the parts, administer ether, incise, clear away the muscle from between the fragments, saw the ends, bore each end and hold them in contact by means of kangaroo-tendon or silver wire. The same procedure should be pursued when a fracture is compound or threatens to become so, or if signs indicate pressure upon vessels or nerves. If a large vessel has been injured, the operation is imperatively necessary. If a patient suffering under a fracture which threatens to become compound refuses the aid of operation, keep him in bed and hold the arm in abduction. In a number of cases I have wired the fragments with excellent results. Year by year I become more inclined to recommend wiring in cases of fractured collar-bone. It secures union without deformity, saves the vessels and nerves, and obviates the necessity of prolonged and very uncomfortable fixation of the arm and forearm.

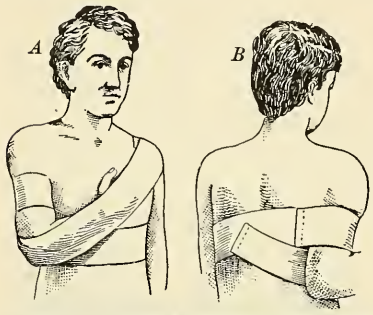


Fig. 296.—Sayre's adhesive-plaster dressing for fracture of the clavicle (Stimson): *A*, First piece; *B*, second piece.

After a broken collar-bone has united, if the shoulder is found to be stiff, make passive movements daily; if these fail, move the joint forcibly, first giving ether or nitrous oxid.

**Fractures of the acromial end of the clavicle** are due to direct force. If the fracture is between the two coracoclavicular ligaments, deformity is very slight, crepitus is elicited by manipulating with the fingers, and pain exists, but

loss of function is not markedly manifest unless it is due to pain. These fractures are treated by interposing cotton between the arm and the side, binding the arm to the side with the second roller of Desault, and hanging the hand in a sling. In fractures external to the ligaments crepitus is manifest on moving the shoulder, the outline of the bone is irregular, severe pain is developed by movement, and deformity is pronounced. The deformity is due to the serratus magnus muscle rotating the scapula forward, the inner end of the outer fragment of the clavicle often coming in contact with the anterior surface of the outer portion of the inner fragment. Fracture of the acromial end of the clavicle is reduced by pulling both of the shoulders strongly backward, and it is kept reduced by the use of a posterior figure-of-8 bandage. In fracture external to the ligaments the displacement frequently cannot be corrected by position and manipulation. Such cases demand incision and wiring. In either variety of fracture the dressings are worn for four weeks.

**Fractures of the sternal end of the clavicle** are very rare. They are caused by either direct or indirect force. In such a fracture there are found crepitus, projection at the seat of fracture, rigidity of the sternocleidomastoid muscle, and shortening of the clavicle. The inner end of the outer fragment always passes forward, and often also downward and inward. Reduce these fractures by pulling the shoulders back, and treat them by means of the posterior figure-of-8 bandage worn for four weeks. Wiring may be necessary.

**Fractures of the Scapula.**—This bone is not often broken, as it rests upon thick muscles and elastic ribs; it is freely movable, and it has attached to it a bone which easily breaks.

**Fractures of the Body of the Scapula.**—These are due to direct violence. The *symptoms* are pain (which becomes agonizing on attempting to rotate the shoulder-blade), ecchymosis, and swelling. Crepitus is sought for by placing the hand over the bone and making movements of the arm: also by holding the point of the shoulder and lifting up the lower angle of the bone. The latter plan may develop mobility. The spine of the scapula is uneven only when it is itself fractured. Examine for unevenness of the vertebral border of the shoulder-blade. In fractures of the body of the scapula a shoulder-cap is applied, a gutta-percha splint is molded over the scapula, the arm is bound to the side, and the hand is carried in a sling. The apparatus is worn for four weeks.

**Fractures of the spine of the scapula** are treated as fractures of the body of the bone, and for the same time.

**Fractures of the Neck of the Scapula.**—Fracture of the *anatomical neck* has not been proved to exist. Fracture of the *surgical neck* is evinced by flattening of the shoulder, prominence of the acromion, and the presence of a lump in the axilla, crepitus being developed by pressing the axillary prominence upward and backward. The coracoid process descends with the humerus. The deformity is reduced with ease, but it at once recurs. The condition is treated by placing a pad in the axilla, a shoulder-cap on the shoulder, applying the second roller of Desault, and supporting the forearm and elbow in a sling. A Velpeau dressing can be used, associated with the application of a folded towel in the axilla. The dressing is to be worn for five weeks.

**Fractures of the glenoid cavity** are not very unusual, and may occur with or without dislocation. Fracture of this region arises from direct force applied to the shoulder. The existence of this fracture is determined by excluding fractures of other bones and by detecting crepitus when the arm is at a right angle to the body and the humerus is pushed against the glenoid cavity, the crepitus not being found when the arm hangs by the side.

*Treatment* is by the second roller of Desault and a forearm sling worn

for four weeks; careful passive movements limit ankylosis. If ankylosis occurs, adhesions must be broken up while the patient is under ether or nitrous oxid.

**Fractures of the acromion process** are often met with as the result of direct violence. The existence of fracture of the acromion is indicated by pain, by inability to abduct the arm, by flattening of the shoulder, by sudden lowering of the point of the shoulder, by mobility, and by crepitus. To treat a case of this kind, put a large pad with the base down in the axilla, bind the arm over the pad with the second roller of Desault, lifting the elbow with turns of the roller carried over it and the opposite shoulder, thus splinting the bone in place by the head of the humerus pushing against the coracoacromial ligaments. The dressing is to be worn for four weeks.

**Fractures of the coracoid process** rarely happen alone, and may arise from direct force or from muscular action. But little displacement is found. Crepitus and mobility are usually detected. Inability to shrug the shoulder inward was pointed out as a symptom by Byers. Such a case is well treated by a Velpeau bandage, which is to be worn for four weeks.

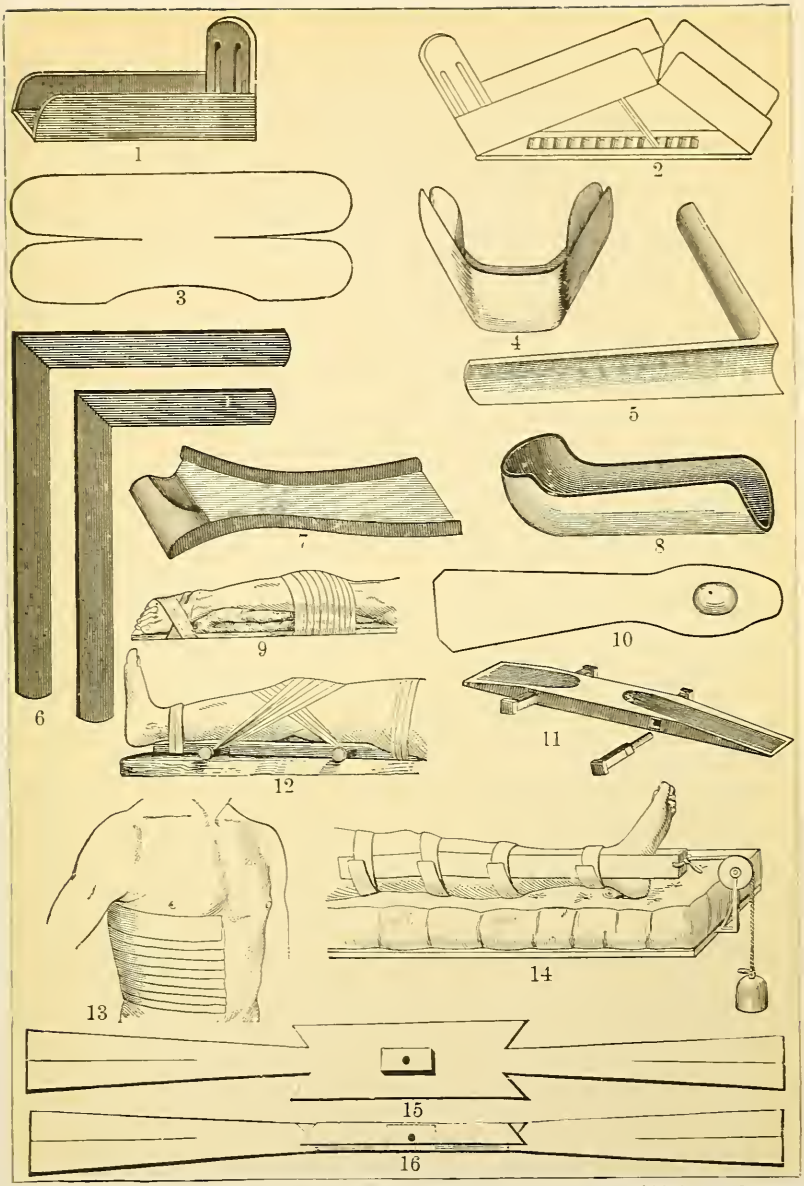
**Fractures of the humerus** are divided into: (1) fractures of the upper extremity; (2) fractures of the shaft, and (3) fractures of the lower extremity. In examining any fracture of the humerus, feel at once for the pulse, so as to ascertain if the artery has been torn; in any fracture near the head of the humerus be certain that dislocation does not exist.

*Examination of the Shoulder.*—In some cases ether must be administered. Compare the injured shoulder with the sound shoulder, the patient, if not anesthetized, being seated on a chair or stool. The direction of the axis of the arm is noted. The surgeon grasps the flexed elbow with one hand and the shoulder with the other; he thus can move the extremity and palpate the joint and adjacent points. The shoulder is moved gently in every direction, and the surgeon notes if the head of the bone moves with the shaft. Examination shows if the head of the bone is in place or if the glenoid cavity is vacant—if the head of the bone is in an abnormal situation, if it is altered in contour, if there is crepitus or preternatural mobility, and if any movement is impaired. The acromion process, outer end of the clavicle, coracoid process of the scapula, and neck of the scapula are also investigated. The length of the arm is obtained by measuring from the apex of the acromion process of the scapula to the apex of the external condyle of the humerus, and it is compared with the length of the sound extremity.

**Fractures of the upper extremity of the humerus** include: (*a*) fractures of the anatomical neck; (*b*) fractures of the surgical neck; (*c*) fractures of the head, oblique and longitudinal, and (*d*) separation of the upper epiphysis.

**Fractures of the Anatomical Neck of the Humerus.**—The anatomical neck is the constricted circumference of the articular surface, and fractures of it, though rare, do occur, especially in the aged. The line of fracture in some cases follows the insertion of the capsule, in others it is entirely within the capsule, but in most it is without the capsule above and within the capsule below; hence the term "intracapsular" is rarely correct as a designation. Such a fracture may be impacted. The *cause* is direct violence or a fall or a blow upon the elbow when the arm is abducted. Polloson, of Lyons,[1] has reported a case due to muscular action. The patient died in eclampsia, and at the necropsy it was found that both humeral heads were fractured and impacted. The fractures must have been produced by the muscles throwing the heads of the bones violently against the glenoid cavities, probably by adduction.

[1] "Rev. de Chir.," vol. viii, 1888.




1. Fracture-box. 2. Double Inclined Plane Fracture-box. 3. Jaw-cup (unfolded). 4. Jaw-cup (folded). 5. Anterior Angular Splint. 6. Internal Angular Splint. 7. Bond Splint. 8. Shoulder-cap. 9. Dupuytren Splint in Pott's Fracture. 10. Agnew Splint for Fracture of the Metacarpus. 11. Agnew Splint for Fracture of the Patella. 12. Agnew Splint applied. 13. Strapping the Chest in Fractured Ribs. 14. Extension Apparatus in Fracture of the Femur. 15, 16. Adhesive Strips for Extension Apparatus.

The *symptoms* of fracture of the anatomical neck are pain, tendinous swelling, ecchymosis, slight irregularity of the shoulder (which irregularity is soon hidden by tumefaction), and inability to actively abduct the arm. Deformity, as a rule, is slight or is absent, because the capsule is rarely entirely torn from the lower fragment. If deformity exists, it is due to the muscles inserted on the bicipital groove and to the coracobrachialis, which pull the lower fragment inward and forward. Treves says that a tear of the reflected fibers of the capsule leads to subsequent necrosis of bone because this joint has no ligamentum teres. In unimpacted cases there is crepitus, and mobility of the shaft can be detected near the head of the bone. In some cases impaction occurs, the upper fragment impacting into the lower. In this condition there are very slight shortening and trivial shoulder-flattening, no crepitus unless the tuberosity is broken off, no mobility, and, as Erichsen says, the head of the bone, while it can be felt through the axilla, is not in the axis of the limb.

Fig. 297.—Fracture at upper end of the humerus. Note hand, forearm, and elbow bandaged; axillary pad and strap, plaster-of-Paris shoulder-cap, sling (Scudder).

Fig. 298.—Fracture at upper end of the humerus. Arm and elbow bandaged. Axillary pad and shoulder-cap in position. Application of circular bandage to trunk and shoulder. Sling not shown (Scudder).

The *prognosis* of fracture of the anatomical neck is usually good for bony union (Hamilton, Pick, and R. W. Smith), but a stiff joint is apt to result.

*Treatment.*—Feel the pulse to be sure the artery is untorn. In most cases an anesthetic should be given in order to examine with ease and dress with satisfaction. Sometimes the fragments are readily coaptated; occasionally they are not. In a case reported by Carl Beck the axes of the fragments were at right angles and they could only be kept in contact by holding the arm at a right angle to the body ("New York Med. Jour.," April 5, 1902). Albee, of New York, reported a series of these fractures treated by wiring and maintaining the arm at a right angle with the body. The result was complete preservation of function. Some surgeons treat this fracture by simply hanging the wrist in a sling and suspending a bag of shot from the elbow to make extension. The usual plan of treatment is as follows: abduct the arm to a right angle with the body, and carry up from the base of the fingers to above

the elbow the turns of a spiral reversed bandage made of flannel. Interpose lint between the arm and the side, and place a V-shaped pad with the apex upward in the axilla, tying the tapes over the opposite shoulder. A shoulder-cap made of pasteboard (Pl. 7, Fig. 8) or plaster of Paris (Fig. 297), molded to fit and well lined with cotton, is applied. The plaster-of-Paris cap is the most satisfactory. It is applied "so as to cover the whole shoulder, the anterior and posterior aspects of the chest, and the outer side of the upper arm down to the external condyle of the humerus" (Scudder, on "The Treatment of Fractures") (Fig. 297). The arm with the shoulder-cap is fixed to the side by the second roller of Desault, and the wrist is hung in a sling (Fig. 298). The edges of the bandage should be stitched together. This apparatus is changed daily for the first few days, the body and arm being rubbed at each change with alcohol, soap liniment, or ethereal soap. After this period a change every third or fourth day is often enough. Massage is begun at the end of one week, but rotation and motion of the joint are not employed until after three weeks. The dressings are removed at the end of four weeks, the forearm being carried in a sling for two weeks more. In impacted fracture do not pull apart the impaction, do not use a pad, but apply a cap to the shoulder and fix the arm to the side for five weeks. The fracture unites with deformity.

**Fractures of the Surgical Neck of the Humerus.**—The surgical neck is the constricted portion of bone between the tuberosities and the upper line of the insertion of the muscles on the bicipital groove. Fractures in this region are usually transverse, but they may be oblique. The *causes* are: direct force almost always; indirect force occasionally; and muscular action in rare instances.

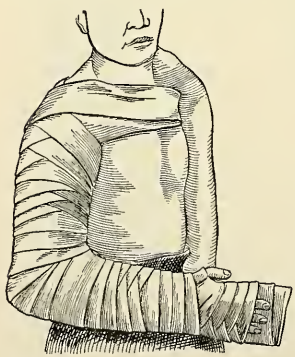


Fig. 299.—Internal angular splint and shoulder-cap in fracture of the surgical neck of the humerus.

The *symptoms* in fracture of the surgical neck are: pain running into the fingers from pressure upon the brachial plexus; crepitus; mobility on extension; and flattening, which differs from the flattening of dislocation in that it occurs farther below the acromion and that this process is not so prominent. Shortening to the extent of an inch is noted. The head of the bone can be felt in the glenoid cavity, but it does not move on rotating the arm. The upper end of the lower fragment is felt and moves on rotating the arm. The displacement is pronounced. The lower fragment is pulled upward by the deltoid, biceps, coracobrachialis, and triceps; inward by the muscles of the bicipital groove, and forward by the great pectoral; thus, the upper end of the lower fragment projects into the axilla, and the elbow lies from the side and backward. Péan holds that the violence drives the lower fragment forward. The upper fragment is abducted and rotated outward, which position is due, it is generally taught, to the action of the supraspinatus, infraspinatus, and teres minor muscles. In some cases displacement is forward, and in other cases it is not obvious. The lower fragment may impact into the upper, in which case the symptoms are obscure and the diagnosis is made by exclusion. If the impaction is solid and complete, there are the history of direct force, the impaired movements, the slight deformity, and the absence of crepitus. In all fractures of the upper end of the humerus the distinction can be made from dislocation by feeling the head of the bone under the acromion and by noting that it does not move on rotating the arm.

The *prognosis* of fracture of the surgical neck of the bone is good.

*Treatment.*—Some surgeons treat a fracture of the surgical neck in exactly the same manner as a fracture of the anatomical neck. I prefer the following plan: In many cases give ether in order to examine and dress. Feel the pulse to see that the artery has not been damaged. Reduce by traction and manipulation; if there is an impaction, pull it apart. Take an internal angular splint (Pl. 7, Fig. 6) and pad it well, putting on extra padding at the points that are to rest against the palm, the inner condyle, and the axillary folds. Lay the arm and pronated forearm upon the splint. Apply a padded shoulder-cap. Fix the splint and cap in place with a spiral reversed bandage terminating as a spica of the shoulder, and hang the hand or forearm in a sling (Fig. 299). The dressing is to be worn for four weeks, and the rules to be followed in changing it are the same as in fracture of the anatomical neck. Massage is used after one week, and passive motion to amend stiffness after four weeks. In rare cases—those with strong anterior projection of the lower end of the upper fragment—apply an anterior angular splint. In some cases when the deformity strongly tends to recur support by a plaster-of-Paris trough on the back and sides of the arm and shoulder, or maintain extension by weights and pulleys, the patient being kept in bed (Stimson). In a case with great deformity abduction with extension is a very useful method. I have reached the conclusion that many cases of fracture of the surgical neck are best treated by incision and fixation.

Fig. 300.—Fracture of humerus below surgical neck.

Fig. 301.—Preliminary splinting for complete dressing as shown in Fig. 304.

**Longitudinal and Oblique Fractures of the Head of the Humerus.**—By this term may be designated separation of the great tuberosity or separation of a portion of the articular surface, together with the great tuberosity, from the shaft and lesser tuberosity (Pickering Pick, Guthrie, and Ogston). The *cause* is usually direct violence to the front of the shoulder, but the greater tuberosity may be torn off by muscular action.

The *symptoms* in longitudinal and oblique fracture of the head are broadening and flattening of the shoulder with projection of the acromion. The upper fragment passes upward and outward, and the lower fragment passes

upward and inward to rest on the margin of the glenoid cavity below the coracoid process. The elbow is drawn from the side, there is some shortening, and the patient cannot abduct his arm. If the surgeon grasps the patient's elbow and holds it to the side and rotates the arm while with his other hand he grasps the upper fragment, crepitus is very positive. Examination develops wide separation of the fragments. The deformity cannot be entirely corrected, because the biceps tendon is apt to get between the fragments (Ogston), but a useful limb can usually be obtained.

Fig. 302.—Linear fracture of humerus.

*Treatment.*—The plan which gives the best result in treating longitudinal and oblique fracture of the head of the bone is to place the patient on his back upon a hard bed with a small, firm pillow under his head, abduct the arm above the head, rotate it outward so that the back of the hand rests on the bed, and hold it in place by sand-bags. This position should be maintained for three weeks, at the end of which period the fracture can be treated for three weeks more as a fracture of the anatomical neck. If the patient refuses to go to bed, treat the injury as a fracture of the anatomical neck, padding well over the tuberosities. The dressings should be worn for five weeks, passive motion being made after four weeks. In the above injury feel at once for the pulse, to see if the artery has been torn.

**Separation of the Upper Epiphysis of the Humerus.**—The epiphysis is united during the twentieth year. Separation is a rare accident and is produced by direct force.

*Symptoms.*—The chief symptom in separation of the upper epiphysis is projection of the upper end of the lower fragment inward, forward, and upward beneath the coracoid, and consequently a projection of the elbow backward and from the side. If the lower fragment passes forward and not inward, the elbow simply passes back. The upper end of the lower fragment is smooth and convex. Rotation of the shaft develops soft crepitus when the fragments are in contact.

The *prognosis* is good for bony union, though the future growth of the limb may be impaired.

The *treatment* for separation of the upper epiphysis is a pad in the axilla, a shoulder-cap, binding the arm to the side, and hanging the hand in a sling. Wear the dressing for four weeks, and begin passive motion as directed when dealing with fracture of the upper end of the humerus.

**Fracture of the shaft of the humerus** is a very common accident. The *cause* is usually direct violence, such as a blow. The fracture may arise from indirect violence, such as a fall upon the elbow. Muscular action is not rarely also a cause, as in throwing a ball, in catching a tree-limb while falling, or in turning another's wrist outward as a test of strength (Treves). This test of strength is known by the French as "le tour de poignet."

The opponents sit opposite to each other and each rests his elbow on a table. They clasp hands and each one strives to rotate the other's hand outward. Ashhurst collected 57 cases due to throwing a ball and 23 cases due to "le tour de poignet." He believes that in some cases the humerus is broken as a stick may be broken by holding one end and swinging the other through the air and that in other cases fracture results from twisting (Astley P. C. Ashhurst, in "University of Pennsylvania Med. Bulletin," Feb., 1906). In fracture due to muscular action the break is nearly always below the deltoid insertion and the line of fracture approaches the transverse.

The *symptoms* of fracture of the shaft of the humerus are pain, tendinous swelling, ecchymosis, inability to move the arm, mobility, and distinct crepitus. Shortening to the extent of ¾ inch occurs. The displacement varies with the situation of the fracture and the direction of the force. If the fracture is above the insertion of the deltoid, the lower fragment is pulled up by the triceps, biceps, and deltoid, and pulled out by the deltoid, and the upper fragment is pulled inward by the arm-pit muscles. In fracture below the deltoid this muscle is apt to pull the lower end of the upper fragment outward, while the lower fragment passes inward and upward because of the action of the biceps and triceps. Injury of the *musculospiral nerve* sometimes occurs. The nerve may be contused, producing pain at the seat of bruising, and tingling and numbness in the region supplied by the nerve. In most cases the symptoms soon pass away, but sometimes neuritis ensues. A severe contusion produces not only pain, but paralysis of the muscles supplied by the nerve, and surface anesthesia. In most cases this condition is recovered from in a few weeks, but sometimes it lasts a long while or even permanently. In musculospiral paralysis the patient is unable to extend the wrist and fingers or to supinate the forearm. There is "complete loss or impaired sensation in the lower half of the outer and anterior aspect of the arm and in the middle of the back of the forearm as far as the wrist" (Scudder, in "The Treatment of Fractures"). The nerve may be divided by a sharp fragment, paralysis of motion and anesthesia resulting at once. In some cases the nerve is caught in and compressed by callus, scar-tissue, or fragments, motor and sensory disturbances resulting.

Fig. 303.—Fracture of middle of humerus.

Fig. 304.—Apparatus for fracture of the humerus at any point above the condyles.

The *prognosis* is good, but the fact should always be remembered that ununited fractures are commoner in the humerus than in any other bone. Treves believes this to be due to entanglement of muscle between the fragments, lack of fixation of the shoulder-joint, and imperfect elbow support. Hamilton believes that it is due to the facts that the elbow soon becomes fixed at a right angle, and that any movement of the forearm moves the seat of fracture and not the elbow.

*Treatment.*—It is rarely necessary to anesthetize unless the patient be a nervous woman or an excitable child. Feel the pulse, to be certain the artery has not been lacerated. Reduce the fracture by extension, counterextension, and manipulation. Apply four humeral splints (Fig. 301). The internal splint

reaches from the axilla to just above the internal condyle of the humerus. A short straight splint is applied front and another back, each being the length of the arm. A shoulder-cap is applied, which cap "is prolonged below into an external angular splint reaching as far down as the lower third of the forearm" (Fig. 304) ("Manual of Surgical Treatment," by Cheyne and Burghard). The elbow is brought to a right angle with the arm and the forearm is placed midway between pronation and supination. As Cheyne and Burghard say: "It is necessary that the arm should hang vertically at the side with the long axis of the forearm parallel with the anteroposterior diameter of the trunk; if the forearm be brought at all forward across the chest, rotation of the lower fragment upon its vertical axis is apt to take place." Splints are to be worn for five or six weeks, and after the removal of the splints the wrist is hung in a sling. The sling is dispensed with eight weeks after the infliction of the injury. Passive movements are not to be made until the

Fig. 305.—Fracture of the shaft of the humerus. Note bandage to hand, forearm, and elbow; axillary pad and strap; coaptation splints and sling. Bandage does not cover fracture (Scudder).

Fig. 306.—Fracture of the shaft of the humerus. Note bandage to hand, forearm, and elbow; adhesive-plaster swathe holding arm upon axillary pad and covering coaptation splints. Sling (Scudder).

fracture is well united (after five or six weeks), for, if made too soon, they predispose to non-union, and, as no joint is involved, genuine ankylosis will not occur. Many surgeons treat these fractures by applying plaster of Paris to the forearm and the arm (the elbow being flexed to a right angle), binding the arm to the side, and hanging the wrist in a sling. Others apply a trough to the arm and forearm. Scudder prefers to bandage the hand, forearm, and elbow, and apply an axillary pad, coaptation splints, a swathe of adhesive plaster holding arm to the side, and a sling (Figs. 305, 306). In any case in which it is impossible to obtain and maintain correct apposition of the fragments, cut down upon them, and apply a plate. If the nerve is divided, an incision must be made at once, the nerve sutured, and the bone plated. If the nerve is caught in the callus, after repair has taken place it must be liberated by chiseling the callus away. Neuritis is treated by blisters over the nerve, the use of the descending galvanic current, and the administration of salicylate of ammonium and the bromids.

**Fractures of the Lower Extremity of the Humerus.**—These fractures are spoken of as fractures in, or in the neighborhood of, the elbow-joint, and they include: (*a*) fractures of the external condyle; (*b*) fractures of the internal condyle; (*c*) fractures of the internal epicondyle; (*d*) fractures at the base of the condyles; (*e*) T- or Y-shaped fractures; (*f*) epiphyseal separation, and (*g*) fractures of the capitellum and trochlea. There may be more than one fracture, or there may be also a dislocation of the humerus, of the ulna, or of both bones. Rarely the fracture is compound. These fractures are frequent injuries in childhood, and are not uncommon in adults.

*Method of Examination.*—A fracture of the elbow is rapidly followed by great swelling, and the diagnosis is often very difficult. In most cases, when possible, the *x*-rays should be used in arriving at a diagnosis. In every case in which the *x*-rays are not used, and in most cases in which they are, the surgeon examines the parts carefully while the patient is under ether. If swelling is very great, it is necessary to abate it in order to reach any conclusion as to the condition. We can bandage the arm, rest it semiflexed on a pillow, and apply evaporating lotions or even an ice-bag for a day or two, or, what is better, temporarily diminish the swelling by Gerster's plan, which is as follows: Apply an Esmarch bandage from the hand to well above the seat of fracture; this will drive away extra-articular swelling and permit of thorough examination. It is a great advantage to have the patient anesthetized, for then not only can we make an accurate diagnosis, but we can reduce the fracture satisfactorily and apply a careful first dressing.

Compare the injured with the sound elbow. Note swelling and local ecchymosis. Feel the radial pulse. Note the "carrying angle" (Fig. 308). Measure each arm from the tip of the acromion process of the scapula to the tip of the external condyle of the humerus. Feel each prominent bony point and note if it is mobile (condyles, olecranon, head of ulna). Feel the shaft just above the condyles. Mark with ink on each elbow the tip of the external condyle, the tip of the internal condyle, and the tip of the olecranon, and observe the relation between these points of each elbow in flexion and in extension. In an uninjured elbow a straight line transverse to the long axis of the limb with the joint in extension will pass through the condyles and leave the tip of the olecranon just a shade above it. "When the elbow is at a right angle, these three points will be found in the same plane with the back of the upper arm" (Scudder, in "The Treatment of Fractures"). Rotate the radius while a thumb is held against the head of the bone. Make flexion and extension of the elbow and determine if there is any lateral motion. Test for mobility just above the condyles. The above maneuvers will determine the presence or absence of crepitus, preternatural mobility, deformity, etc.

**Fractures of the External Condyle of the Humerus.**—A fracture of the external condyle runs into the joint and the capitellum is usually broken off. Such an injury occurs oftenest in children, being due to falling on the hand; but it may occur from direct force, and may happen to adults.

The *symptoms* of fracture of the external condyle are severe pain, great swelling, and crepitus (found on pressing or moving the condyle and on rotating the radius). Mobility may also be discovered. A projection is felt on the outer and posterior surface of the elbow. The forearm is semiflexed and supinated. The patient cannot use the joint.

**Fractures of the Inner Epicondyle of the Humerus.**—The inner epicondyle is an epiphysis which unites during the seventeenth year. It not infrequently breaks from muscular action or from direct violence, and the fracture does not involve the joint. Crepitus and mobility can be detected. Displacement is slight. The *outer epicondyle* is never fractured alone.

**Fractures of the Internal Condyle of the Humerus.**—The line of fracture after a break of the internal condyle runs into the joint, to the trochlear surface of the humerus. The *cause* is nearly always direct violence. Packard, of Philadelphia, observed a case in which the condyle had been torn off while lifting a tub.

*Symptoms.*—In fracture of the internal condyle the fragment, accompanied by the ulna, goes upward and backward, and when the forearm is extended the ulna projects posteriorly, the lower end of the humerus being felt in front. The fragment forms a projection back of the elbow. Crepitus and preternatural mobility can be found if swelling is not too great. Crepitus is detected by flexing and extending the forearm. The space between the condyles is broader than normal, and the forearm takes a bend toward the ulnar side, the "carrying function" of the forearm being lost (Fig. 307). When a person carries a heavy object, such as a bucket of coal, he instinctively rests the inner condyle upon the pelvis, and the normal deviation of the forearm outward keeps the bucket from striking the leg. This deviation outward when the inner condyle rests against the ilium gives us the carrying function. In fracture of the inner condyle the broken condyle ascends and the "carrying function" is lost (Fig. 308). This deformity is known as *gunstock deformity.*

Fig. 307.—Loss of "carrying function" after fracture of inner condyle of the humerus.

**Fractures at the Base of the Condyles of the Humerus** (Figs. 309 and 310).—A fracture in this region is just above the level of the tip of the olecranon and is on a higher level behind than in front. The *cause* is direct force acting upon the olecranon.

The *symptoms* are loss of function, and pain from injury to the median or ulnar nerve. Crepitus and mobility are readily found. The lower fragment is drawn backward and upward by the action of the triceps, biceps, and brachialis anticus muscles. The lower end of the upper fragment projects in front of the joint. This lesion may be mistaken for dislocation of the bones of the forearm backward. In fracture the limb is mobile; in dislocation, it is rigid. In fracture the deformity is easily reduced and strongly tends to recur; in dislocation the deformity is reduced with difficulty and does not tend to recur. In dislocation there is shortening of the forearm, but not of the arm; in fracture there is shortening of the arm, but not of the forearm. In dislocation there is a smooth, large projection below the crease in front of the elbow; in fracture there is a sharp projection above the crease. In fracture there is crepitus; in dislocation there is no crepitus.

The *diagnosis* can be settled by the *x*-rays.

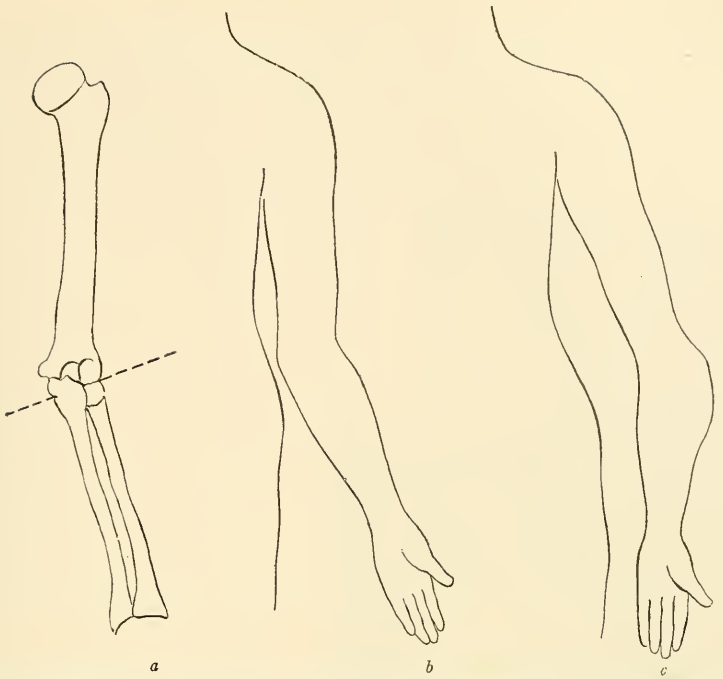



Fig. 308.—Diagram to exhibit the "carrying function" of the forearm, and the loss of this function in fracture of the inner condyle of the humerus: *a* and *b* show the normal relation of the parts when carrying; *c* shows the alteration of axis of the forearm when the inner condyle is fractured, what is known as gunstock deformity resulting (after Allis).

**T-fractures of the Humerus.**—A T-fracture consists of a transverse fracture above the condyles plus a vertical fracture between them. The *cause* is violent direct force applied posteriorly.

The *symptoms* are increase in breadth of the joint (Fig. 311), preternatural mobility, crepitus, pain and swelling, mounting up of the inner condyle back of the elbow on the inner side, and of the outer condyle back of the elbow on the outer side. The forearm is semiflexed and supinated, and the carrying function is lost.

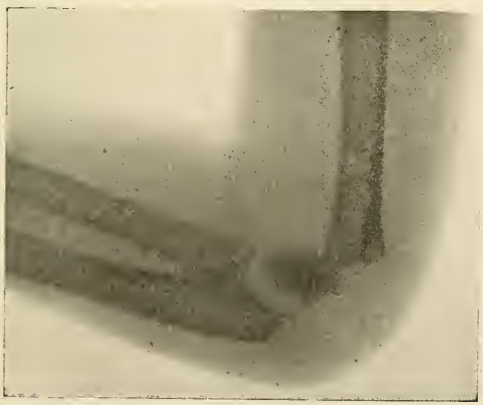


Fig. 309.—Supracondylar fracture of the humerus.

Fig. 310.—Fracture of the humerus above the condyles.

**Prognosis of Fractures In or Near the Elbow-joint.**—In many fractures it is difficult or impossible to obtain reduction, and in some it is impossible to maintain reduction. Stimson is undoubtedly right when he says that "in intercondyloid fracture with marked separation there is no practicable means merely to maintain reduction."[1] In fracture in or about the elbow-joint I have regarded the prognosis for complete restoration of function as poor, and believed that in most of these fractures some deformity and considerable stiffness are inevitable. Of late, however, influenced largely by Astley P. C. Ashhurst's study of "Fractures of the Lower End of the Humerus" ("Samuel D. Gross Prize Essay of Phila. Acad. of Surgery," 1910), I have been studying this question anew, and as a result take a less gloomy view of the situation, and am disposed to agree with Ashhurst that "in the vast majority of cases the ultimate results will be perfectly satisfactory." Nevertheless I am still cautious as to what I can safely predict to the patient and the patient's family. Ankylosis, partial or complete, is always a possible sequence. Ankylosis may result from prolonged immobilization, the muscles contracting and becoming fibrous, the fascia and ligaments about the joint shortening, the capsule shrinking and thickening, some of the cartilages becoming fibrous, and the joint being partly obliterated. It may result from extravasation of blood into the joint and tendon-sheaths with subsequent formation of fibrous tissue. It may arise from organization of inflammatory exudate within and about the joint and in the sheaths of muscles and tendons. It may arise from the formation of an excess of callus. Bruns claims that in fracture in the joint excess of callus rarely forms, and that masses of callus form chiefly in the line of fracture near but not in a joint.[2] Excessive callus formation is sure to take place if reduction is not thoroughly accomplished or if the fragments are not well immobilized, but move upon each other. A mass of callus in or about a joint limits or prevents motion. The two greatest causes of impaired function are blocking by callus and stiffness from traumatic arthritis (Jones, of Liverpool).

Fig. 311.—Deformity following fracture of the humerus between the condyles.

**Treatment of Fractures In or Near the Elbow-joint.**—Thoroughly set the fracture while the patient is under ether. It is advisable, when it can be done conveniently, to use the *x*-rays to confirm the diagnosis and to use them again after dressings have been applied, to be sure that the bones remain in good position. If swelling is very great, it may be necessary to delay setting for two or even three days, the arm being bandaged and laid upon a pillow or lightly supported on an anterior angular splint during the waiting period.

In all cases except transverse fracture above the condyles and fracture of the olecranon reduction is best effected by drawing upon the forearm, supinating it, extending it, and then bending it slowly into a position of acute flexion, the degree of flexion being in inverse ratio to the amount of swelling.

[1] "Transactions American Surgical Association," vol. ix.
[2] Max Oberst, in Volkmann's "Sammlung Vorträge."

In transverse fracture above the condyles reduction is effected by drawing the forearm and the lower fragment downward and forward and at the same time pushing the upper fragment back.

Some surgeons advocate dressing the fracture on an anterior angular splint, the forearm being fully supinated (Fig. 312). The advantage claimed for this splint is that if ankylosis occurs the joint is in a position to be useful, which it is not if ankylosed in extension. Some deformity is usually apparent after treating a case with this splint; the deformity following fracture of the inner condyle is not corrected by it, but if the splint is carefully applied the result is usually a useful extremity in all cases except fracture of the inner condyle. In transverse fracture of the shaft of the humerus above the condyles the anterior angular splint is the best method of treatment, as it prevents displacement. The splint must not be applied when there is great swelling, and swelling must be removed by resting the extremity on a pillow, the elbow being semiflexed, applying evaporating lotions or even an ice-bag, employing massage, and gently compressing by bandaging. In some cases the joint should be aspirated. In order to apply this dressing, take a right-angled splint and pad its outer surface, being careful to place thick, soft pads over the convexity which will press in front of the elbow and over each end of the splint. Fasten the upper end to the arm, then make extension of the forearm, and if the fracture is found to be well reduced, fasten the hand and forearm to the splint (Fig. 312). If the hand and forearm are first fixed to the splint, there will be no extension from the elbow and deformity will result. If posterior projection exists, a pasteboard cup is molded over the elbow. The extremity is hung in a triangular sling. At night the extremity is kept in the sling or laid on a pillow. Every third or fourth day, while the extremity is carefully steadied, the splint is removed, the arm and forearm well rubbed with alcohol, massaged, and the splint reapplied. The splint is worn between five and six weeks. At the end of the third week, after removing the dressings, slightly flex, slightly extend, and slightly pronate the forearm, and reapply the splint. At the end of the fourth week repeat this maneuver, making movements of greater range. In the middle of the fifth week and at the end of the fifth week do it again, and flex and extend as much as possibe. Very early and very frequent passive motion is objectionable, as it leads to overproduction of callus and ankylosis, but passive motion as above described is imperatively necessary. Many surgeons at the end of the second week apply a Stromeyer splint, which permits the patient and the surgeon to make some motion by means of the screw without removing the dressings. In very stout persons an anterior angular splint will not stay in place. In such a case the forearm may be placed at a right angle to the arm and plaster of Paris be used. After the dressings are removed employ passive motion, massage, hot and cold douches, baking, inunctions of ichthyol or mercurial ointment, iodin locally, corrosive sublimate and iodid of potassium internally, and direct the patient to systematically use the arm. If in any case after four weeks non-union exists, put up the arm in a plaster splint for three or four weeks more. Some surgeons use a posterior right-angled trough instead of an anterior angular splint (Fig. 304).

Fig. 312.—Anterior angular splint for fractures in or near the elbow-joint.

Allis warmly advocates treating fractures in and about the joint in extension. He holds that the extended position secures the best circulation, and if either condyle is unbroken secures the benefit derivable from a natural splint. Furthermore, in fractures of the inner condyle it restores the carrying function, which the flexed position does not do. For one week after the accident the patient stays in bed, with his arm extended upon a pillow. After swelling subsides the limb is wrapped firmly in a spiral flannel bandage and plaster of Paris is rubbed in or the bandage is covered with plaster.

Some surgeons extend the limb and apply an ordinary plaster bandage, and in about three weeks substitute an anterior angular splint. The trouble with treatment in extension is that if ankylosis ensues the limb is nearly useless. Furthermore, treatment in extension requires confinement to bed.

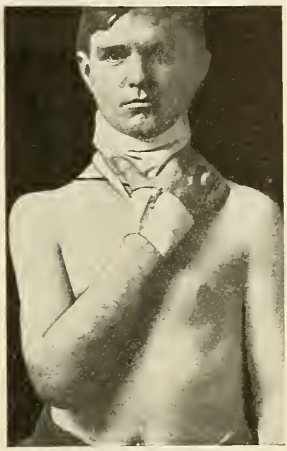


Fig. 313.—Jones's dressing for injuries of the elbow-joint.

Jones, of Liverpool, thinks that splints and bandages are largely responsible for the stiffness which so commonly ensues upon an elbow injury. He advocates treatment by supination and acute flexion in all elbow injuries except fracture of the olecranon. It has been demonstated that the position of acute flexion forces the fragments into place and holds them firmly between the coronoid process of the ulna, the trochlear surface of the ulna, the fascia, and the triceps tendon. The surgeon must be certain that the radial pulse is perceptible *after* the elbow has been flexed. Flexion is maintained by fastening a bandage around the wrist and neck. The bandage around the neck passes through a rubber tube, which serves to protect the neck. The ball of the thumb should rest against the neck. The bandage is fastened to a leather band around the wrist or to a glove, the fingers of which have been cut off. The most convenient dressing to maintain Jones's position is shown in Fig. 313.

After the dressing has been applied certain precautions are to be observed. For the first week or ten days look at the arm daily. If the swelling grows worse, diminish the degree of flexion, and do the same if there is severe pain. If the radial pulse disappears, diminish the flexion until free circulation is obtained. This position is maintained from three to six weeks.[1] After the first two weeks lower the wrist an inch or two. At the end of three weeks make a little passive motion (just one movement in each direction). Passive motion and massage are applied as if an anterior splint were being used. The author has treated many cases by Jones's method, and now prefers it to any other plan in all fractures of the elbow except fracture of the olecranon, transverse fracture above the condyles, fracture of the inner condyle near the line of the ulnar nerve, and fracture between the condyles in which the coronoid process gets between the fragments in flexion. The first-mentioned injury must be dressed in extension, the transverse fracture above the condyles requires an anterior angular splint, and the other two injuries should be treated in extension. If a fracture near the line of the ulnar nerve is treated in acute flexion, the callus poured out will be apt to entangle and press upon the nerve.

If it is found impossible to reduce the fragments or to maintain reduction we should make an incision and nail or screw the fragments in place. A comminuted fracture requires operation.

In *young children* the anterior angular splint must not be used. It will

[1] "Provincial Medical Jour.," Dec., 1894, and Jan., 1895.

become loosened, and motion will inevitably take place at the seat of fracture. Such cases can be treated satisfactorily in Jones's position, the arm being held to the chest by plaster-of-Paris bandages, or they can be treated in extension. Bertomier's plan is very useful in young children.[1] The extremity is dressed without pressure in extension and supination. This can be effected by flannel bandages. In from four to eight days a silicate of sodium bandage is applied in order to prevent pronation. About the sixteenth day the bandage is cut so as to form two troughs. From this period every third day the splints are removed and gentle passive motion is made. The splints are removed permanently at the end of four weeks.

If *false ankylosis* follows fracture of the elbow, the adhesions should be broken up under ether, and for some time the hot-air apparatus should be used daily, and massage, passive motion, and the hot and cold douche should be employed. In *true ankylosis* an operation should be performed and the interlocking callus or the interposed tissue or fragment be removed, if a skiagraph shows that operation promises success. If gunstock deformity results and produces marked disablement, it should be operated upon. An osteotomy is performed on the inner condyle. The arm is set in the extended position, plaster of Paris applied, and is not removed for six weeks.[2]

Fig. 314.—Fracture between the condyles treated by Jones's position. Degree of voluntary flexion obtained.

**Separation of the lower epiphysis of the humerus** is a not unusual accident. The inferior extremity of the humerus may be separated, or the condyles may be separated from each other and from the shaft of the bone.

The *symptoms* are prominence in front of the joint, caused by the lower end of the shaft of the humerus; projection backward of the olecranon; the forearm rests midway between pronation and supination. Epiphyseal separation may retard growth and produce deformity.

*Treatment.*—Jones's position or an anterior splint as above directed.

**Fractures of the ulna** comprise the following varieties: (1) fracture of the coronoid process; (2) fracture of the olecranon process; (3) fracture of the shaft, and (4) fracture of the styloid process.

**Fractures of the coronoid process of the ulna** (Fig. 316) are rarely observed, and practically occur only as a complication of backward dislocation of the ulna or in association with other fractures.

*Symptoms.*—When fracture of the coronoid process is associated with a dislocation, crepitus is appreciated on reduction, and it is found that the deformity of the dislocation promptly returns on cessation of extension. The upper fragment may be pulled upward by the brachialis anticus muscle, and there exists an inability to flex the forearm completely. The position is one of extension with posterior projection of the olecranon. The broken piece is felt in front of the joint.

The *treatment* is by an anterior splint the angle of which is less than a right angle. Jones's position may be used in treating such a case. A stiff joint may follow.

[1] "Révue de Chir.," vol. viii, 1888. [2] G. G. Davis, "Phila. Med. Jour.," May 13, 1889.

**Fractures of the olecranon process of the ulna** occur not uncommonly in adults. Hulke states that such a fracture never occurs before the age of fifteen, but the writer has seen in the Jefferson Medical College Hospital a girl aged fourteen with a fractured olecranon. The *cause* is direct violence or muscular action. Only a small fragment may be torn away, or the entire

Fig. 315.—Fracture between the condyles treated by Jones's position. Degree of voluntary extension obtained.

olecranon may be broken off, and the break may be comminuted or may even be compound.

The *symptoms* of most fractures of the olecranon are: swelling; partial flexion of the forearm; separation of the fragments, the upper piece being pulled up from ½ inch to 2 inches by the triceps; the space between the fragments is

Fig. 316.—Fracture of coronoid process.

increased by flexion at the elbow, and lessened by extension at the elbow; and there is inability to extend the arm. Bulging of the triceps and crepitus on approximating the fragments are observed. In some few cases there is no separation, the periosteum being untorn or the fascial expansions from the triceps holding the fragments in apposition. In such cases crepitus can be elicited by rocking the upper fragment from side to side.

When treated by non-operative methods the *prognosis* is usually fair, fibrous union being the rule. Some joint stiffness usually occurs, and much ankylosis may be unavoidable. The prospect of a freely movable joint is better when extra-articular wiring is practised.

*Treatment.*—Fracture of the olecranon is usually treated with a well-padded anterior splint almost, but not quite, straight. A perfectly straight splint is uncomfortable, and by opening a retiring angle between the fragments and into the joint favors non-union and ankylosis. The splint should reach from a level with the axillary margin to below the fingers. If the upper fragment does not come in contact with the lower, pull it down by adhesive plaster and fasten the strips to the splint. The author in 1 case employed a glove to which strings from the adhesive plaster were attached. After applying the splint keep the patient in bed for three weeks. The danger of ankylosis in this fracture is very great, and, in case it occurs in the position of exten-

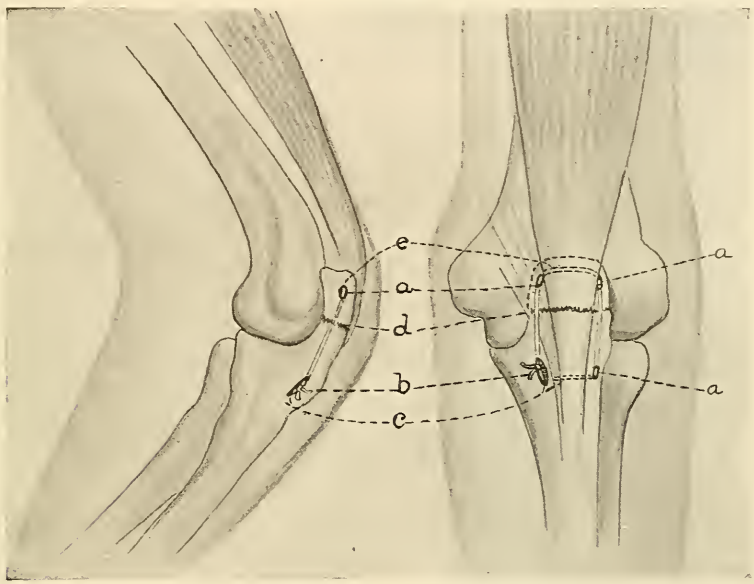



Fig. 317.—Murphy's method of treating fracture of the olecranon by subcutaneous exarticular wiring. Lateral view and posterior view. Wiring the fragments of the olecranon together: *a*, Incision; *b*, twist or tie of wire; *c*, hole drilled in bone for passage of wire; *d*, fracture; *e*, passage of wire through tendon of triceps (Murphy).

sion, an almost useless arm results. Follow the rule of T. Pickering Pick, and at the end of three weeks anesthetize the patient, press the thumb firmly down upon the top of the olecranon, carry the forearm slowly to a right angle, and apply an anterior angular splint and direct it to be worn for two weeks. When the anterior splint has been applied, passive motion should be made every other day, or every third day, and massage should be used at the same time. When the splint is removed, try to increase the range of motion as previously directed. Surgeons usually incise and apply wires only when it is found impossible to secure apposition of the fragments after fracture of the olecranon. Such a course is, I am persuaded, injudicious conservatism. I do not advise that the rule should be to treat fractures of the olecranon as a routine by opening and wiring, but I do advise that we should treat them by extra-articular operation and wiring as advocated by John B. Murphy ("Jour. Am. Med. Assoc.," Jan. 27, 1906). The conservative non-operative treatment

often fails. Sometimes the fragments cannot be approximated, frequently they cannot be maintained in approximation, not unusually a stiff or actually ankylosed joint results. Murphy thus describes the operation which should be done ("Jour. Am. Med. Assoc.," Jan. 27, 1906) (Fig. 317): "A longitudinal incision $\frac{1}{3}$ inch long was made on the external aspect of the ulna, $\frac{1}{2}$ inch from its articular surface, and tissues were divided to the bone. A smaller incision was made on the corresponding inner side. I perforated the base of the olecranon with an eyelet drill, which ran transversely from outward inward. I threaded the drill with a fine aluminum-bronze wire, drawing it through this transverse canal. The wire was carried upward under the skin on the inner surface of the elbow and then drawn out through another small incision, $\frac{1}{16}$ inch, made at the level of the apex of the olecranon. The wire was then reinserted and directed transversely from inward outward, passing it through the tendon of the triceps above the olecranon, and then drawn out to corresponding outward point through a very small incision similar to that made on the inner side. The wire was again reinserted and pushed downward under the skin until it was finally brought out through the initial external incision. The circle once completed, traction was exerted on the wire until I was sure that the two fragments were in perfect coaptation, the latter being easily and satisfactorily accomplished. The ends of the wire were twisted several times and then divided by scissors close to the bone. By this procedure the skin was incised at four points, the largest incision being $\frac{1}{3}$ inch in length." The extremity is placed in flexion upon an anterior splint, which is worn for four weeks. Passive motion is begun on the third day. A compound fracture and a comminuted fracture always require operation, in which the joint is freely opened. Non-union requires opening of the joint and wiring of the fragments.

Fig. 318.—Fracture of the shaft of the ulna (case in the Pennsylvania Hospital; skiagraphed by Dr. Gaston Torrance).

**Fractures of the shaft of the ulna** alone are most usual near the middle of the bone, are always due to direct violence, and are not infrequently compound. An injury which breaks the ulna is very apt to break the radius also.

*Symptoms.*—By running the finger along the inner surface of the bone there are detected inequality and depression; crepitus and mobility are easily developed; there are pain and the evidence of direct violence. The long axis of the hand is not on a line with the long axis of the forearm, but is internal to it. If deformity exists, it is due to the lower fragment passing into the interosseous space because of the action of the pronator quadratus; the upper fragment, acted on by the brachialis anticus, passes a little forward (Fig. 318). The

forearm at and below the seat of fracture is narrower and thicker than normal.

*Treatment.*—In treating fracture of the shaft of the ulna place the forearm midway between pronation and supination, so as to bring the fragments together and to obtain the widest possible interosseous space, and thus limit the danger of union taking place between the radius and ulna. The position midway between pronation and supination is obtained by flexing the forearm to a right angle with the arm and pointing the thumb to the nose. Take two well-padded straight splints, one long enough to reach from the inner condyle to below the fingers, the other from the outer condyle to below the wrist; place a long pad of lint over the interosseous space on the flexor side of the limb, and another on the extensor side; apply the splints and hang the forearm in a triangular sling (Fig. 319). Passive motion is to be begun in the third week, and the splints are to be worn for four weeks. Fractures of the ulna can be treated very efficiently by plaster of Paris. The best results are secured by wiring or plating.

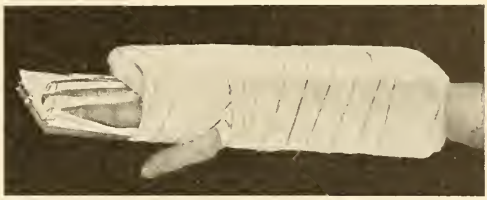


Fig. 319.—Two straight splints in fracture of both bones of the forearm.

**Fractures of the styloid process of the ulna** are due to direct force. The displacement is obvious.

*Treatment.*—In treating fracture of the styloid process push the fragment back into place and use a Bond splint with a compress for four weeks, or apply a plaster-of-Paris dressing.

**Fractures of the radius** include the following varieties: (*a*) Fractures of the head; (*b*) fractures of the neck; (*c*) fractures of the shaft, and (*d*) fractures of the lower extremity.

**Fracture of the head of the radius** has been studied by Dr. T. Turner Thomas ("University of Penna. Med. Bulletin," Oct., 1905; and "Annals of Surgery," August, 1907). He has furnished me with the following résumé of his views, with which I entirely agree:

"Fracture of the head of the radius is not infrequent, and is usually the result of a fall on the hand with the elbow in extension and the forearm in pronation. In extension of the elbow the capitellum of the humerus is in contact with only the anterior part of the radial head. Since the carpus articulates almost entirely with the radius, the force of the usual fall on the hand is transmitted almost entirely through this bone, and at the elbow is received by only the anterior part of the radial head. According to the degree of violence applied more or less damage may be done to the head, or head and neck. The anterior portion of the rim may be split off, the intact ulna preventing the humerus from pursuing the detached fragment and pushing it away from its fellow, and the intact orbicular ligament holding the two in close apposition. This uncomplicated fracture is the most common, but the least troublesome. Union with ultimately good function is the rule, even though the fracture go unrecognized and untreated, because of the close splinting of the untorn orbicular ligament. Since it is the same accident, a fall on the outstretched hand, which usually produces Colles's fracture, fracture of the external condyle of the humerus, fracture of the neck of the radius, fracture of the coronoid process, and posterior dislocation of both bones of the forearm, any one or several of these injuries may complicate the fracture of the radial head. When the anterior part of the head breaks off the bony resistance to the descent of the capitellum by the radius is lost, so that if the force of the accident

is severe enough, lateral bending of the elbow to the radial side with further descent of the capitellum may result with greater separation of the fragments, tearing of the orbicular ligament, and more damage to the head. The descent of the condyle in a posterior dislocation may carry before it the detached fragment of the head, and this fragment be left buried in the tissues of the forearm several inches below its normal position after the dislocation has been reduced.

"*Prognosis.*—In the vertical or oblique fissured fracture of the head with close approximation of the fragments, after union takes place, limitation of all movements of the elbow results from adhesions and slight irregularities in the bone. The function rapidly returns in the succeeding weeks, although some limitation of extension will continue for months. More marked irregularity in the circumference of the head may last much longer and may produce permanent limitation of rotation. Bony union between the head and corresponding surface of the ulna will prevent all rotation of the forearm. In either case flexion and extension usually return almost if not completely. Non-union of the fragments may induce pain and limitation of movement, especially rotation; but with close approximation of the fragments and an untorn orbicular ligament, an apparently perfect return of function may follow, the fragments moving smoothly as one piece within the ligament. The prognosis will depend chiefly upon the degree of damage done to the head and the separation of the fragments.

"*Symptoms.*—The most characteristic feature of this fracture is its obscurity. The thick muscular covering of the radial head, except posteriorly, the splinting effect of the orbicular ligament, and the close contact of the head with the humerus and ulna, make the diagnosis of the small intra-articular fracture particularly difficult. In the uncomplicated vertical fracture there will usually be no movements of the fragments on each other, and, therefore, no crepitus, and there will be no deformity. A history of a fall on the hand; some swelling of the elbow, particularly on the radial side; severe pain and limitation in all movements of the elbow, particularly in rotation; pain and tenderness distinctly localized to the head of the radius; and the exclusion of fracture of the humerus, ulna, and the shaft of the radius, will point strongly to a fracture of the head of the radius. If the injury is treated as a fracture, and two or three weeks later, when fixation is removed, there is marked limitation of all movements of the elbow, the diagnosis will be more than reasonably assured. The *x*-ray may be misleading unless directed in the line of the fracture, and this is not easy to accomplish, since the position of the fragments in their relation to the humerus vary with every change in rotation of the forearm. In most cases an exposure to the *x*-ray in the transverse plane of the humeral condyles, with the forearm in pronation, will give a successful skiagraph. If the *x*-ray is directed at right angles to the line of fracture the skiagraph will usually be negative. If the fragments are freely separated and if crepitus is elicited, the diagnosis will be more easily determined.

"*Treatment.*—In the uncomplicated fracture without crepitus or deformity, Jones's position or a right-angled splint for three weeks will be sufficient. The resulting fibrous ankylosis will largely disappear from forced use in the following weeks, but several months will be necessary before extension is complete. If crepitus is present, four or five weeks' fixation will be better, and in this case the return of function will probably take longer. As a rule, if union is obtained function will return. If pain on movement persists for many months, a detached fragment or the whole head should be excised. Marked limitation of movement from enlargement of the head or bony union between it and the ulna calls for excision of the head."

**Fracture of the neck of the radius** (Fig. 320) is by no means so rare an accident as was thought before the discovery of the *x*-rays. It seldom occurs

alone and is usually associated with fracture of the radial head. These fractures are transverse and frequently impacted. The cause is a fall upon the pronated hand when the forearm is extended.

*Symptoms.*—In this fracture the forearm is pronated and the patient is found to have the lost power of voluntary pronation and supination. Under forced pronation and supination it will be noted that the head of the radius does not move unless there is impaction. Crepitus is sometimes absent because of impaction. Thomas points out that there is angulation of the neck due to driving of the radial head downward and forward ("Annals of Surgery," August, 1907). Reduction is always difficult and may be impossible.

The *treatment* for fracture of the neck of the radius is the same as for other fractures of the elbow-joint—namely, an anterior angular splint or Jones's position.

**Fracture of the shaft of the radius** (Fig. 321) is far commoner than fracture of the shaft of the ulna. It may occur above or below the insertion of the

Fig. 320.—Fracture of the neck of the radius.

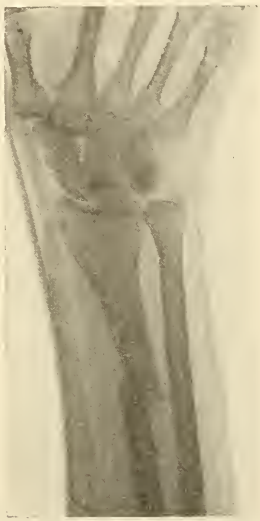


Fig. 321.—Fracture of radius alone.

pronator radii teres muscle. It may arise from either direct or indirect force. Fracture of the shaft of the ulna may coexist as a result of the same accident.

**Fracture of the Shaft of the Radius Above the Insertion of the Pronator Radii Teres Muscle.**—*Symptoms.*—The upper fragment is drawn forward by the biceps and is fully supinated by the biceps and the supinator brevis. The lower fragment is fully pronated by the pronator quadratus and pronator radii teres, and its upper end is pulled into the interosseous space. There are crepitus, mobility, pain, narrowing and thickening of the forearm below the seat of fracture, and loss of the power of pronation and supination. The head of the bone is motionless during passive pronation and supination. The hand is prone.

*Treatment.*—In treating this fracture do not put the forearm midway between pronation and supination, as this position will not bring the fragments into contact, the upper fragment remaining flexed and supinated. To bring the lower fragment in contact with the upper, flex and fully supinate the forearm. Apply an anterior angular splint to the extremity for four weeks, and begin passive motion in the third week.

**Fracture of the Shaft of the Radius Below the Insertion of the Pronator Radii Teres Muscle** (Fig. 321).—In this variety of fracture the upper fragment is acted on by the biceps, the supinator brevis, and the pronator radii teres, and it remains about midway between pronation and supination, passing forward and also into the interosseous space. The lower fragment is acted on by the supinator longus and the pronator quadratus, the latter being the more powerful of the two, hence the lower fragment is moderately pronated, its upper extremity being drawn into the interosseous space. Other symptoms are identical with those of fracture above the insertion of the pronator radii teres.

*Treatment.*—In treating fracture below the pronator radii teres the forearm is flexed and is placed midway between pronation and supination; two interosseous pads and two straight splints are applied as for fracture of the ulna (see Fig. 319). The splints are worn for four weeks, and passive

Fig. 322.

Fig. 323.

Figs. 322, 323.—Fracture of both bones of the forearm.

motion is begun in the third week. Plaster of Paris is a most satisfactory dressing. Loss of function is best obviated in this fracture by incision and fixation.

**Fracture of the shafts of both bones of the forearm** (Figs. 322–326) is not frequently seen. It is caused either by direct or indirect force. If due to indirect force the radius breaks first.

*Symptoms.*—After fracture of both bones of the forearm the hand is pronated, and the two lower fragments come together and are drawn upward and backward or upward and forward by the combined force of flexor and extensor muscles, shortening being manifest and the projection of the lower fragments being detected on either the dorsal or the flexor surface of the forearm. The upper fragment of the ulna is somewhat flexed by the brachialis anticus; the

upper fragment of the radius is flexed by the biceps and is pronated and drawn toward the ulna by the pronator radii teres. The forearm is narrower than it should be (the ends of the fragments having passed into the interosseous space) and is thicker than normal from front to back (the contents of the interosseous space having been forced out). Crepitus, mobility, pain, and inequality exist, the power of rotation is lost, and on passive rotation the head of the radius does not move. The forearm is prone and semiflexed.

*Treatment* of this fracture usually consists in the application of two straight splints and two interosseous pads, the forearm being flexed to a right angle and placed midway between pronation and supination (see Fig. 319). The splints are worn for four weeks, and passive motion is begun in the third week. Instead of these splints a plaster-of-Paris dressing can be used. I am persuaded that pronation and supination are best preserved by incision and fixation (Figs. 325 and 326).

Fig. 324.—Fracture of both bones in the lower third of the forearm.

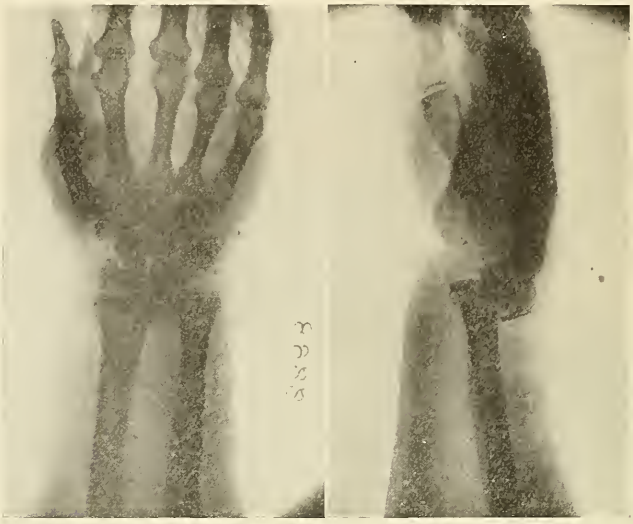
Fig. 325.

Fig. 326.

Figs. 325, 326.—Ununited compound comminuted fracture of both bones of forearm (*x*-ray by Dr. Senders).

**Fractures of the Lower Extremity of the Radius.**—*Colles's fracture* is a transverse or nearly transverse fracture of the lower end of the radius, between the limits of $\frac{1}{4}$ inch and $1\frac{1}{2}$ inches above the wrist-joint, the lower fragment sometimes mounting upon the dorsum of the upper fragment (Fig. 327), the two fragments sometimes impacting (Fig. 328). An oblique fracture beginning within $\frac{1}{2}$ inch of the joint and passing into the joint is known as *Barton's fracture* (Fig. 329). Colles's fracture was first recognized as a fracture by Colles, of Dublin, in 1814. Before this time the injury was called backward dislocation of the wrist. It is a very common injury, is met with most frequently in those beyond the age of forty, and oftener in women than in men. It is due to transmitted force (a fall upon the palm of the pronated hand). Some think that the force is received by the ball of the thumb and passes to the carpal bones and the edge of the radius; a fracture beginning posteriorly rather than anteriorly and the force driving the lower fragment upon the dorsal surface of

the radius, the carpus and lower fragment moving upward and outward. It is much more likely that this fracture is due to cross-strain on the bone. There



A B

Fig. 327.—Colles's fracture before reduction: A, Anteroposterior; B, lateral views.

is sudden traction upon the anterior ligaments, which drag upon the bone and break it at a point where the cancellous end of the radius joins the compact shaft (Fig. 330). The fragments are not unusually impacted (Fig. 328). In the author's experience dislocation of the lower end of the

Fig. 328.—Impacted Colles's fracture.

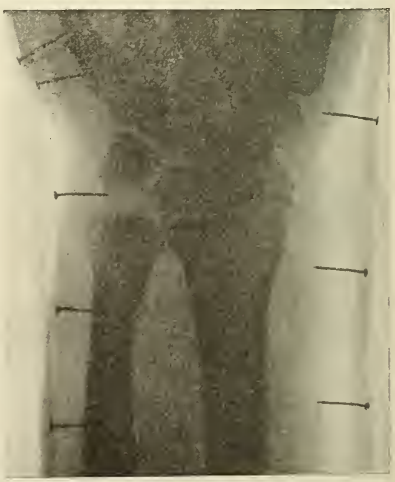


Fig. 329.—Barton's fracture.

ulna is not a very unusual complication. It arises from a fracture of the ulnar styloid or tearing off of the internal lateral ligament of the wrist.

*Symptoms.*—In Colles's fracture the hand is abducted (drawn to the radial side of the forearm) and pronated, the head of the ulna is prominent, the styloid process of the radius is raised, and the lower fragment may mount on the back of the lower end of the upper fragment, causing a dorsal projection, termed by Liston the "silver-fork deformity" (Figs. 331, 332). The lower end of the upper fragment can be felt beneath the flexor tendons above the wrist. The position in deformity is produced by the force. Some consider it is maintained by the action of the supinator longus and the flexor and extensor muscles, but particularly by the extensors of the thumb. Pilcher has demonstrated the fact that in this fracture a portion of the dorsal periosteum is untorn, and this untorn portion acts as a binding band to hold the fragments in deformity. Pronation and supination are lost. In this fracture the hand can be greatly hyperextended (*Maisonneuve's symptom*). Crepitus, which is best obtained by alternate hyperextension and flexion, can be secured unless swelling is great or impaction exists. Crepitus on side movements is rarely obtainable. Impaction may greatly modify the deformity, though displacement generally exists to some extent, and the fragments do not ride

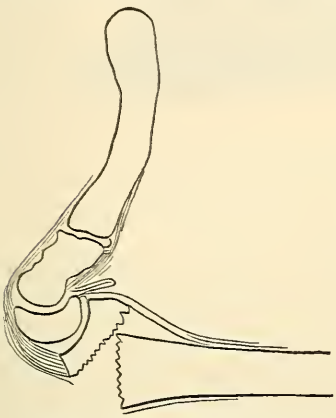


Fig. 330.—Effect upon the lower end of the radius of the cross-breaking strain produced by extreme backward flexion of the hand (Pilcher).

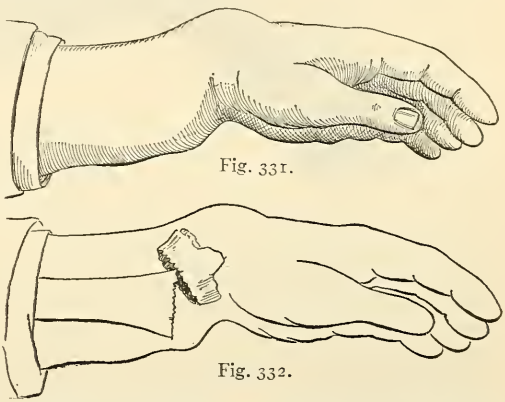


Fig. 331.

Fig. 332.

Figs. 331, 332.—Deformity at the wrist consequent upon displacement backward of the lower fragment of the radius after fracture at its lower extremity (Levis).

easily on each other. The styloid process of the ulna may be broken, or the inferior radio-ulnar articulation may be separated (dislocation of the lower end of the ulna). This latter complication allows the lower fragment to roll freely upon the upper, and the characteristic silver-fork deformity does not appear. If the styloid process of the ulna is broken, pressure over it causes great pain. If a person in falling strikes the back of the hand and a fracture of the radius occurs, the lower fragment is driven upon the front surface of the upper fragment and is felt under the flexor tendons at the wrist (*reversed deformity*). An elaborate study of fracture of the radius with forward displacement of the lower fragment has been published by John B. Roberts.[1]

*Treatment.*—In treating an ordinary Colles's fracture reduce the deformity by hyperextension to unlock the fragments and relax the dorsal periosteum, follow by longitudinal traction to separate the fragments, and forced flexion to force them into position. This formula was introduced many years ago by the late R. J. Levis. It is of the first importance to thoroughly reduce this fracture, and very often it is not thoroughly reduced. Imperfect reduction means permanent deformity, stiffness of the tendons and wrist, and possibly an almost useless

[1] "Am. Jour. Med. Sci.," Jan., 1897.

hand. The extremity can be placed upon a Levis splint (Fig. 333), the position maintaining reduction and the tense extensor tendons giving dorsal support. Some surgeons use Gordon's pistol-shaped splint. The favorite splint in Philadelphia practice in the past has been Bond's (Pl. 7, Fig. 7). It places the hand in a natural position of rest (semiflexion of the fingers, semi-extension of the wrist, and deviation of the hand toward the ulna). Two pads are used: a dorsal pad which overlies the lower fragment, and a pad for the flexor surface which overlies the lower end of the upper fragment. A bandage is applied,

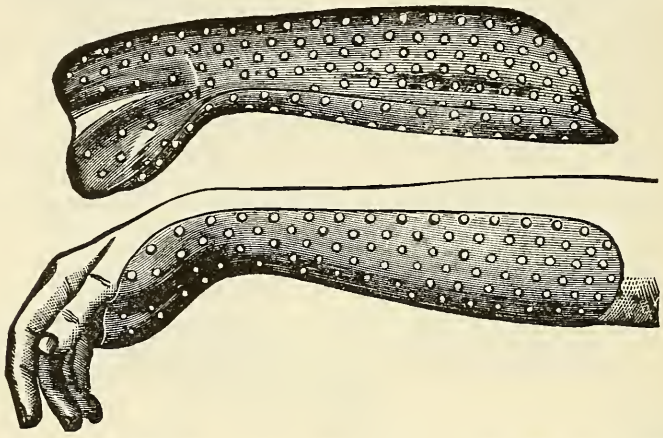


Fig. 333.—Levis's radius-splints, right and left, for fracture of the lower end of the radius.

the thumb and fingers being left free (Fig. 334). Passive motion is begun upon the fingers in three or four days, and upon the wrist during the second week. The splint is removed in three weeks, and a bandage is worn for a week or two more because of the swelling. In applying the Bond splint, do not pull the hand too much up on the block, or the fracture will unite with a projection upon the flexor surface of the extremity and the tendons of the wrist will be apt to be caught in the callus. The most satisfactory dressing is the straight dorsal splint advised by Roberts (Fig. 335). It runs from just below the external condyle to the beginning of the fingers. I use it almost invariably. It prevents the recurrence of deformity and is mechanically the proper mode of treatment. It should be worn for three weeks. Undoubtedly more or less stiffness often follows Colles's fracture, and some very able surgeons have been so impressed with the frequency of crippling stiffness that they have dispensed with the use of a splint. Sir Astley Cooper long ago spoke of simply placing the arm in a sling as proper treatment for fracture of the radius. Moore, of Rochester, applied a cylindrical compress over the ulna, held in place for six hours by adhesive plaster, then cut the plaster, placed the forearm in a sling, and let the hand hang over the edge of the sling. Pilcher applies a band of adhesive plaster around the wrist and supports the wrist in a sling, but, as Storp says, dispensary patients are apt to disarrange this dressing. Storp wraps a piece of rubber plaster 4 inches wide around the wrist, and places a second piece around the first so arranged

Fig. 334.—Bond's splint in Colles's fracture.

as to form a fold over the radius; an opening is made through the fold for the passage of a sling. In ten days the plaster is removed and the forearm is carried in a sling. Massage is begun in the third week. Impaired function follows in about 40 per cent. of these fractures. If a stiff joint and limited tendon motion eventuate from the fracture, use massage, frictions, sorbefacient ointments, tincture of iodin, electricity, hot and cold douches, and the hot-air apparatus, or give ether and forcibly break up adhesions. If reduction was not thoroughly effected and too great a length of time has not elapsed, and the hand is helpless and painful, the bone should be refractured. In a young or middle-aged person, in whom a useless hand has folowed an ill-reduced fracture, osteotomy is justifiable.

**Fracture of Both the Radius and Ulna Near the Wrist.**—Colles's fracture may be complicated by a fracture of the ulna other than of its styloid process.

*Symptoms.*—In fracture of the radius and ulna near the wrist the lower ends of the upper fragments come together, the upper fragment of the radius is pronated, and the lower fragment of the radius is drawn up. Pain, crepitus, mobility, shortening, and loss of function exist.

*Treatment.*—Fracture of the radius and ulna near the wrist should be treated by the straight dorsal splint, as is Colles's fracture.

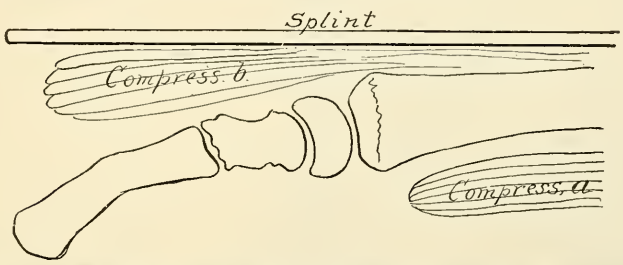



Fig. 335.—Diagram showing the arrangement of compresses and splint best adapted to retain fragments in proper position after reduction (Pilcher).

**Separation of the Lower Radial Epiphysis.**—This accident occurs in children from falling upon the palm of the hand. It never happens after the twentieth year.

*Symptoms.*—In separation of the lower radial epiphysis the lower fragment mounts upon the upper and produces a dorsal projection like that found in Colles's fracture, but the hand does not deviate to the radial side. The deformity resembles that of a backward carpal dislocation, but is differentiated from dislocation by the unaltered relation in the fracture between the styloid processes and the carpal bones.

The *treatment* in separation of the lower radial epiphysis is the same as for Colles's fracture.

**Fractures of the carpus** were until recently thought to be infrequent, but the *x*-rays have taught us differently, and we now know that many supposed sprains of the wrist are, in reality, simple fractures of the carpus. Ernest Amory Codman and Henry Melville Chase show that a majority of carpal injuries "are either simple fractures of the scaphoid or anterior dislocations of the semilunar bone," the two injuries being frequently combined ("The Diagnosis and Treatment of Fracture of the Carpal Scaphoid and Dislocation of the Semilunar Bone," in "Annals of Surgery," March and June, 1905). The *cause* of carpal fracture may be violent direct force or a fall upon the extended palm.

*Symptoms.*—Fractures of the carpus in general are indicated by pain,

tenderness, swelling, evidences of the infliction of direct force, sometimes crepitus, loss of power in the hand, and a very little displacement.

*Treatment.*—Many compound comminuted fractures of the carpus require amputation. In an ordinary compound fracture asepticize, drain, dress with antiseptic gauze and a plaster-of-Paris bandage, cutting trap-doors in the plaster over the ends of the drainage-tube. In a simple fracture dress the hand upon a well-padded straight palmar splint (Pl. 7, Fig. 10) reaching from beyond the fingers to the middle of the forearm, and place the hand and forearm in a sling. The splint is worn for four weeks, and passive motion of the wrist is begun in the second week.

**Fracture of the carpal scaphoid** (see previously quoted article by Codman and Chase) usually results from falls upon the palm of the extended hand and is most common in males between the ages of twenty-five and thirty-five. It is rarely recognized at the time of the accident; the patient complains of severe pain, tenderness, and disability and is thought to have a sprain. According to Codman and Chase, the symptoms improve up to a certain point, but not beyond it, and the joint remains in a condition of irritation and weakness. After months or, perhaps, years the diagnosis is made. In one case of my own, a locomotive engineer, the injury resulted from a blow on the palm with the reverse lever. He came to me three years after the injury, when I recognized the condition as the one described by Codman and Chase. These writers say that the fingers are normally flexible, active and passive movements of the wrist are restricted to one-half or more of the normal excursion, and movements of flexion or extension beyond this are limited by muscular spasm, resembling the spasm occurring in a tuberculous joint. Any attempt to forcibly evercome the spasm produces violent pain. Crepitus is absent. The radial side of the wrist-joint exhibits some swelling, which obscures somewhat the flexor tendons of the thumb. There is tenderness on pressure over the scaphoid and it is most acute in the anatomical snuff-box. The *x*-ray shows a transverse fracture of the scaphoid bone ("Annals of Surgery," March and June, 1905). Professor Dwight considers the above-described injury to be due to the two portions of the bone (there are two centers of ossification) having never formed a bony union and having been wrenched apart by violence. Codman believes the injury is the result of violence acting on a normal bone, the resulting non-union being due to lack of fixation and the presence of synovial fluid between the fragments.

The fracture may be accompanied by forward dislocation of the semilunar bone. If for several weeks after an accident causing fracture of the scaphoid the wrist is immobilized, union may occur, otherwise non-union will surely result.

*Treatment.*—This injury should be thought of when violence has been applied to the carpus. It may be treated by a straight palmar splint if the case is seen early. If seen when there is non-union, the proximal half of the scaphoid should be excised (the incision being posterior and external to the extensor communis digitorum tendons) and passive motion should be begun within one week (Codman and Chase, Ibid.).

**Fractures of the Metacarpal Bones.**—Fracture of the metacarpus is very common. One or more bones may be broken. The first metacarpal bone is oftenest broken; the third is seldom broken (Hulke). The *cause* is direct or indirect force. Fracture at the base of the first metacarpal bone was described by E. H. Bennett in 1881. It is called *Bennett's fracture*, or, as its discoverer named it, *stave of the thumb*. The fracture may be transverse at the neck or longitudinal, "the anterior basal projection being broken off" (Raymond Russ, in "Jour. Amer. Med. Assoc.," June 16, 1906). This injury results from violent force applied to the distal end of the metacarpal (as in striking with

the fist) or to the end of the extended thumb, and Russ regards it as the most common metacarpal fracture. It is usually mistaken for a sprain of the thumb and is sometimes regarded as subluxation backward of the first metacarpal.

*Symptoms.*—The signs of a metacarpal fracture are: dorsal projection of the upper end of the lower fragment or the lower end of the upper fragment; pain, crepitus; and often evidences of direct violence. In fracture of the first metacarpal (Bennett's fracture) there is swelling, particularly evident in the flexor tendon sheaths on the thenar eminence (Russ), disability, pain, tenderness near the base of the metacarpal, and deformity, apparent shortening of the thumb, and crepitus on reduction. The *x*-rays solve a doubtful case.

*Treatment.*—To treat a fracture of a metacarpal bone reduce by extension; place a large ball of oakum, cotton, or lint in the palm to maintain the natural rotundity, and apply a straight palmar splint like that used for fracture of the carpus. It may be necessary to apply a compress over the dorsal projection. The duration of treatment is three weeks, and passive motion is begun after two weeks. A plaster-of-Paris dressing is often used. Raymond Russ ("Jour. Amer. Med. Assoc.," June 16, 1906) describes the following splint as successfully used in a case of Bennett's fracture (Fig. 336). I have used it in a case with much satisfaction. "The thumb was put in strong abduction and three wooden butcher's skewers neatly padded were placed about the metacarpal, one posteriorly in the interosseous space, one along the outer border, and the third over the thenar eminence. These extended from well above the metacarpal bone to the first phalangeal joint. They were fastened tightly in place by two strips of adhesive plaster. Traction was then exerted on the thumb and maintained by strips of adhesive plaster passing about the first phalanx and the projecting ends of the three skewers. This dressing was reinforced by a rectangular cardboard splint. Accurate coaptation and sufficient traction to overcome the deformity and muscular action are most necessary in the treatment of this fracture. Slate pencils or small lead pencils can be used in place of the wooden skewers. The soapstone slate pencils are less brittle than the ordinary kind."

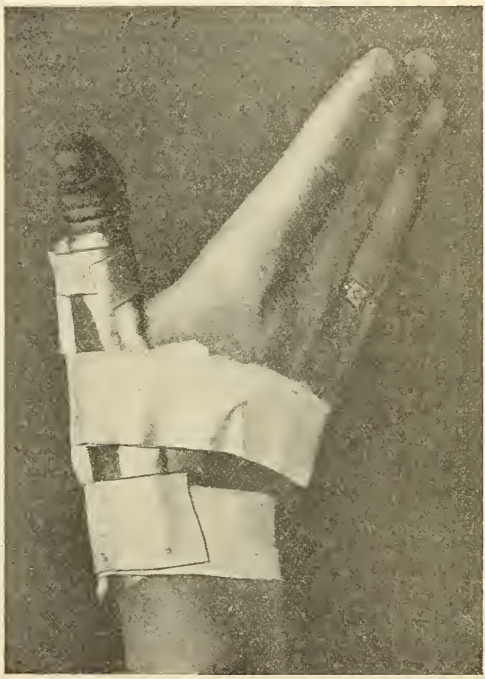


Fig. 336.—Coaptation traction splint of Russ.

**Fractures of the Phalanges.**—The phalanges are often broken. The fracture may be compound. The *cause* usually is direct force.

*Symptoms.*—Fracture of a phalangeal bone is indicated by pain, tenderness, bruising, crepitus, and mobility, with very little or no displacement.

*Treatment.*—If the middle or distal phalanx is broken, mold on a trough-like splint of gutta-percha or of pasteboard, which splint need not reach into the palm. If the proximal phalanx is broken, carry the splint into the

palm of the hand. Make the splint of gutta-percha, pasteboard, wood, or leather. The splint is worn three weeks. A sling must be worn, otherwise the finger will be knocked and hurt. Some cases require a dorsal as well as a palmar splint. These cases may be treated very satisfactorily with a silicate of sodium or plaster-of-Paris bandage.

**Fracture of the femur** is a very common injury. The divisions of the femur are: (1) the upper extremity; (2) the shaft; (3) the lower extremity.

**Fractures of the upper extremity of the femur** are divided into: (*a*) intracapsular; (*b*) extracapsular; (*c*) of the great trochanter, and (*d*) epiphyseal separation (either of the great trochanter or the head).

*Examination of the Hip.*—It is sometimes though seldom necessary to give ether. Remove all the patient's clothing and place him recumbent upon a

Fig. 337.—Intracapsular fracture of the hip (author's case).

table. Note the position of the extremity. Feel with care the great trochanter and femoral neck. Very gradually and gently make movements to determine if there is impairment, undue mobility, or crepitus. Never make sudden or violent movements in looking for crepitus. The diagnosis can be made even if crepitus is not obtained, and rapid or violent movements may tear apart an impaction. Measure the sound extremity and the injured extremity. The measurement is made from the anterior superior spine of the ilium to the inner malleolus. Other symptoms to be looked for are set forth on pages 583 and 584.

**Intracapsular Fracture of the Femur.**—Intracapsular fracture of the neck of the femur is transverse or only slightly oblique (Fig. 337), and is not unusually impacted (see Figs. 267, 268, 269). Stokes follows Gordon, of Belfast,

in classifying fractures of the femoral neck. He divides them into intracapsular and extracapsular, and subdivides intracapsular fractures into fracture with penetration of the cervix into the head; fracture with reciprocal penetration; intraperiosteal fracture at the junction of the cervix and head; intraperiosteal fracture of the center of the cervix; extraperiosteal fracture, with laceration of the cervical ligaments. The last-named fracture is the most common. The first four forms may unite by bone, the fifth form will not because of non-apposition, lack of nutrition, effusion of blood, synovitis, or interstitial absorption.[1] Stokes points out that we may have penetration without impaction. The *cause* is often slight indirect force, of the nature of a twist, acting upon a person of advanced years (more often a woman than a man), but not unusually a fall upon the great trochanter is the cause. A fall upon the knees, a trip, or an attempt to prevent a fall may produce this fracture. It often happens that the fall is due to the fracture rather than that the fracture arises from the fall. Intracapsular fracture is never caused by direct force unless it is due to gunshot violence. The aged are more liable to intracapsular fracture than the young or the middle-aged, because, first, the angle which the neck forms with the axis of the femur becomes less obtuse with advancing years, and may even become a right angle; this change is more pronounced in women than in men; second, the compact tissue becomes thinned by absorption, the cancelli diminish, the spaces between them enlarge, the bony portions of the cancellous structure are thinned and destroyed, and the cancellous structure becomes fatty and degenerated. The injury is not, however, limited to the aged. It has been positively shown that this fracture may occur in the young, even before the union of the epiphyses. In fact, fracture of the femoral neck is not very uncommon in children and in young and vigorous adults (Royal Whitman, "Med. Record," March 19, 1904). I have seen one case in a man of twenty-five, one in a man of twenty-eight, and several cases in those under forty-five. In the aged the fracture is, of course, complete, but in children and even in young adults it is usually incomplete, and for this reason the fracture is often not recognized in children and young adults.

*Symptoms.*—In intracapsular fracture there is usually *shortening* to the extent of from ½ to 1 inch, but in some cases no shortening can be detected. Shortening of ¼ inch does not count in making a diagnosis, for one limb is often naturally a little shorter than the other. If the reflected portion of the capsule is not torn, the shortening is trivial in amount or is entirely absent. In some cases shortening gradually or suddenly increases some little time after the accident. This is due to separation of a penetration or of an impaction, tearing of the previously unlacerated fibrous synovial reflection, or restoration of muscular strength after traumatic paresis has passed away. A gradually increasing shortening arises from absorption of the head of the bone. Shortening is due chiefly to pulling upon the lower fragment by the hamstring, the glutei, and the rectus muscles.

*Pain* is usually present anteriorly, posteriorly, and to the side. The area of pain is localized, and motion or pressure greatly increases the suffering. Pain is not commonly severe except upon motion or from pressure, when it may be localized in the joint. In some cases the pain is violent.

*Eversion* exists and is spoken of as *helpless eversion*, though in a very few instances the patient can still invert the leg. The eversion is due to the force of gravity, the limb rolling outward because the line of gravity has moved externally. That eversion is not due to the action of the external rotator muscles, as was taught by Sir Astley Cooper, is proved by the fact that when a fracture happens in the shaft below the insertion of these muscles

[1] Stokes, in "Brit. Med. Jour.," Oct. 12, 1895.

the lower fragment still rotates outward. This is further demonstrated by the considerations that the internal rotators are more powerful than the external, that some patients can still invert the limb after a fracture, and that eversion persists during anesthesia.[1] In some unusual cases *inversion* attends the fracture. Inversion, if it exists, is due to the fact that the limb was adducted and inverted at the time of the accident, and after the accident it remains in this position (Stokes). Besides shortening and eversion, the leg is somewhat flexed on the thigh and the thigh on the pelvis, the extremity when rolled out resting upon its outer surface. *Abduction* is commonly present. Limited abduction suggests impaction.

*Loss of power* is a prominent symptom: the limb can seldom be raised or inverted; although in rare cases, when the fibrous synovial envelope is untorn, the patient may stand or even take steps. *Crepitus* often cannot be found, either because the fragments cannot be approximated, because there is impaction or penetration, or because the bone is greatly softened by fatty change. To obtain crepitus the front of the joint must be examined while the limb is extended and rotated inward. But why try to obtain crepitus? The diagnosis is readily made without it; in many cases it cannot be detected, and the endeavor to obtain it inflicts pain and may produce damage. These fractures in the aged have a not very flattering chance for repair, and efforts to find crepitus may produce serious damage.

*Altered Arc of Rotation of the Great Trochanter* (*Desault's Sign*).—The pivot on which the great trochanter revolves is no longer the acetabulum, and the great trochanter no longer describes the segment of a circle, but rotates only as the apex of the femur, which rotates around its own axis. It is needless to try to obtain this sign; to do so inflicts violence on the parts.

*Relaxation of the fascia lata* (*Allis's sign*) simply means *shortening*. The fascia lata is attached to the ilium and the tibia (iliotibial band), and when shortening brings the tibia nearer to the ilium, this band relaxes and permits the surgeon to push his fingers more deeply inward on the injured side, between the great trochanter and the iliac crest, and nearer the knee above the outer condyle, than on the sound side. In this examination each limb should be adducted. Allis has pointed out another sign: when the patient is recumbent the sound thigh cannot be lifted to the perpendicular without flexing the leg; the injured thigh can be.

*Lagoria's sign* is relaxation of the extensor muscles.

*Ascent of the Great Trochanter Above Nélaton's Line.*—This line is taken from the anterior superior iliac spine to the most prominent part of the ischial tuberosity (Fig. 338). In health the great trochanter is below, and in intracapsular fracture it is above, this line.

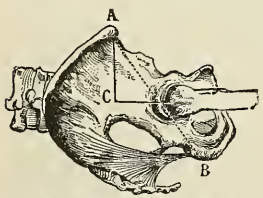


Fig. 338.—A-B, Nélaton's line; A-C-D, Bryant's iliofemoral triangle (Owen).

*Relation of the Trochanter to Bryant's Triangle* (Fig. 338).—Place the patient recumbent, carry a line around the body on a level with the anterior superior iliac spines, draw a line from the anterior iliac spine on each side to the summit of the corresponding great trochanter, and measure the base of the triangle from the great trochanter to the perpendicular line to determine the amount of ascent. The difference in measurement between the two sides shows the amount of ascent of the trochanter; that is, shows the extent of shortening.

*Morris's measurement* shows the extent of inward displacement. Measure from the median line of the body to a perpendicular line drawn through the trochanter on each side of the body.

[1] Edmund Owen, "A Manual of Anatomy."

*Diagnosis.*—The *x*-rays are a valuable aid to diagnosis (Fig. 339). Intracapsular fracture without separation of fragments may be mistaken for a mere contusion, and the diagnosis may continue obscure unless the fragments separate. Loss of function in contusion is rarely complete or prolonged, although occasionally the head of the bone undergoes absorption. Early after a contusion, and usually throughout the case, there is no alteration between the relation of the spine of the ilium and the trochanter, and no shortening. Some time *after* a severe contusion the head of the bone may be absorbed. Contusion of a rheumatic joint leads to much difficulty in diagnosis. Intracapsular fracture may be confused with *extracapsular* fracture or with dislocation of the hip-joint. Extracapsular fracture, which is common in ad-

Fig. 339.—Author's case of recent intracapsular fracture in a woman aged forty successfully nailed. Nail is still in place after five years.

vanced life, but is met with not unusually in middle life and even occasionally in the young, results usually from great violence over the great trochanter; if non-impacted, there are noted shortening of from $1\frac{1}{2}$ to 3 inches, crepitus over the great trochanter, and usually, but not invariably, eversion; if impacted, there is less eversion, crepitus is absent, and the shortening is limited to about an inch. The extensor muscles are relaxed. Great tenderness exists over the great trochanter in both impacted and non-impacted fractures. In dislocation on the dorsum of the ilium the patient is usually a strong young adult. There is a history of forcible internal rotation. There are inversion (the ball of the great toe resting on the instep of the sound foot), rigidity, ascent of the great trochanter above Nélaton's line, and shortening of from 1

to 3 inches. The head of the bone is felt on the dorsum of the ilium, and the trochanter mounts up toward the spine of the ilium, and pressure upon it causes no pain. In dislocation into the thyroid notch there is possibly eversion, but it is linked with lengthening.

In *fracture of the brim of the acetabulum* there is shortening, which occurs on the removal of extension, inversion, abduction, flexion of the knee, the head of bone is drawn upward and backward with the acetabular fragment, and there is retention of the power of eversion and of adduction (Stokes). Crepitus is most distinctly appreciated by a hand resting on the ilium.

In *fracture of the fundus of the acetabulum* (see Fig. 294) there is shortening, and the head of the bone enters the pelvis (Stokes).

The *prognosis* is not very favorable. Some aged patients die in a day or two from shock. Not a few perish later from hypostatic congestion of the lungs, kidney failure, or exhausion. The majority of cases recover with a little shortening, some stiffness, and a permanent limp. There is a much better chance for firm union if the fracture is impacted than if it is not. Even if non-union results after an intracapsular fracture, and it is not unusual, a patient may get about fairly well with a proper support. In some cases after intracapsular fracture rheumatoid arthritis develops. Many surgeons have maintained that bony union never occurs, but it certainly does sometimes take place. Stokes holds that bony union is possible in fractures with penetration, and even in fractures without penetration when the fracture is within the periosteum.[1]

*Treatment.*—In treating a very feeble old person for intracapsular fracture make no attempt to obtain union. Keep the patient in bed for two weeks; give lateral support by sand-bags; tie around the ankle a fillet, attach a weight of a few pounds to the fillet, and hang the weight over the foot-board of the bed. When pain and tenderness abate, order the patient to get into a reclining-chair, and permit him very soon to get about on crutches. If hypostatic congestion of the lungs sets in, if bed-sores appear, if the appetite and digestion utterly fail, or if diarrhea persists, abandon attempts at cure in any case, and get the patient up and take him into the sunshine and fresh air, simply immobilizing the fracture as thoroughly as possible by means of pasteboard splints or plaster of Paris. In the vast majority of cases, no matter how old the patient may be, undertake treatment. We may be forced to abandon it, but should at least attempt to obtain a cure. If it is determined to treat the case, place the patient on a hair mattress, several boards being laid transversely under the mattress in order to prevent unevenness and the formation of hollows. A fracture-bed is a valuable adjunct to treatment.

*Treatment by the Extension Apparatus of Gurdon Buck.*—Extend the knee and place the leg in a natural posture, and put a pillow beneath the knee. Combine extension with lateral support by means of sand-bags. The extension should be gentle, never forcible. It is not wise to pull apart an impaction in an old person, but it should always be done in a young or middle-aged person. Place the subject on a firm mattress or a fracture-bed. If the patient be a man, shave the leg. Cut a foot-piece out of a cigar-box, perforate it to admit the passage of a cord, wrap it with adhesive plaster as shown in Plate 7, Figs. 15 and 16, run the weight-cord through the opening in the wood, and fasten a piece of adhesive plaster on each side of the leg, from just below the seat of fracture to above the malleolus (Pl. 7, Fig. 14). The plaster is guarded from sticking to the malleoli by having another piece stuck to its under surface opposite each of these points. Apply an ascending spiral reversed bandage over the plaster to the groin (Fig. 340), and finish the bandage by a spica of the groin. Slightly abduct the extremity. Put a brick under each leg of the bed at its

[1] See the masterly paper by Stokes, before quoted.

foot, thus obtaining counterextension by the weight of the body. Run a cord over a pulley at the foot of the bed, and obtain extension by the use of weights. In an adult from 15 to 20 pounds will probably be necessary at first, but after a few days from 8 to 10 pounds will be found sufficient (remember that a brick weighs about 5 pounds). Dawbarn's rule as to the proper weight to be attached is 1 pound for every year up to twenty. When the foot of the bed is raised and the weight to make extension is applied, very gently rotate the extremity, put the foot at a right angle with the leg, and make a bird's-nest pad of cotton or oakum to save the heel from pressure. Take

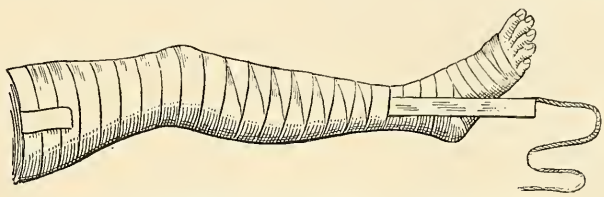


Fig. 340.—Adhesive plaster applied to make extension.

two canvas bags, one long enough to reach from the crest of the ilium to the outer malleolus, the other long enough to reach from the perineum to the inner malleolus. Fill the bags three-quarters full of dry sand, sew up their ends, cover the bags with slips, and put the bags in place in order to correct eversion. The slips may be changed every third or fourth day. Keep the bed-clothing from coming in contact with the extremity by means of a cradle (Figs. 341, 342). The bowels are to be emptied and the urine is to be voided in a bed-pan, unless using a fracture-bed. For two weeks the patient remains recumbent, after which time he can be propped up on pillows. Maintain extension for three

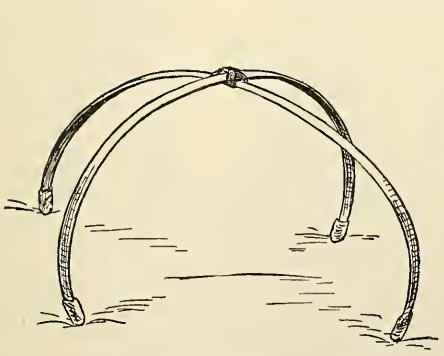


Fig. 341.

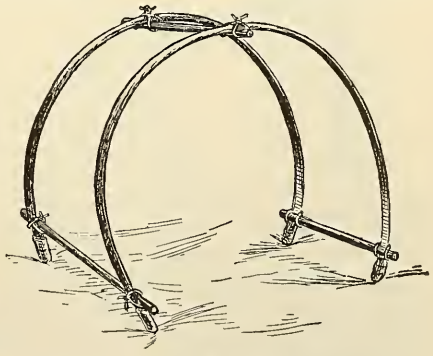


Fig. 342.

Figs. 341, 342.—Cradle to keep clothing from leg, made from two barrel-hoops (Scudder).

weeks, then simply maintain support by sand-bags or molded pasteboard splints upon the part, and keep up this support three to five weeks more. After removing the extension he can be transferred daily to a couch. In from six to eight weeks after the infliction of the injury he can be moved about in a wheeling-chair, the leg being extended or the knee flexed in accordance with the dictates of comfort. After a week or so of such movement a thick-soled shoe is placed on the sound foot and the patient is allowed to use crutches; but weight is not put upon the injured extremity until from ten to twelve weeks have elapsed from the time of the accident. For many months, at least, and possibly permanently, he walks with the aid of a cane. Union, if it takes place, is usually

cartilaginous, but is sometimes bony, and there will surely be some shortening and also some stiffness of the joint. Passive motion is not made until at least eight weeks have elapsed since the accident. Treatment by the extension apparatus is far from satisfactory, as it does not afford sufficient immobilization.

*Senn's Method.*—Senn claims that by this method of "immediate reduction and permanent fixation" bony union is obtained in fractures of the neck of the femur within the capsule. He "places the patient in the erect position, causing him to stand with his sound leg upon a stool or box about 2 feet in height; in this position he is supported by a person on each side until the dressing has been applied and the plaster has set.

"Another person takes care of the fractured limb, which in impacted fractures is gently supported and immovably held until permanent fixation has been secured by the dressing. In non-impacted fractures the weight of the fractured limb makes auto-extension, which is often quite sufficient to restore the normal length of the limb; if this is not the case, the person who has charge of the limb makes traction until all shortening has been overcome as far as possible, at the same time holding the limb in position, so that the great toe is on a straight line with the inner margin of the patella and the anterior superior spinous process of the ilium. In applying the plaster-of-Paris bandages over the seat of fracture a fenestrum, corresponding in size to the dimensions of the compress with which the lateral pressure is to be made, is left open over the great trochanter.

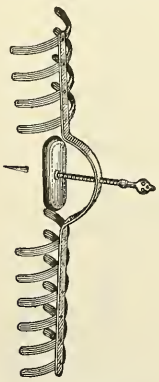


Fig. 343.—Senn's apparatus.

"To secure perfect immobility at the seat of fractures it is not only necessary to include in the dressing the fractured limb and the entire pelvis, but it is absolutely necessary to also include the opposite limb as far as the knee and to extend the dressing as far as the cartilage of the eighth rib.

"The splint (Fig. 343) is incorporated in the plaster-of-Paris dressing, and it must be carefully applied, so that the compress, composed of a well-cushioned pad with a stiff, unyielding back, rests directly upon the trochanter major, and the pressure, which is made by a set-screw, is directed in the axis of the femoral neck. Lateral pressure is not applied until the plaster has completely set. Syncope should be guarded against by the administration of stimulants.

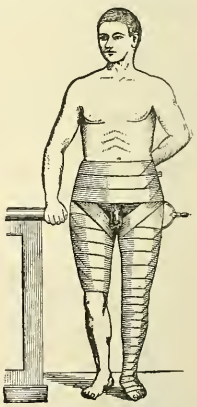


Fig. 344.—Senn's apparatus applied.

"As soon as the plaster has sufficiently hardened to retain the limb in proper position the patient should be laid upon a smooth, even mattress, without pillows under the head, and in non-impacted fractures the foot is held in a straight position and extension is kept up until lateral pressure can be applied.

"No matter how snugly a plaster-of-Paris dressing is applied, as the result of shrinkage it becomes loose, and without some means of making lateral pressure it would become necessary to change it from time to time in order to render it efficient. But by incorporating a splint in the plaster dressing (Fig. 344) this is obviated, and the lateral pressure is regulated, day by day, by moving the screw, the proximal end of which rests on an oval depression in the center of the pad."

*Treatment by Thomas's Splint.*—Scudder, in his valuable treatise on "The Treatment of Fractures," advocates in intracapsular fracture the use of Thomas's hip splint. If the bones are unimpacted, the fragments are brought into apposition by extension, inversion, and pressure upon the great trochanter, and the Thomas splint is bent to fit, is padded, and is applied (Figs. 345, 346). When the bed-pan is to be used or the bed is to be smoothed, the patient can be lifted without disturbing the fracture. He can be turned on the sound side. If hypostatic congestion is developing, raise the head of the bed and tie the splint to the iron of the head of the bed. In addition to the use of the splint Scudder advocates the making of lateral pressure over the great trochanter by a graduated compress and a bandage. The splint is worn for six or eight weeks. It is then removed, the patient remaining in bed four weeks longer without any apparatus (Scudder, from Ridlon).

Jones, of Liverpool, treats fractures of the femoral neck by means of an extension frame (see Fig. 357).

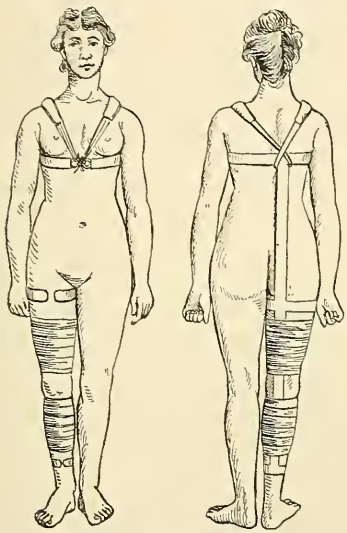


Fig. 345.—Thomas's single hip-splint in position (Ridlon).

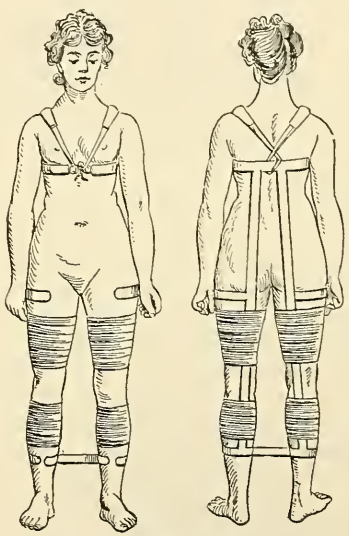


Fig. 346.—Thomas's double hip-splint in position (Ridlon).

*Whitman's Treatment in Abduction.*—The plan advocated by Royal Whitman ("Med. Record," March 19, 1904) is a most excellent one. It aims to abolish traumatic depression of the neck of the femur.

We can apply this plan in a young person to any fracture even if impacted. In an aged person we apply it only in a complete non-impacted fracture. In a young person we usually give ether and pull apart an impaction by abduction. In an aged person we should not do so.

In regard to impaction Jones says, if the shortening is trivial and there is no rotation an impaction is not to be pulled apart; if there is marked shortening and eversion, it is to be.

Make extension, counterextension, and internal rotation until the foot is at a right angle with the table and shortening is abolished, and then slowly abduct. The abduction relaxes the muscles which interfere with reduction and carries the outer fragment against the inner.

The extremity is set in extension and extreme abduction and plaster of Paris is applied. The tension of the capsule pushes the outer fragment against the inner and holds it; fixation is obtained by the neck of the femur being in

contact with the acetabulum and the great trochanter with the pelvis, deformity cannot be caused by muscular action, and the psoas helps pull the fragments together (Whitman).

The limb is kept fixed in abduction for six weeks and then a Thomas splint with extension is used.

**Extracapsular Fracture** (*Fracture of the Base of the Neck of the Femur*).—The line of extracapsular fracture is at the junction of the neck with the great trochanter, and is partly within and partly without the capsule, the fracture being generally comminuted and often impacted. The *cause* is violent force over the great trochanter (as by falling upon the side of the hip). This fracture is most usual in elderly people, but is met with in the middle aged and is not very uncommon in young adults. Stokes has described six forms of extracapsular fracture: extracapsular fracture with partial impaction posterior; fracture with complete impaction; fracture with partial impaction above; fracture with partial impaction below, the shaft being split; splitting of the neck longitudinally without impaction; comminuted non-impacted fracture.[1]

*Symptoms.*—When impaction is absent there is marked crepitus on motion, which is manifested most distinctly when the fingers are placed upon the great trochanter; there is severe pain, pressure upon the great trochanter is very painful, swelling and ecchymosis are marked; there is absolute inability on the part of the patient to move the limb, and passive movements cause violent pain; there is shortening to the extent of at least 1½ inches, and sometimes to the extent of 3 inches, which shortening is made manifest by noting the ascent of the trochanter above Nélaton's line, by a comparison of measurements of the injured limb and the sound limb, and by measuring the base-line of Bryant's triangle on each side. Absolute eversion usually exists with slight flexion both of the leg and the thigh. In some rare cases there is inversion. This happens if at the time of the accident the limb was inverted and adducted (Stokes). Langoria's sign, Desault's sign, and Allis's sign are present. All these symptoms follow violent direct lateral force to the great trochanter. In the *impacted* form of extracapsular fracture, in addition to the aid given the surgeon by the history, there is severe pain, which is intensified by movement or pressure; shortening probably to the extent of 1 inch, which is not corrected by extension; limited abduction; great loss of function; and whereas the limb may be straight or even inverted, it is usually everted. The trochanter is above Nélaton's line, the base-line of Bryant's triangle is shortened, but not so much as in the unimpacted form; there is no crepitus unless the impaction is loose or is pulled apart, and the arc of rotation of the great trochanter is larger than in a non-impacted fracture.

*Treatment.*—In impacted extracapsular fracture it is best to pull apart the impaction if the patient is in good physical condition. Southam, of Manchester, in an impressive article has insisted on the absolute necessity of pulling apart an impaction. He gives ether, and when the patient is anesthetized unlocks the fragments.[2] This unlocking is best accomplished by abduction, the rim of the acetabulum acting as the fulcrum of the lever (Whitman). In treating extracapsular fracture we can use the extension apparatus with sand-bags (see page 586) for three weeks and then apply a plaster dressing. Get the patient on crutches after the plaster has been in place for two weeks. Remove the plaster at the end of four weeks. Thomas's splint may be used instead of Buck's extension or the treatment suggested by Whitman may be employed (see page 589).

**Fractures of the Femoral Neck in Children.**—Fracture of the femoral neck in children and in young adults can scarcely be regarded as very unusual, and is certainly more often encountered than separation of the upper epiph-

[1] "Brit. Med. Jour.," Oct. 12, 1895. [2] "Lancet," Dec. 21, 1895.

ysis. The accident results from a fall rather than, as so often in an adult, from a twist, and it is the product of considerable violence rather than of slight force. In children such fractures may be impacted, and most of those which are unimpacted are of the green-stick variety. The disability is not nearly so great as

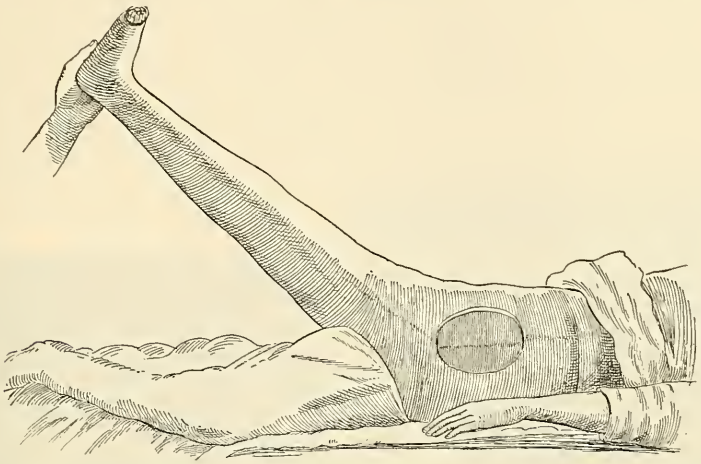


Fig. 347.—The long spica as applied for fracture of the neck of the femur in the adult; illustrating the advantage of an appliance which permits movement without danger of displacing the fragments; an opening has been made to lessen the constriction of the abdomen (Whitman).

in an adult; in fact, it is not unusual for the victim of such an injury to be able to hobble about a few days afterward. The symptoms are shortening, some eversion, impairment of joint movements, and a limp when the patient gets about. Fractures of the hip in children are often unrecognized and lead frequently to permanent impairment because of the development of coxa vara. The *x*-rays should be used in making the diagnosis.

A green-stick fracture may be treated with Thomas's splint, and after four weeks in bed "the child may be allowed up, wearing a traction hip-splint for several months until union is so firm that the danger from coxa vara is practically eliminated. A light plaster-of-Paris spica bandage from the calf to the axilla will maintain immobility after the splint is omitted" (Scudder, on "The Treatment of Fractures"). An impacted fracture, after the impaction has been pulled apart, is treated exactly as a green-stick fracture. Royal Whitman's plan for treating a green-stick fracture is very satisfactory. This surgeon ("Med. Record," March 19, 1904) dresses these cases by placing the limb in extreme abduction and holding it so by means of a plaster-of-Paris spica (Figs. 347, 348). In a case of acute disability of the hip-joint in a child, following some time after fracture of the femoral neck, make a careful differentiation from tuberculous disease of the joint and apply

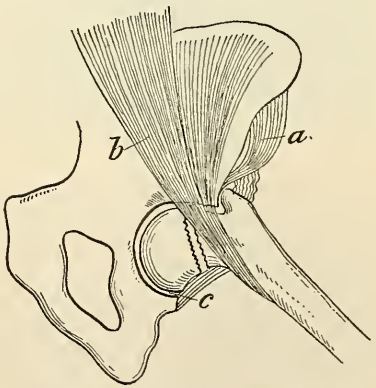



Fig. 348.—Reduction and fixation in abduction, showing security assured by direct bony contact of the neck and trochanter with the pelvis, also the effect of the attitude on muscular action: *a*, Abductor group; *b*, iliopsoas; *c*, capsule (Whitman).

a traction splint to support the body and give rest to the joint. If coxa vara becomes marked and causes great disability, osteotomy is justifiable.

**Operative Treatment of Fracture of the Femoral Neck.**—I have practised this but once. The patient was a woman forty years of age and the result was excellent (see Fig. 339). The operation is not indicated in elderly subjects. It is not indicated at all if the fragments can be coaptated and retained by extension and counterextension or by abduction. In a youth or a middle-aged person in whom retention in correct posture is impossible it is indicated.

Some advocate incision, suture of the torn capsule, and nailing. In my case I nailed through a very small skin incision and did not suture the capsule. It is held that suture of the capsule improves the circulation in the broken off head, but possible attainment of the object is not justification for the risk. The small cutaneous incision and nailing answers the purpose. It is not necessary to drill the trochanter as König did.

Fig. 349.—Comminuted fracture of upper third of femur.

Fig. 350.—Epiphyseal separation of head of the femur.

Before operating an *x*-ray picture is taken, while the fragments are brought into apposition by abduction or by extension and counterextension. From this picture the angle of the neck is noted and the length of nail required is determined. A silver nail is used. When ready to operate, the fragments are again brought into apposition by extension and counterextension or abduction. A small incision is made through the skin over the external aspect of the great trochanter, and the nail is driven through the trochanter and neck into the head. The wound is closed and dressed. The pelvis and extremity are then put up in plaster, a trap-door being cut over the seat of incision. The plaster dressing is retained for five or six weeks. Cure is obtained without shortening and with retention of joint mobility. This operation is also used for ununited fracture. Dr. G. G. Davis was the first to do a nailing operation for a recent intracapsular fracture of the femur.

**Separation of the upper epiphysis of the femoral head** (Fig. 350) is a very rare result of accident; it occurs most often from disease. It is met with in early youth, results in considerable permanent shortening, and perhaps in coxa vara.

*Symptoms and Treatment.*—The *symptoms* are like those of fracture of the neck, except that the crepitus is soft. The *treatment* is as for fracture of the neck.

**Fractures of the Great Trochanter.**—This is a very rare injury. There seem to be only 8 cases on record, but probably the diagnosis has been missed in some cases in which the fragment was held to the bone by periosteum. This process may be (1) broken off without any other injury. In some cases it is completely broken off; in some it remains attached by periosteum and fibrous tissue. (2) The line of fracture may run through the trochanter and leave one portion of the trochanter attached to the head and neck and the other part attached to the shaft of the femur. The *cause* is violent direct force over and behind the great trochanter or a fall (Armstrong, in "Annals of Surgery," August, 1907). Neck reported a case due to muscular action ("Zentralb. für Chir.," 1903).

Fig. 351.—Intertrochanteric fracture.

*Symptoms and Treatment.*—The *symptoms* of the first form resemble those of epiphyseal separation and, of course, there is no shortening. The symptoms of the second form are similar to those of the extracapsular fracture. On rotating the femur the lower part of the trochanter moves with it, but not the upper. The lower fragment goes upward and backward and projects by the side of the sciatic notch. There are shortening, eversion, crepitus, and altered position of the trochanter. The *treatment* of the second form is like that in extracapsular fracture, and the first form is treated like separation of the epiphysis of the trochanter.

**Separation of the epiphysis of the great trochanter** is a rare accident. The *cause* is direct violence and the injury occurs in those under eighteen years of age. Poland in 1898 collected 12 cases.

*Symptoms.*—The trochanter, if completely separated, is found to have ascended and passed posteriorly; there is no shortening of the thigh; all the motions of the hip-joint can be obtained; if the thigh is flexed, abducted, and rotated externally, and the fragment is pushed downward and forward, crepitus may be obtained—soft in epiphyseal separation, hard in fracture. There is no shortening. If the process is not completely separated, diagnosis is impossible without the *x*-rays.

*Treatment.*—If the epiphysis is not completely separated, immobilize the limb in the position of abduction. If it is completely separated, incise the soft parts and either suture or nail the fragment in place.

**Fractures of the shaft of the femur** may affect any portion of the shaft, but especially the middle third, and may occur at any age. Fracture of the upper third is a rare accident. Allis estimates that each year in Philadelphia there is 1 case of fracture of the upper third of the femur to every 100,000 inhabitants. Separation of the lower epiphysis occasionally occurs.

The *cause* of fractures in the upper third is usually indirect force; fractures in the lower third are due to direct force; and in fractures of the middle third these two causes are about equally potential. Fracture from muscular action occasionally occurs. Oblique fracture is the usual variety. In many cases the soft parts are badly lacerated and sometimes a great vessel is torn.

*Symptoms.*—The chief symptom in fracture of the shaft of the femur is great displacement, except when impaction occurs, when the break is due to direct force, or when the injury is in a child. In a child the line of fracture is often transverse and the periosteum may be untorn. Green-stick fractures occur in children. As a rule, in fracture of the shaft of the femur the lower fragment is drawn upward and the upper end of the lower fragment

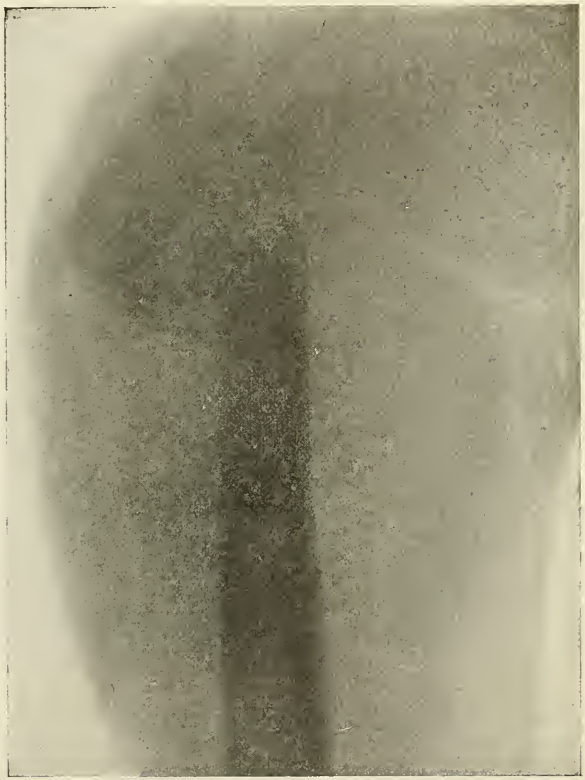


Fig. 352.—Deformity following fracture of upper third of femur.

is found posterior and somewhat to the inside of the lower end of the upper fragment, and the lower fragment also undergoes external rotation (the drawing up is due to the rectus and hamstrings; the passing inward is due to the adductor muscles; the rotation outward arises from the weight of the limb). If a fracture of the lower two-thirds of the shaft is produced by direct force, there is usually but little deformity, because the line of fracture is nearly transverse. If produced by indirect force, there is often great deformity, the line of fracture being oblique. In fracture of the lower third of the shaft the gastrocnemius pulls upon the condyles and tilts the lower fragment, so that its upper end projects into the popliteal space and may damage the vessels. In fracture of the upper third the upper fragment is apt to be thrown strongly forward and outward (Fig. 352). Some attribute this to the action of the

psoas, iliacus, and external rotator muscles, but Allis thinks it is due chiefly to the lower fragment pushing the upper fragment into this position, a part of the tendon of the gluteus maximus acting as a hinge for the fragments.[1] In rare cases the angular deformity is backward. In fracture of the shaft of the femur there is complete loss of function, the thigh and leg are slightly flexed and usually everted. In some cases the leg and lower fragment are inverted. There are shortening to the extent of 2 or 3 inches, pain on movement, preternatural mobility, crepitus, and obvious deformity, and the ends of the fragments can be felt by the surgeon. In impaction there is alteration of the axis of the limb and some shortening. Always feel for the pulse below the fracture to learn if the artery is damaged.

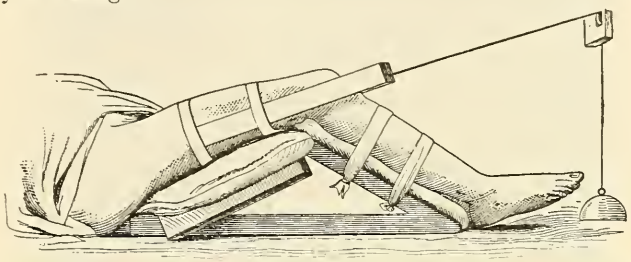


Fig. 353.—Dressing of fracture of the femur in the upper third with extension upon a double inclined plane (Agnew).

*Treatment.*—In setting and dressing a fracture of the thigh ether should be given and the parts must be handled with great care to prevent a sharp end of bone from tearing the soft parts and puncturing the skin. In fracture of the shaft of the femur, if impaction exists, the fragments must be pulled apart, when the case should be treated exactly as is a non-impacted fracture. After a fracture of the shaft of the femur some amount of permanent shortening is almost inevitable. In *fracture of the upper third* in an adult conservative treatment is usually unsatisfactory, and there is permanent shortening from angular union or from overlapping. In youths under fifteen a good result is obtained in over 90 per cent. of cases. Horizontal extension fails to correct the displacement of the upper fragment in fracture of the upper third. The double inclined plane will not correct the tilting of the upper fragment while shortening exists. Agnew used a double inclined plane and corrected shortening by the use of extension in the axis of the partly flexed thigh (Fig. 353). This plan is one of the most serviceable of those usually employed, but it too fails to completely correct the displacement. If, notwithstanding position and extension, the upper fragment projects, it should be pushed into place and be retained if possible by short splints bound upon the thigh. In many cases a Thomas knee-splint is the best apparatus. In fracture of the upper third with marked projection of the upper fragment the abduction frame may prove satisfactory. Extension should be continued for four weeks, a plaster-of-Paris

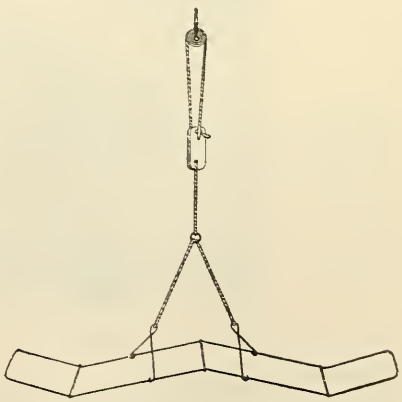


Fig. 354.—Smith's anterior splint.

[1] "Fracture in the Upper Third of the Femur Exclusive of the Neck," by Oscar H. Allis, "Medical News," Nov. 21, 1891.

bandage being used for four weeks more, the patient being then allowed to go about on crutches. Some surgeons, in fracture of the upper third, apply a plaster-of-Paris bandage to the leg, thigh, and pelvis, extension being made from the foot while the dressing is being applied. This method does not give good results because such extension will not correct the tilting of the upper fragment. The anterior splint of Nathan R. Smith is used by some in treating fractures of the upper third of the femur (Fig. 354). It is bent to the desired shape, fastened to the anterior surfaces of the leg and thigh, and hung to a gallows, the limb being suspended at the desired height. This splint is open to the same

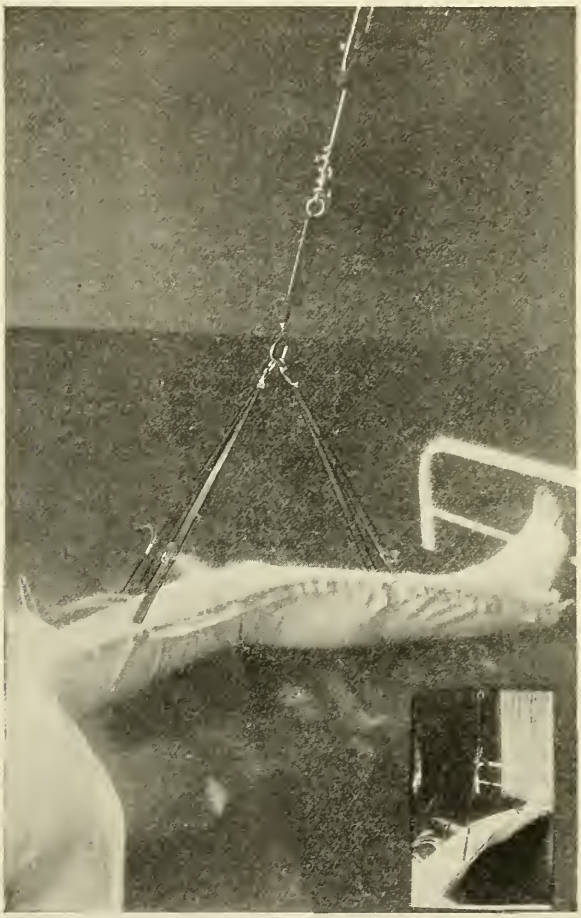


Fig. 355.—Hodgen's apparatus as applied by Dr. George S. Brown.

objection as the double inclined plane. In fact, in many fractures of the upper third of the shaft of the femur no apparatus will maintain reduction. In such cases it is advisable to incise, separate the muscles from between the fragments, and fasten the ends of the bone-fragments together with bone plates. This radical treatment has certain dangers of its own, but it is the plan which promises best to secure a thoroughly good limb. In *fracture of the middle third* or *upper part of the lower third* of the shaft of the femur the Thomas knee-splint is an excellent instrument. The extension apparatus and sand-bags will usually secure a satisfactory result (Pl. 7, Fig. 14). The

strips of adhesive plaster are carried to just below the seat of fracture, and the turns of the roller bandage should be taken to a little above this point. Extension should be continued for four weeks, when the plaster-of-Paris bandage ought to be applied. The plaster is kept in place for four weeks. Many surgeons use Hodgen's splint in treating fractures of the thigh. The limb is suspended in a cradle and extension is obtained by strapping the foot of the crossbar of the frame and pulling upon the frame by cords (Fig. 355). Hodgen's apparatus as applied by Brown, of Birmingham, Ala., is one of the most satisfactory methods of treatment in fracture below the upper third. The extremity can be raised or lowered at will without disturbing the approximation of the fragments, extension to the required degree can be obtained, and the patient can be moved in bed. I consider this apparatus one of the most comfortable appliances which can be worn and excellent results are obtained by its use. In fracture of the middle third or upper part of the lower third of the shaft if the line of fracture is transverse and there is little deformity, as is seen often after a fracture by direct force, and often in children, immobilization in an immovable dressing may be all that is required; but if shortening exists, extension must be used. If extension is used, continue it for four weeks and then substitute a plaster-of-Paris dressing for four weeks. The amount of weight

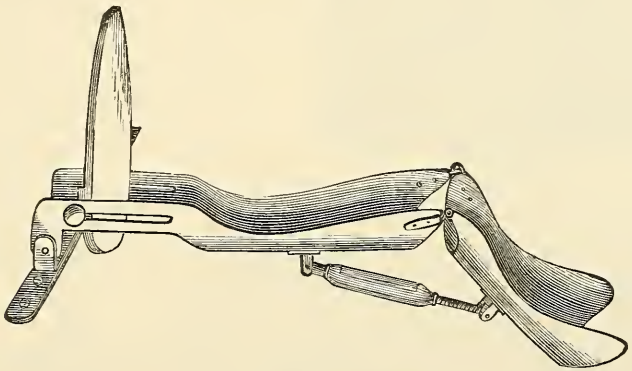


Fig. 356.—McIntyre's splint.

required is pointed out by Dawbarn—1 pound for each year up to twenty.[1] In *fracture near the knee-joint* (*lower part of the lower third* of the femur) it may be impossible to effect reduction by horizontal traction. In such a case make traction, and while it is being made gradually bring the leg to a right angle. Place the limb in a double inclined plane (Pl. 7, Fig. 2). A McIntyre splint (Fig. 356) is a useful form of double inclined plane. After four weeks of the use of a double inclined plane apply a plaster-of-Paris dressing, which is to be worn for four weeks.

**Fractures of the Shaft of the Femur in Children.**—In children under three years of age the extension apparatus will not satisfactorily immobilize the fragments. Fractures of the thigh in children are reduced by extension and counterextension; a well-padded splint reaching from the axilla to below the sole of the foot may be applied to the outer side of the limb and body. This splint is held in place by bandages which are overlaid by plaster of Paris. It is worn for four weeks, at which time it is removed and a plaster bandage, applied so as to include the entire limb, is worn for four weeks.

The abduction frame (Fig. 357) is a very useful plan of treatment.

*Bryant's extension* (Fig. 358) is very satisfactory in treating a child. Both

[1] "Annals of Surgery," Oct., 1897.

the injured limb and the sound limb should be flexed to a right angle with the pelvis, fixed by light splints, and fastened to a bar above the bed. The weight of the body produces counterextension and the child can be easily cleaned.[1]

Another plan is that of Theodore Dunham.[2] The child is placed upon a table, and the knee and hip are partly flexed. After first applying flannel rollers, plaster-of-Paris bandages are applied from the roots of the toes to the spine of the tibia, and as a spica about the upper part of the thigh and pelvis. Two pieces of iron, suitably bent, are used to anchor the two plaster bandages together. One end of one iron is attached to the plaster over the groin and one end of the other iron is attached to the plaster over the front of the leg. The free ends of the irons overlap. At the points over the joints and the front of the leg where the irons are to rest masses of plaster are placed. The iron is sunk into the plaster and supported at each spot by several turns of a plaster bandage. While the irons are being adjusted the thigh is so held as to prevent bending or rotation, and the hip and knees are semiflexed. When the plaster has set an assistant makes extension on the leg and another assistant makes counterextension by pressing on the pelvis. Any shortening is thus reduced and the two irons are lashed together by strong cord (Fig. 359).

Fig. 357.—Jones's abduction frame, showing continued traction and counterextension (courtesy of Mr. Robert Jones, Liverpool, England).

*Van Arsdale's triangular splint* is a very useful appliance. It is made of binders' board. A. Ernest Gallant[3] describes its preparation and application as follows: Measure the length of the sound thigh from the middle of the groin to the end of the femur. Draw upon cardboard an outline of a double spade (playing-card spade) (Fig. 360). Each of the four sections (*A*, *B*, *C*, *D*) must be equal to the length of the child's thigh, the flanged portions being equal to the widest part of the thigh. The figure is then cut out. The cardboard is moistened on one side and folded on the dotted line, section *A* being lapped over *D*, so as to form a triangle. It is fastened together by adhesive plaster. The thigh is flexed and the triangle is applied so that one flanged portion embraces the thigh and the other flanged portion rests upon the abdomen (Fig. 361). The triangle is fixed in position by bandages, figure-of-8 turns being made around the knee and around the thigh and body. Plaster or starch bandages are then applied to fix the splint firmly. The leg should be bandaged from the toe to the knee to prevent swelling (Fig. 361). This splint

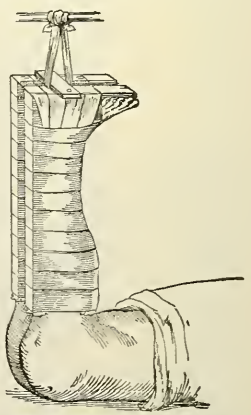


Fig. 358.—Bryant's extension for fracture of the thigh in a child.

[1] Thomas Bryant's "Practice of Surgery."
[2] "Phila. Med. Jour.," April 23, 1898.
[3] "Jour. Amer. Med. Assoc., Dec. 18, 1897.

is worn for three weeks. A child wearing this splint can sit on a chair, nurse, play on the floor and crawl about, may sleep on either side, and the dressing is not soiled by the evacuations.

If a thigh is fractured during parturition, or during the first few weeks of life, *Wyeth's dressing* may be very serviceable. It is applied as follows: The leg is flexed on the thigh and the thigh on the abdomen. A flannel bandage is applied so as to include the leg, the thigh, and the body from the axilla to the pelvis. Plaster of Paris is applied over this; the dressing is worn for four weeks. A better dressing than the above is *Ware's*, a modification of *Van Arsdale's splint* ("Annals of Surg.," August, 1905) (Fig. 362). It is lighter, the patient can be moved about with ease, the child's toilet can be easily carried out, and breathing is not embarrassed. A right-angled triangle is

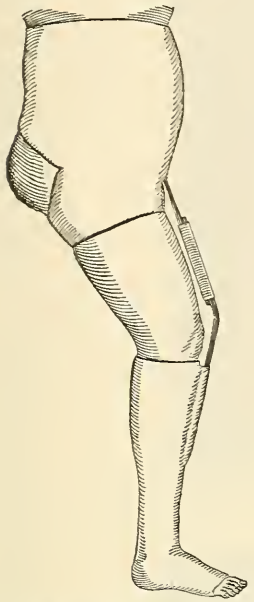


Fig. 359.—Dunham's apparatus for treating fractures of the thigh in infants and children.

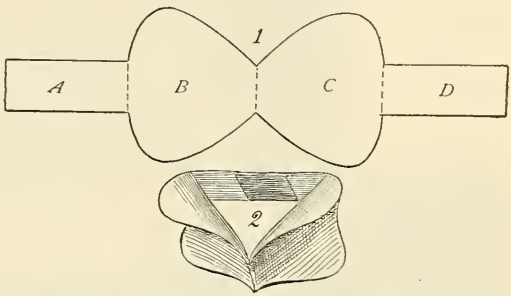



Fig. 360.—*1*, Diagram showing outline of Van Arsdale's splint; the end band to be folded on the dotted lines; each section to equal the length of the child's thigh. *2*, Diagram, splint folded, fastened by rubber plaster, flanges bent to embrace the thigh and abdomen, ready for adjustment (Gallant).

made of bookbinders' board. The length of one side is the distance from the trunk at the level of the lower angle of the scapula to the inguinal fold. The length of the other side is the length of the thigh. The hypotenuse is, of course, longer than the sides. The cardboard is marked, bent into the triangle, and the overlapping edges are secured by means of

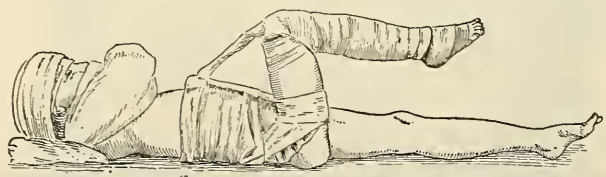


Fig. 361.—Showing Van Arsdale's triangular splint in position. Note the wide space between the dressings and the excretory passages (Gallant).

adhesive plaster. The thigh is flexed and abducted, the inner surface of the splint is padded, the apparatus is applied and retained by a muslin spica about the trunk and thigh. Several turns of a dextrin bandage are applied over this to give strength. The leg hangs free. The dressing is worn for three or four weeks. Figure 362 shows this dressing applied for fracture of the right femur, and Fig. 363 shows it applied when both bones are broken.

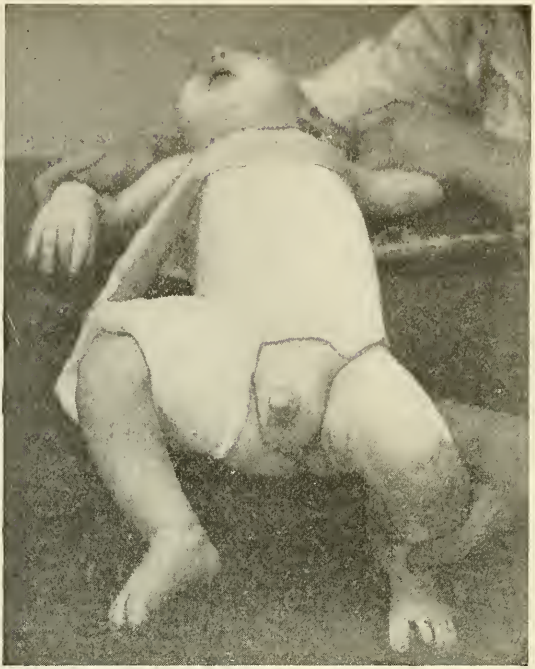


Fig. 362.—Ware's combined pasteboard triangle and plaster-of-Paris spica apparatus for fracture of the femur in infancy (Ware, in "Annals of Surgery," August, 1905).

Fig. 363.—Ware's apparatus for treatment of fracture of both femora (Ware, in "Annals of Surgery," August, 1905).

**Fractures Just Above the Condyles of the Femur.**—The *line* of fracture above the condyles is well above the epiphyseal line. The popliteal artery is in danger from the fragments. The *cause* of the break, as a rule, is direct violence. Indirect force is sometimes responsible (falls upon the feet). The knee-joint may be opened. The fracture is sometimes compound.

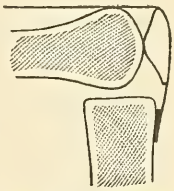


Fig. 364.—Mechanism of fracture of the patella by muscular action (after Treves).

*Symptoms.*—The upper end of the lower fragment is drawn upward and backward, because of the action of the rectus, hamstrings, gastrocnemius, and popliteus. The upper fragment passes inward, and the deformity is very manifest. There are pain, tenderness, shortening, crepitus, and mobility. The ends of the fragments can be felt by the surgeon. If the force has been very great, a T-fracture results. In T-fracture the knee is broadened and crepitus is obtained by moving the condyles, one up and the other down. The popliteal vessels may be torn. Always feel for the pulse below the fracture.

*Treatment.*—In treating fracture above the condyles, reduce the deformity by horizontal extension. If this fails, make traction at the same time, gradually bringing the leg to a right angle with the thigh. Place the limb on a double inclined plane for five weeks, then begin passive motion once every other day, reapplying the splint after the movements are completed. At the end of eight weeks after the accident remove the dressings, and, if the knee-joint be stiff, use for some time massage, passive motion, hot air, hot and cold douches, ichthyol inunctions, etc. Bryant treats this fracture in extension, cutting the tendo Achillis, if necessary, to amend deformity. It is occasionally necessary to plate the fragments. Some cases demand amputation because of injury to the structures in the popliteal space.

Fig. 365.—Fracture of the patella.

**Fracture Separating Either Condyle.**—The *cause* is direct force.

*Symptoms and Treatment.*—The broken piece is drawn upward, the leg bends toward the injury, crepitus exists, the knee is much broadened, there is no shortening, and considerable swelling is sure to arise. In treating a fracture separating either condyle, use a double inclined plane as directed above. If there is great displacement, the condyle should be nailed in place.

**Longitudinal fractures** run upward from the knee-joint. The *cause* is a fall upon the feet or the knees.

*Symptoms and Treatment.*—The *symptoms* of longitudinal fracture are often obscure. The femur is broadened when the knee is flexed. The split may be detected between the condyles. The *treatment* is the straight position in plaster for eight weeks.

**Separation of the lower epiphysis** occurs only before the twenty-first year. It is not a very rare accident in children.

The *symptoms* in separation of the lower epiphysis are like those of transverse fracture, but crepitus is moist. The lower fragment is tilted, so that the articular surface looks forward. The lower end of the upper fragment pro-

jects into the popliteal space. There is danger to the structures in the popliteal space. The growth of bone may be stunted. Feel for the pulse in the leg or foot.

*Treatment.*—Reduction may be effected in some cases by horizontal extension. Occasionally this is impossible.[1] In such a case adopt the plan of Hutchinson and Barnard, make extension, and while it is being made gradually place the leg at a right angle to the thigh. This is effected by an assistant making traction on the leg, while the surgeon clasps his hands beneath the lower part of the thigh and draws upward. The treatment for separation of the lower epiphysis is the use of a double inclined plane as above directed. In some cases replacement is impossible without incision. In a case of my own amputation was performed because of laceration of the popliteal vessels.

Fig. 366.—Fracture of the patella, showing wide separation of the fragments (author's case).

**Fracture of the patella** is a very common accident. The *cause* is direct force (often producing vertical, star-shaped, or oblique lines of fracture, but not uncommonly transverse) or muscular action (producing a transverse line of fracture).

**Transverse Fractures of the Patella.**—The knee-cap is more often broken by muscular action than is any other bone. When the knee is partly flexed the middle third of the patella rests upon the condyles of the femur and the upper third of the knee-cap projects above them; when in this position a contraction of the quadriceps may easily cause a fracture near the center of the bone (Fig. 364). The accident may be caused by sudden flexion of the knee when the quadriceps is contracting. The most usual cause is a fall or an

[1] See the case reported by Jonathan Hutchinson, Jr., and Howard L. Barnard, "Lancet," May 13, 1899.

attempt of the patient to save himself from a fall backward. Both patellæ may be broken at once. In fracture of the patella the joint, and often the prepatellar bursa, is opened. Fractures by muscular action and many fractures from direct force are transverse. The injury is more common in males than in females, and is extremely rare in the very young and the old. It is usually an injury of active manhood and middle life, but I have seen a woman of eighty-six with fracture of the patella.

*Symptoms.*—When the accident happens there is often an audible crack. As a rule, the patient will not try to use the limb, although it is possible for him to stand, to walk backward, and to move slowly forward when the extremity is kept straight. After the accident there is rapid and enormous swelling, due to the effusion first of blood and then of synovia and inflamma-

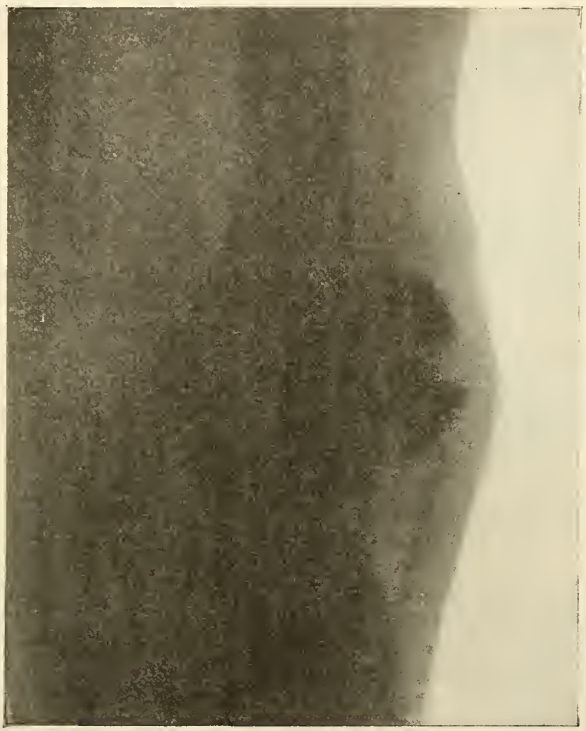


Fig. 367.—Fracture of the patella (Pennsylvania Hospital case; skiagraphed by Dr. Gaston Torrance).

tory products into and around the joint. The patient is absolutely unable to raise the limb from the bed. The fragments are movable and usually widely separated (Fig. 366), this separation being distinctly manifest to the touch unless swelling is great. The separation is accentuated by flexion of the leg. The separation may be to the extent of 1 inch or even more. In cases in which the lateral fibrous expansions and periosteum are but slightly torn, there may be slight separation or no separation. Separation is due in part "to the retraction of the quadriceps and the tension of the fascia lata, and in part to distention of the joint by blood and exudate.[1] If fragments are not approximated and union does not occur, the separation becomes gradually greater because of the progressive shortening of the muscle and the retraction of the

[1] Stimson's "Treatise on Fractures and Dislocations."

ligamentum patellæ (Stimson). In some cases an anterior angular displacement occurs because of the intra-articular distention (Fig. 367). It may be produced by the pressure of bandages or strips of plaster when the fragments have been brought together. Crepitus is detected if the upper fragment can be pushed down until it touches the lower piece; but if swelling is great, or if fibrous tissue is interposed between the bones, crepitus cannot be elicited. It is not necessary to obtain crepitus in order to make the diagnosis: the condition is usually obvious without this sign. The anterior fibroperiosteal layer is torn, and the tear does not correspond exactly with the line of fracture. A portion of this torn fibroperiosteal layer may, as Macewan pointed out, drop between the fragments and prevent union (Fig. 368). The lateral expansions of the capsule are usually extensively torn. If union occurs after a transverse fracture, it will probably be ligamentous, and if the patient gets about too soon, even apparently well-united fragments will by degrees stretch far asunder.

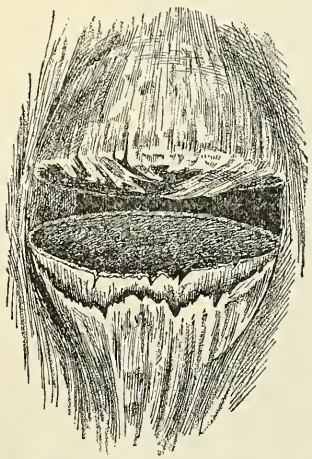


Fig. 368.—Transverse fracture of the patella; fractured surface partially covered by irregular flaps of torn aponeurosis (Hoffa).

**Treatment of Transverse Fractures of the Patella.**—*The Conservative Plan.*—If the swelling is so great as to prevent approximation of the fragments, reduce it by bandaging for a day or two, by using ice-bags, or by aspirating the joint. As a rule, the blood does not coagulate for several days. After it coagulates it cannot be withdrawn by aspiration, but only by incision. When the swelling diminishes, bring the two fragments into apposition, pull them together by adhesive plaster, and put on a well-padded posterior splint. Carry a piece of adhesive plaster over the upper end of the upper fragment, draw the bone down, and fasten the plaster to the splint behind and below the level of the joint. Carry another piece of plaster over the lower end of the lower fragment, draw the bone up, and fasten the plaster to the splint behind and above the joint. Carry a third piece over the junction of the fragments to prevent tilting. Agnew's splint enables us to satisfactorily accomplish this approximation (Pl. 7, Figs. 11, 12). A bandage holds the splint in place, and may be carried around the knee by figure-of-8 turns. The heel is sometimes raised upon a pillow so as to extend the leg and to semiflex the thigh, but this is not essential. Remove and reapply the dressing every few days, as it inevitably becomes loose. At each removal employ massage. At the end of three weeks remove the splint permanently and apply a plaster-of-Paris dressing from just above the ankle to the middle of the thigh, and get the patient about on crutches. Have the plaster cut so that it may be easily removed and every day employ massage and gentle passive movements, the surgeon fixing the upper end of the upper fragment by his thumb during the movements. The dressing is to be worn for five weeks. After eight weeks of treatment allow the patient to walk about on crutches, the joint being left free at night, but kept fixed during the day by pasteboard splints or by a light plaster-of-Paris bandage. After four weeks more he gets about with canes or a cane. For months after removing the splints and plaster a lacing knee-cap of leather should be worn in the daytime to support the joint. The plan of prolonged immobilization render more or less muscular atrophy and joint-stiffness certainties, but there are less serious impediments

than the wide separation of the fragments that inevitably attends an early use of the joint. Bryant, of New York, has devised an ambulatory dressing.

*Operative Treatment.*—Malgaigne's hooks are obsolete.

It is said that John Rhea Barton wired an ununited fracture of the patella in 1843. In 1877 Hector Cameron wired an ununited fracture of the patella, and a few months later Lord Lister operated on a fracture of the knee-cap two weeks after the accident. The question of the advisability of suturing a recent fracture has been very much disputed. The ordinary non-operative plans of treatment do not endanger life and generally give a fairly good functional result, although the joint remains insecure on extension, the patient is apt to fall, and a fall may refracture the bone. The operative method will usually succeed, and is capable of obtaining a better functional result and of obtaining it more rapidly. There is some danger of infection, and if infection should occur, the results may be most disastrous. Some cases obviously cannot be treated by the conservative method with any chance of success; cases, for instance, in which a flap of fibroperiosteum intervenes between the fragments, or cases in which from some other cause the bones cannot be approximated. Such cases should, of course, be operated upon. But in the great majority of cases a good result will follow conservative treatment, and conservative treatment should be trusted unless the case is in the hands of a surgeon and in a place where every antiseptic precaution can be taken. We agree with Stimson when he says that operative methods can be used with confidence when surrounded by every protection; he habitually uses them, but he never teaches

Fig. 369.—Needle specially designed to carry a thick wire. The eye is drilled obliquely, and should receive only a little loop on the end of the wire; this loop should be made previously.

them as proper routine practice, and strongly advises against their use except by those who have had experience in operating, who have formed the habit of taking precautions, and who have the aid of skilled assistants.[1] Operation should only be performed on healthy persons of suitable age, when the separation is over $\frac{1}{2}$ inch or when there is much laceration or interposition of the capsule.[2] If a patient is still able to extend the limb and the lateral expansions of the quadriceps have not been widely torn, a useful limb will be obtained by conservative treatment even if the bone-fragments separate, and operation is not demanded. A working man needs the operation more than a gentleman of leisure because he is in more vital need of a sound knee-joint. A young or middle-aged person is more active than an elderly person, hence, in him operation is more strongly indicated than in an elderly man. Barker believes strongly in wiring recent transverse fractures. He does it with antiseptic care soon after the accident, and permits passive motion or even slight active motion immediately after the operation. Massage is begun the day after the operation, and is practised daily for two weeks.

Barker[3] uses a special needle (Fig. 369) and silver wire of the thickness of a No. 1 English catheter. This wire is straightened and softened in a spirit-flame. He rubs the bone-fragments together in order to dislodge blood or fibrous

[1] "Annals of Surgery," August, 1898.

[2] Powers, in "Annals of Surgery," July, 1898.

[3] See the objections of Sir William Stokes to Barker's method, in "Brit. Med. Jour.," Dec. 3, 1898.

material, and when marked grating occurs, introduces the wire. A puncture with a small knife is made through the middle of the upper attachment of

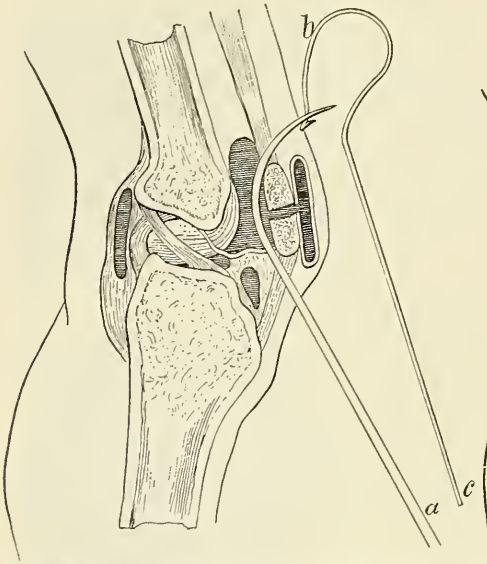



Fig. 370.—Needle (*a*) introduced behind the fragments, and receiving one end (*b*) of the silver wire (*b*, *c*) (Barker).

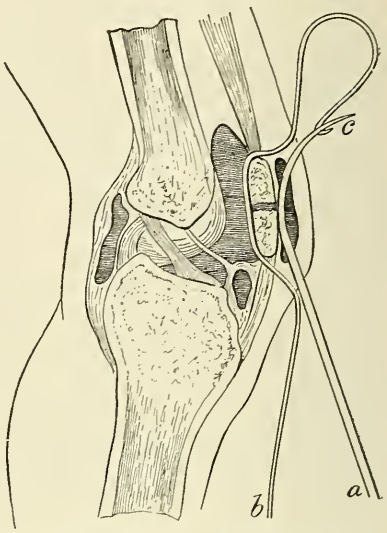



Fig. 371.—Needle (*a*) passed in front of the fragments and receiving the other end (*c*) of the silver wire (*b*, *c*) (Barker).

the patellar ligament. The needle, not carrying any wire, is made to enter through this opening into the joint, is passed back of the fragments, pierces the tendon of the quadriceps at the upper edge of the upper fragment, and its point is cut upon with a knife. The wire is inserted into the eye of the needle and the needle is withdrawn and unthreaded. The empty needle is pushed through the lower opening, is carried in front of the patella, is made to emerge at the upper opening, is threaded with the protruding wire and withdrawn (Figs. 370, 371). The wires are threaded into bars and twisted (Fig. 372), the ends are cut off, and antiseptic dressings are applied. There are objections to Barker's operation: It does not allow us to remove blood-clots from the joint; if a bit of tissue intervenes between the fragments, it cannot be removed; and a foreign body is left permanently in the joint.[1] If an operation is thought advisable, we deem it best to do an open operation, making a semilunar or a central longitudinal incision, freeing the joint from blood-

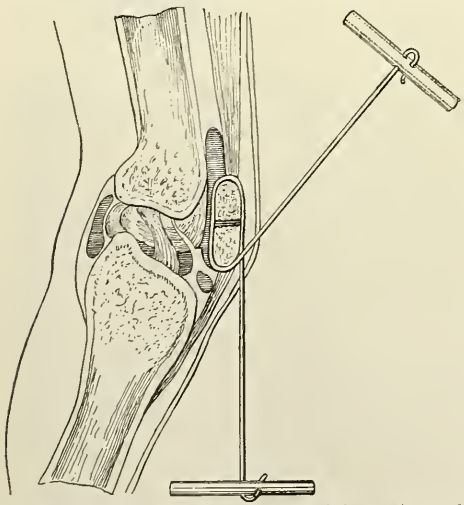


Fig. 372.—Wire in position around fragments and threaded through metal bars. The lower and posterior wire runs upward to the left of the upper, ready for twisting (Barker).

[1] "Brit. Med. Jour.," April 11, 1896.

clots by irrigation with hot salt solution, removing all tissue from between the fragments, drilling the fragments, passing silver wire, twisting the wire and drawing the fragments together, and closing the wound (Fig. 373). Instead of wire, silk may be used. In cases in which there is no very strong tendency to separation the fragments can be held together by several catgut sutures through the periosteum at the fractured edges or by a strong catgut suture passed through the ligamentum patellæ and the quadriceps tendon and carried in front of the fracture (Stimson). The limb should be placed on a posterior splint. In seven or eight days the superficial sutures are removed and a plaster-of-Paris splint is applied. In a few days the patient gets about on crutches.

Fig. 373.—Wired fracture of the patella (St. Joseph's Hospital case; operated upon and skiagraphed by Dr. Nassau).

In a month the dressing is cut down the front and worn only in the daytime, and passive motion is begun. The splint is discarded at the end of the third month.[1] Among other operative procedures we may mention the following: Encircling the fragments by a silk suture (the circumferential suture). This suture may impair bone nutrition and retard union. Ceci drills the bones subcutaneously and passes wire through the drill-holes in the form of a figure of 8. Passing subcutaneously a ligature around and over the fragments (Butcher). Incision and approximation of the fragments by fixation hooks or metal pins.

[1] Stimson, "Annals of Surgery," August, 1898.

**Fractures of the patella by direct force** are vertical, stellate, oblique, V-shaped, or transverse; are often incomplete and occasionally compound or comminuted (Fig. 374). Ransohoff maintains that fractures from direct force are more common than from muscular action and are usually transverse and associated with some comminution ("Jour. Amer. Med. Assoc.," Oct. 13, 1906).

*Compound fracture* of the patella is very rarely seen in Philadelphia hospitals. The records of the Boston City Hospital (an institution in which multitudes of fractures are treated) show only 8 compound fractures of this bone in forty-two years (Scannell, in "Boston Med. and Surg. Jour.," Nov. 15, 1906).

*Symptoms of Simple Fracture.*—Fractures of the patella by direct force are followed by discoloration, swelling, great difficulty in movement, and much pain. There may or may not be crepitus. The degree of separation of the fragments depends upon the direction of the line of fracture and the extent of bone involved. Bony union is apt to occur after such a fracture when there was not wide separation.

*Treatment.*—A fracture resulting from direct force may often be treated by a posterior splint and the application of a bandage. If there is any separation, the fragments should be approximated by adhesive strips, bandages, and compresses. At the end of three weeks remove the posterior splint, apply a plaster-of-Paris splint, and get the patient about on crutches. The danger in these cases is ankylosis rather than non-union; hence, in the fourth week cut the plaster splint down the front and begin passive motion of the knee-joint. At the end of six weeks cease wearing the dressing in the daytime, and at the end of three months discard it entirely. In those cases in which an oblique fracture or a transverse fracture with wide separation arises from direct force, treat as advised for transverse fracture from muscular action. The question of operation is practically the same as for transverse fracture from muscular action. In every compound fracture of the patella, if amputation can be avoided, incise the soft parts freely and irrigate the joint with hot saline fluid. Remove hopelessly loosened fragments. Those not completely separated may in some cases be sutured into place. Drain for twenty-four or forty-eight hours.

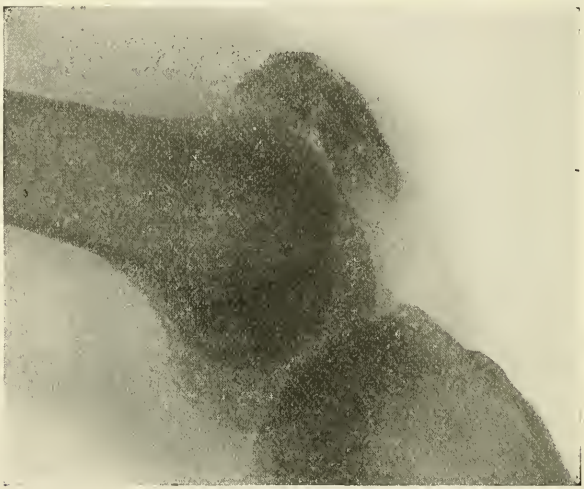


Fig. 374.—Comminuted fracture of patella.

**Ununited and Badly United Fracture of the Patella.**—There is usually a band of union, but it may be very thin and the fragments may be far asunder. It is commonly taught that the degree of functional impairment depends

directly on the amount of separation. This is not strictly true. There may be great separation and but little impairment of function, the fragments being firmly united by a dense fibrous band. There may be little separation and yet lameness, stiffness of the joint, and imperfect power of extension. The reason for this has been pointed out by Bruns, of Tübingen.[1] He says there may be complete failure of union, even when the separation is trivial, and failure of union produces impaired function. If separation is considerable, the fragments are apt to tilt and tissue is often interposed between them. Functional difficulty is more often met with when the fragments are far apart than when they are near together, because non-union is more common. Even if non-union occurs, in some cases the quadriceps is still able to act upon the tibia by means of the fascia lata, ligaments at the sides of the joint, or bands from the vasti to the lower fragment. Besides non-union, functional impairment may be due to anchoring of the upper fragment to the femur. The upper fragment may be anchored to the femur by the interposition of the fibrous investment of the knee-cap, which covers the fractured surface of the upper fragment and may grow fast to the capsule of the joint (Bruns).

The *treatment* of ununited and badly united fracture is discussed on page 534.

**Fractures of the Leg.**—In leg-fractures both bones or only one bone may be broken.

**Fractures of the tibia** are divided into: (1) fractures of the upper end; (2) separation of the upper epiphysis; (3) fractures of the shaft; (4) fractures of the lower end, and (5) separation of the lower epiphysis.

**Fractures of the upper end of the tibia** are uncommon. They may be transverse, oblique, or vertical, running into the joint. The *cause* is direct violence.

*Symptoms.*—In fracture of the upper end of the tibia there is contusion of the soft parts. In a *transverse* fracture there are mobility and crepitus, but there is little displacement. In *oblique* fracture crepitus and mobility are marked, the axis of the limb is altered, and the fragments may be displaced. In fractures entering the joint there is great swelling of the knee-joint. *Comminuted* fractures exhibit marked signs, union is readily obtained, but if the joint has been damaged, stiffness is sure to ensue.

*Treatment.*—Reduce displacement by extension and manipulation. The special apparatus used for treatment depends on the case. In some cases extension is required, in some a posterior splint is applied and the limb is suspended from a gallows, in some a double inclined plane is employed, and in some a plaster-of-Paris splint is used.

The double inclined plane in the form of McIntyre's splint is frequently employed, or a double inclined plane in the form of a fracture-box may be preferred. The extremity should be immobilized for four weeks, when passive motion should be begun. Passive motion is to be made daily, the dressing being reapplied after each séance. In five or six weeks the dressings are removed and the patient allowed to go about on crutches. The crutches are soon abandoned for a cane, and later all support is dispensed with. If a fracture extends into the knee-joint and the ill-adjusted fragments block the articulation, the joint should be opened and the fragments placed and fixed in proper position.

**Separation of the tubercle of the tibia** is due to violent contraction of the quadriceps, and occurs only in those under twenty years of age. The fragment is drawn up and can be felt, and the patient is unable to use the limb. In a case in which the tibial spine has been torn off, it may be nailed in place,

[1] "Beiträge zur klinischen Chirurgie," "Mittheilungen aus der chirurg. Klinik zu Tübingen," Bd. iii, Heft 2, 1888.

or the limb should be placed on a posterior straight splint and the fragment should be pulled down into place by adhesive strips and bandages. The splint should be worn for five weeks.

**Avulsion of the Spine of the Tibia.**—This is a very rare accident. There are but 4 cases on record, and in only 1 of them (Pringle's) was the diagnosis made during life, and it was made in that case by exploratory incision. The tibial spine is torn off by the anterior crucial ligament. The causative force is probably flexion, abduction, and internal rotation of the leg (J. Hogarth Pringle, "Annals of Surg.," August, 1907). After the acccident the leg at the knee is in extreme abduction. Exploratory incision may be necessary for diagnosis. The treatment is to suture or nail the bone-fragment in place.

**Separation of the Upper Epiphysis of the Tibia.**—This is an injury of extreme rarity. It does not seem to occur after the sixteenth year. It is caused by a twist or by violent abduction or adduction of the leg. It may lead to lessened growth of the limb. The *treatment* is as for a fracture of the upper end of the bone.

**Fractures of the Shaft of the Tibia.**—The *causes* of these fractures are direct force, indirect force, or torsion. A fracture in the upper part of the bone is usually transverse; in the lower part it is usually oblique (T. Pickering Pick).

*Symptoms.*—In transverse fracture of the shaft of the tibia there is no deformity, and the support of the fibula may even permit of walking; there is fixed pain; there may or may not be inequality of the fragments felt by the finger; and there are pain, tenderness, crepitus, mobility, and often linear ecchymosis. In oblique fractures there usually exist crepitus, a little mobility, and distinct deformity. The deformity depends on the direction of the line of fracture, and, as this line is usually from above downward, inward, and a little forward, the lower fragment usually passes behind the upper fragment and rotates inward.

*Treatment.*—In treating fractures of the shaft of the tibia effect reduction by making extension from the foot and counterextension from the knee, the knee-joint being in partial flexion. If there is much swelling, put the limb in a fracture-box (Figs. 375, 376, and Pl. 7, Fig. 1), swing the box from a gallows, and apply an ice-bag for twenty-four hours. A silicate of sodium or a plaster-of-Paris dressing is applied when the swelling subsides, or the dressing may be used at once instead of a fracture-box if swelling is slight. As soon as the limb is immobilized in a silicate or plaster dressing the patient gets about on crutches. The dressing is removed after five weeks, and the patient goes about for one week on crutches, lightly using the foot, and then for a time walks with the aid of a cane. At the end of eight or nine weeks the cane may often be dispensed with, the amount of use of the leg being daily augmented.

**Fractures of the Lower End of the Tibia: Fracture of the Inner Malleolus.**—The *cause* of fracture of the inner malleolus is direct force or traction upon the internal lateral ligament.

*Symptoms and Treatment.*—The *symptoms* of fracture of the inner malleolus are pain, tenderness, some downward displacement, depression above the ends of the fragments, mobility, and crepitus. The *treatment* is to push the fragments into place and use side-splints or a fracture-box for two weeks, when a plaster-of-Paris or a silicate dressing may be substituted and the patient ordered to use crutches. Remove the plaster four or five weeks after it is applied, and direct the patient to gradually bear his weight upon the leg, as outlined above.

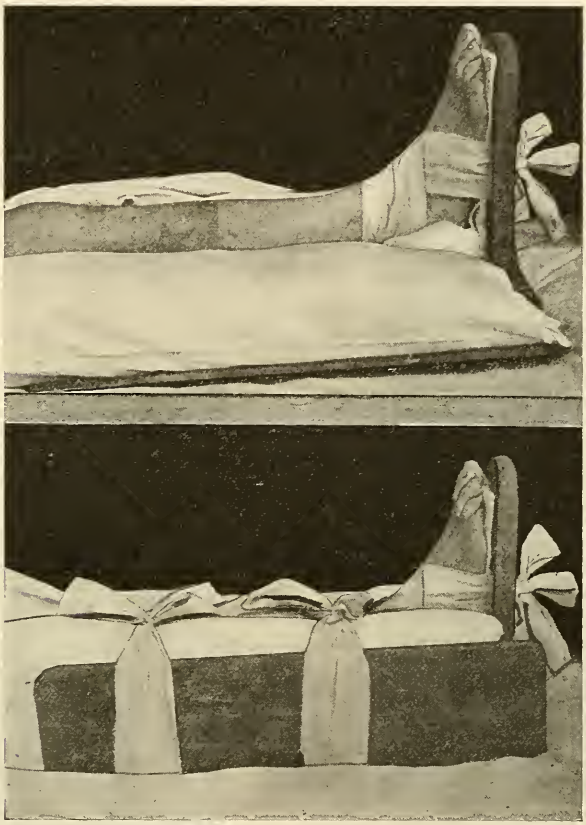
**Separation of the lower epiphysis of the tibia** is a rare accident, but is commoner than separation of the upper epiphysis. The *treatment* is a fixed dressing for six weeks.

**Fracture of the fibula** alone is commoner by far than is fracture of the tibia alone. Fractures in the upper two-thirds, which are rare, are usually

due to direct force. Fractures in the lower third are frequent, and arise from indirect force.

**Fractures of the Upper Two-thirds of the Fibula.**—In these fractures the *cause* is direct force.

*Symptoms.*—In fracture of the upper two-thirds of the fibula the patient is frequently able to walk. The bone is deeply situated, and displacement cannot often be detected. There is a fixed pain, which is intensified by movement and by pressure. Pressure upon the lower fragment does not move the upper fragment. Crepitus is sometimes obtained, and linear ecchymosis is



Figs. 375, 376.—Fracture-box in fractures of the bones of the leg.

apt to appear. The bone is normally elastic, hence slight mobility is of no value diagnostically.

*Treatment.*—In treating a fracture of the upper two-thirds of the fibula apply a plaster-of-Paris or a silicate bandage and direct that it be worn for five weeks. Weight is not to be put upon the foot for six weeks after the accident.

**Fractures of the Lower Third of the Fibula.**—In these fractures the *cause* is usually indirect force, especially twists of the foot. Forcible inversion of the foot pulls upon the external lateral ligament and the external malleolus, forces the fibula outward, and tends to break it, the lower fragment being displaced outward. Forcible eversion pulls the internal lateral ligament off from the inner malleolus (often breaks the malleolus) and fractures the fibula above the ankle, the bone being displaced inward.

**Pott's Fracture.**—By the term "Pott's fracture" is meant a fracture of the lower fifth of the fibula produced by eversion and abduction of the foot. Stimson points out that the production of Pott's fracture is often aided by the weight of the body. The lesions which arise depend upon whether the chief force is eversion or abduction. "If eversion is the sole, or main, movement, the force is exerted through the internal lateral ligament and breaks the internal malleolus squarely off at its base; then it presses the external malleolus outward, rupturing the tibiofibular ligament, and breaks the fibula close above the malleolus. Sometimes instead of pure rupture of the tibiofibular ligament there is avulsion of the portion of the tibia to which it is attached."[1] Stimson further points out that if abduction is the preponderating force there is an oblique fracture of the anterior portion of the internal malleolus or more frequently rupture of the anterior portion of the internal lateral ligament. There are, as in the former case, rupture of the tibiofibular ligament and an oblique fracture of the fibula several inches above the external malleolus. It is evident that the degree of injury produced by eversion and abduction depends on the point at which the force is arrested. It may be arrested after the inner malleolus has been

Fig. 377.—Pott's fracture. Dupuytren's splint. Note length of splint; position of straps; arrangement of padding; space between foot and splint (Scudder).

Fig. 378.—Pott's fracture.

separated or the anterior fibers of the deltoid ligament torn, and in this case the tibiofibular articulation remains intact and the fibula is not broken. It may cease after separating the tibiofibular articulation, and in this case too the fibula escapes. It may be continued until the fibula breaks. In this fracture the astragalus passes outward, somewhat backward and also upward, the later deviation being due to separation of the tibiofibular articulation. The chief trouble in reduction and treatment is this outward and backward dislocation of the foot.

*Symptoms.*—The foot is displaced outward, and a little backward and upward, and the inner malleolus or the tibia from which it was torn is ex-

[1] "A Practical Treatise on Fractures and Dislocations," by Lewis A. Stimson.

tremely prominent. There is great lateral mobility and often anteroposterior mobility at the ankle-joint. Stimson points out that there are three points where pressure is certain to provoke pain: in front of the tibiofibular ligament, at the base or anterior border of the inner malleolus, and over the seat of fracture through the fibula.

*Treatment.*—Thorough reduction is of the greatest importance. If thorough reduction is effected, a good result will probably be obtained; but if thorough reduction is not effected, the patient will be permanently crippled to a greater or less extent. In order to effect reduction it may be necessary to anesthetize the patient. The patient's knee is flexed, the heel is pulled strongly forward and inward, and the lower end of the tibia is pushed backward. This corrects the ankle dislocation, and then the valgus must be overcorrected (Jones). Inversion of the ankle is imperative.

Some surgeons at once after reduction apply a plaster-of-Paris bandage. This treatment is objectionable because the deformity may be partially reproduced after the application of the dressing, the surgeon being unable to see it and unable to correct it.

If there seems to be no strong tendency to a recurrence of deformity, a fracture-box can be used. After reducing displacement in such a case, place the limb in a fracture-box containing a soft pillow. A bird's-nest pad of cotton or oakum is made for the heel (see Figs. 375, 376). A fillet around the ankle fastens the foot to the foot-piece of the box; a pad of oakum rests between the foot-piece and the sole. A compress is placed below the outer malleolus and another one above the inner malleolus. Close the sides of the box and tie them together with a bandage, and swing the box on a gallows. Every day let down the sides of the box and rub the leg, the ankle, and the foot with alcohol. In ten days apply a plaster-of-Paris bandage and let the patient get about on crutches. Remove plaster at the end of the fifth week after the accident, and let patient go about with crutches for one week and with a cane for a week longer.

I am accustomed to dress most cases of Pott's fracture with a *Dupuytren splint.* This is a straight splint (Fig. 377 and Pl. 7, Fig. 9) which reaches from above the head of the tibia to below the sole of the foot. This splint is padded, and a pyramidal pad with the base down is laid upon the inner surface of the leg, above the inner malleolus, the splint being put upon the inner surface of the leg, over the pad. The splint is fastened as shown in Plate 7, Fig. 9, and Fig. 377. If the short splint shown in Plate 7 is used (it only goes to the head of the tibia), the leg is semiflexed upon the thigh and is laid upon the outer surface on a pillow. After ten days of Dupuytren's splint, apply the plaster-of-Paris bandage, which is to be worn as above directed. Bryant treats Pott's fracture with a posterior splint, two lateral splints, and a swing. Stimson uses a posterior and lateral splint of plaster of Paris. This splint does not slip, as may Dupuytren's dressing, and does not hide the seat of fracture from view, as does complete encasement with plaster of Paris. It is a most useful dressing. When the patient begins to walk the callus may bend. If it does, eversion will be produced. In order to prevent this make the patient walk for a number of weeks upon a shoe heel raised on the inside in order to induce the position of varus. A very heavy man should also wear for several weeks an iron brace on the outside. I have given above the conservative treatment of Pott's fracture. I have come to the conclusion that operation should be the rule in this injury and not the exception. The fracture is so often followed by pain, swelling, and disability. Pain and disability are so largely due to malunion or fibrous union of the broken malleolus, and it is so easy to fix the fragment in place by nails or sutures, that the conclusion seems obvious that in proper subjects operation should be performed. As Heath and Selby show, in an important article ("Annals of Surg.," Jan.,

1908), operation also allows us to return the tibialis posticus to its normal position and thus flat-foot is prevented. The plan suggested by Heath and Selby ("Annals of Surgery," Jan., 1908) is excellent. The fragment may be sutured in place by silver wire or chromic gut. If the tendon of the posterior tibial is displaced, the torn annular ligament is sutured so as to hold the tendon in place, a drain of rubber tissue is carried down to the bone, and the soft parts are sutured. The parts are dressed and a fixation apparatus is applied. The drain is removed in twenty-four hours. In eight weeks the patient should be walking freely (see cases reported by Heath and Selby, Ibid.). Pott's fracture may be *compound*, a portion of the inner malleolus or of the tibia projecting through the wound. If it is necessary to introduce through-and-through drainage, the foot must be placed and kept at a right angle to the leg. If a compound fracture exists, the malleolus must be nailed in place or sutured by silver wire or chromic gut. In a reported case the wire was passed through the joint and around the fragment, and the result was good.[1] Nailing seems a better plan.

Fig. 379.—Fracture of both bones of leg. Bad position.

**Fracture of both bones of the leg** is a very common injury, is often compound, and is not unusually comminuted. Fractures by direct force, such as blows or kicks, are commonest in the upper half of the leg. Fractures by indirect force, as by falls, are commonest in the lower half of the leg. In fractures from indirect force the tibia breaks first, and then the fibula breaks at a higher level. The point of greatest liability to fracture from indirect force is the junction of the lower and middle thirds. Fractures of the leg are usually oblique, but they may be transverse if arising from direct force. Spiral, torsion, or V-shaped fractures and longitudinal breaks sometimes occur. In oblique fractures, as a rule, the line of fracture runs from behind, downward, inward, and a little forward.

*Symptoms.*—Fracture of both bones of the leg is easy of recognition. The fibular fracture is detected as before described. By running the finger along the crest of the tibia displacement will be found, except in transverse fractures, when it may not occur. The common displacement is for the lower fragment to ascend and pass behind the lower end of the upper fragment and to rotate a little outward, and for the upper fragment to project in front. The ascent of the lower fragment is due to the action of the gastrocnemius and soleus muscles. If the line of fracture is in a direction the reverse of that which is usual, the lower fragment ascends in front of the lower end of the upper fragment. In fracture of both bones of the leg there are marked mobility and crepitus, severe pain, and inability to walk. In fractures from direct force there is more or less damage to the soft parts. A fracture of the shaft of the tibia near the ankle is distinguished from a

[1] "Rev. de Chir.," vol. viii, 1888.

dislocation by the fact that the deformity is easily reduced, but tends to recur in the fracture, and, further, that in a fracture the relations of the malleoli to the tarsus are unaltered, whereas in a dislocation they are altered.

*Treatment.*—If the fracture is near the ankle-joint, the action of the tendo Achillis may maintain deformity, and in such cases the tendon should be divided. All fractures of the lower third are difficult to reduce and often require extension by the pulley. In very few will perfect apposition of the fragments be brought about, and the best we can usually attain is good alignment (Jones). In treating a simple fracture of the lower two-thirds of the bones reduce by extension and counterextension, and use a fracture-box (see Figs. 375, 376). The compresses used in Pott's fracture are not required. If the soft parts are bruised, use evaporating lotions for a day; if they are abraded, apply antiseptic dressings. The fracture-box should be swung upon a gallows. After three weeks apply a plaster-or-Paris or silicate of sodium dressing and let the patient sit up in a chair daily during one week; at the end of this time the patient may get about on crutches. At the end of six weeks after the accident remove the plaster, and let the sufferer go about on crutches for two weeks and with a cane for two weeks more. Brinton was accustomed to dress a fracture of both bones of the leg for two weeks in a fracture-box, for two weeks in side-splints made of metal, and for two weeks in an immovable dressing, allowing the patient to get about on crutches as soon as the plaster is put on. Instead of the fracture-box we may use a posterior splint, two lateral splints, and a swing. The Thomas bed-splint is a very useful dressing. Nathan R. Smith's anterior splint is used by some in the treatment of fractures of the leg. Some surgeons apply plaster of Paris in the form of an ambulatory dressing. In this dressing a solid apparatus reaches to the lower third of the thigh and below the sole of the foot. When the patient walks the weight is transmitted to the thigh (see Figs. 274 and 275). In fractures of the upper third of the leg the McIntyre splint or the double inclined plane is used. If the fracture is *compound*, asepticize thoroughly, make a counteropening, insert a drainage-tube, dress with bichlorid gauze, apply a plaster bandage, and cut trap-doors over the openings of the tube (see Fig. 279), or dress with the bracketed splint and plaster of Paris (see Fig. 280). Remove the tube, as a rule, in about forty-eight hours; but the patient's temperature is the guide, not time of retention.

In many fractures of the lower third operation is indicated to accomplish reduction and fixation.

**Fractures of the bones of the foot** are rather rare accidents, although not so unusual as we once thought, for the *x*-ray has taught us that a considerable number of supposed sprains are, in reality, fractures. Owing to the number of the bones and to the elasticity of their connections the force of blows and falls is spread and dissipated. The bones most often broken are the astragalus and the os calcis. Fractures from direct force are often compound. The *cause* of fracture of either the scaphoid, the cuboid, or one of the cuneiform bones is direct force. Simple fractures of the os calcis and astragalus may arise from crushes or twists of the foot, but result, as a rule, from indirect force, such as falls. The calcaneum may be broken by a direct blow. In rare instances the os calcis has been broken by contraction of the great calf-muscles. Forcible dorsal flexion of the foot may fracture the neck of the astragalus (Eisendrath). Compound fractures may result from gunshot-wounds, crushes, and falls.

*Symptoms.*—The history of the nature of the accident is of great importance. In fracture of the os calcis there are severe pain, tenderness, swelling, crepitus, mobility, often an apparent widening of the bone, and not unusually a loss of the arch of the foot (Pick). In some cases the posterior fragment is drawn up by the calf-muscles, and in other cases there is no deformity. In fracture

of the astragalus displacement may occur which resembles that of a dislocation. Crepitus may or may not be detected. It can be elicited, as a rule, by rotating the foot while the heel is firmly held. If crepitus cannot be detected we are not certain that a fracture is present, even though the patient may be unable to stand and there are swelling and pain on pressure. The malleoli may seem on a lower level than normal if the astragalus and os calcis have been crushed. Sometimes the foot is shortened and perhaps the fragments have been dislocated (Eisendrath, in "Annals of Surg.," March, 1905). The *x*-rays will make the diagnosis certain. Fractures of the other bones of the tarsus are difficult of detection except by the *x*-rays. There may or may not be crepitus, which, if it exists, is difficult to localize; there is pain on standing and on pressure, and there is bruising of the soft parts.

*Treatment.*—In simple fracture of the os calcis and astragalus without displacement place the foot at a right angle to the leg and apply a plaster cast. This is cut down the front so that it may be removed easily. On the third or fourth day follow Eisendrath's advice and begin massage to reduce swelling and prevent muscular atrophy (Ibid.). The cast is worn for eight weeks, when the patient may begin to put weight upon the extremity. If a flat-foot has resulted from the accident, a support must be worn (see page 742). If there is displacement in a simple fracture of the os calcis or astragalus it is wisest to operate. Perfect correction is not possible otherwise and no apparatus is satisfactory. The fragments are restored after incision and may be nailed or wired in place. A fragment may require removal or the badly splintered bone itself may have to come away. If the tendo Achillis is torn loose, it should be sutured to the os calcis. Fractures of the other bones of the tarsus are almost always compound, and the injury may require drainage and immovable dressing, excision of bones, or even amputation. If they are not compound, they may be treated by a plaster-of-Paris dressing or may require incision and fixation or removal.

Fig. 380.—Fracture of metatarsal bones.

**Fractures of the metatarsal bones** are almost invariably due to direct force and are almost always compound. Robert Jones has published skiagraphs of a fracture of the fifth metacarpal bone from indirect force. Crepitus may be absent because of impaction or fixation by interosseous ligaments. Jones says such a fracture may be produced by the pressure of the body weight on an inverted foot the heel of which is raised ("Annals of Surgery," June, 1902). When only one bone is broken, displacement is slight, there is severe pain on motion and pressure, and crepitus can generally be obtained. Pain is produced by flexing the toes, putting weight upon the toes, as in walking, and by inverting or everting the foot. Fracture of the third metatarsal is apt to destroy the arch of the foot. A simple fracture of a metatarsal bone is treated by an immovable dressing for four weeks. Fractures from crushes usually demand excision or amputation.

**Fractures of the phalanges of the toes** are due to direct force and are often compound. They may require immediate amputation.

*Treatment.*—In a compound fracture where amputation is unnecessary, drain with strands of catgut for forty-eight hours and dress antiseptically, at the end of this time apply over the bichlorid gauze a gutta-percha or a pasteboard splint extending from beyond the end of the toe to well up upon the sole of the foot, and fix the splint in place with a spiral bandage of the

toe and instep. The splint is to be worn for four weeks. In a simple fracture fasten the injured toe to an adjacent toe or toes by a plaster bandage and wear the dressing for three weeks.

## Diseases of the Joints

**Synovitis** is a primary inflammation of the synovial membrane alone. If other structures besides the synovial membrane are involved the condition is known as "arthritis." Two forms of simple synovitis exist—namely, *acute* and *chronic*. Some surgeons speak also of *subacute* cases.

**Acute Simple Synovitis.**—The *causes* of acute simple synovitis are contusions, sprains, twists, and overuse. The causative influence of exposure to cold or damp has been much debated. It seems probable that in some cases cold produces vasomotor paresis of the vessels of the synovial membrane, a condition which may be followed by inflammation. In synovitis the synovial membrane is red and swollen, and the joint contains an excess of turbid fibrinous fluid. If the inflammation advances, arthritis arises and sometimes blood is effused.

*Symptoms.*—A prominent symptom of acute synovitis is pain, which is increased by motion of the joint, by pressure upon the articulation, and by a dependent position of the limb. The pain is worse at night. Pressure upon the edge of the cartilage does not cause pain, but friction of the synovial membrane at once develops it. The patient places the limb in the position which gives the greatest ease, and the part becomes more or less fixed in this position because the muscles about the joint are rigid. A fluctuating swelling is noted in a superficial joint, most marked between the ligaments, which swelling bulges out the synovial area and hides or obscures the articular heads of the bones. The swelling is due early to excessive secretion of synovia, and later to effusion of liquor sanguinis. Bulging takes place at points where the capsule is thin, and at such points fluctuation may be detected. Fluctuation in the elbow is sought for posteriorly. Fluctuation in the knee is sought for on either side in front. A large effusion in the knee floats the patella up from the condyles (*floating patella*). A small effusion in the knee can be detected by Fiske's plan, which is as follows: Tell the standing patient to bend forward at the hips, resting each hand on the front of the corresponding thigh. The anterior structures of the joint are thus relaxed, and, by tapping the patella, even a small effusion can be discovered. Bulging cannot be distinctly recognized in the hip or shoulder unless effusion is great. The skin over the joint is rarely reddened, but feels hot to the hand of the observer (over superficial joints, but not over the shoulder and hip); the joint is partly flexed; fever exists, varying in degree with the size of the joint, the acuteness of the attack, and if infection occurs fever is a striking feature. Suppuration rarely follows simple synovitis, but it may do so, the area of synovitis being a point of least resistance to organisms carried by the blood or lymph. If suppuration takes place, rigors occur, there is a septic temperature, and the joint soon gives evidences of containing pus. These evidences are violent pain, increased tenderness, dusky discoloration if the joint be superficial, greater muscular spasm, periarticular edema, and constitutional symptoms of sepsis. Traumatic synovitis without infection tends toward cure without suppuration if the patient is healthy, and after it ankylosis is rare.

*Treatment.*—In treating acute synovitis immobilize the joint. In severe cases place it in such a position that the limb will still be useful even if ankylosis occurs. In mild cases immobilize in the position of rest, apply leeches, and use the ice-bag or the Leiter coil. After a day or two apply gentle pressure, intermittent heat, and iodin and ichthyol. If the effusion is very great and persistent, and pressure, heat, and sorbefacients fail to remove it, aspirate with

aseptic care. If effusion recurs after aspiration, use massage and the hot-air oven, or apply plaster-of-Paris dressing or use flying blisters. If there is any evidence of infection, follow Murphy's advice, that is, aspirate the joint and inject a mixture of formalin in glycerin (2 per cent.). As a rule this treatment will rapidly bring about a cure. The mixture of formalin and glycerin should have been made at least twenty-four hours before use. A rubber bandage is often useful toward the termination of a case of acute synovitis.

**Chronic synovitis** follows acute synovitis or it may be chronic from the start. Many cases called chronic synovitis are, in truth, tuberculous disease. The synovial membrane looks nearly natural, but is edematous, and the joint contains an excess of fluid. If the quantity of fluid is large, the disease is called *hydrops articuli*, or *dropsy*. A large amount of fluid in the knee-joint "floats" the patella upward. Tuberculous infection may occur in very prolonged cases. In prolonged tuberculous synovitis the synovial membrane may thicken in some places, soften in others; it is often adherent, and the villous processes hypertrophy. If the membrane becomes extensively softened (*pulpy degeneration*), the softened areas bulge and caseation eventually occurs. In the knee-joint a traumatic synovitis is sometimes linked with *inflammation of the semilunar cartilages*. Roux tells us that this inflammation may be produced by a squeeze, a twist, or a direct force, but a squeeze is the common cause. Hyperextension of the knee may squeeze the cartilage, and so may attempting to rise from a stooping posture.[1] If this injury has taken place, the disability will be prolonged.

*Symptoms.*—In chronic synovitis pain may be absent or may be present only during exercise or from pressure, and be slight even then. There may be seizures of pain; there is some limitation of movement; passive motion may develop creaking or joint-crepitus; fluctuation is apparent and there is atrophy in the muscle about the joint. The atrophy of the muscles associated with an inflamed joint is a reflex atrophy and is named after Charcot. The aspirating needle will give exit to a viscid straw-colored or bloody fluid unless the material is gelatinous.

*Treatment.*—Rest and pressure are of great service. Pressure may be obtained by the application of Martin's rubber bandage. A plaster-of-Paris dressing is probably the best way to combine rest and compression. Massage, douches, frictions, passive movements, and flying blisters should be used. Painting the joint with iodin and spreading over it blue ointment, and rubbing in ointment of ichthyol (50 per cent. with lanolin) may do good. Counter-irritation by the actual cautery is a valuable expedient. The Bier treatment (see page 112) is often of benefit. Injections of dilute carbolic acid in the parts about the joint rapidly relieve the pain (see page 640). Some surgeons advise aspiration, washing out with salt solution, injecting a 5 per cent. solution of carbolic acid or formalin-glycerin (2 per cent.), and immobilizing. Incision and drainage constitute a radical but proper plan in cases unamended by simpler methods. If pulpy degeneration exists, perform an excision or an erasion. If pus forms, incise at once and drain. Internally, treat any existing diathesis and give nutritious food, tonics, and stimulants. Chronic synovitis is often greatly benefited by the use of a hot-air apparatus. The limb is wrapped in flannel and is placed in an oven. The oven is heated by Bunsen burners. The temperature is raised to 250° or even 300° F., and the limb is subjected to this for one hour. The oven should be used daily, and as the patient becomes accustomed to it even a higher degree of heat can be tolerated. This high degree of heat can be borne only when it is perfectly dry. Any moisture scalds the patient. The Lentz oven has in it ventilation openings to get rid of moisture and the sweat is taken up by the flannel. The

[1] "Gaz. des Hôp.," No. 125, 1895.

flannel must not be applied so thickly as to keep the heat notably from the joint, nor must so little of it be used as to permit of its soaking with sweat. Fig. 381 shows the Sprague hot dry-air apparatus. Dr. H. A. Wilson inserts in the oven humidin, a product obtained in the purification of salt, which material absorbs the moisture entirely. Cotton should not be used to wrap the limb, because, if the bottom of the oven becomes very hot, the cotton may ignite and burn the patient. A physician or nurse should constantly watch the apparatus during its employment.[1] Bier's box is of wood lined with asbestos. It is heated through a flue by gas or alcohol lamps. Aspiration and the subsequent use of a plaster-of-Paris bandage may be tried in lingering cases of chronic synovitis.

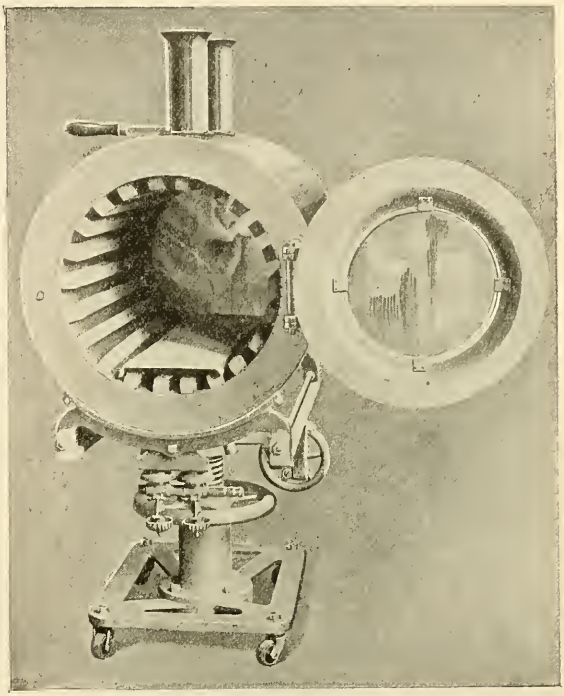


Fig. 381.—Sprague hot dry-air apparatus.

**Arthritis.**—By this term is meant not only inflammation of a synovial membrane, but also of other structures composing and surrounding a joint. It may follow traumatic synovitis; it may be due to pus-organisms, to tubercle bacilli, to infectious diseases (gonorrhea and typhoid fever), to rheumatism, to gout, to syphilis, and to lesions of the spinal cord. Arthritis may be either acute or chronic.

**Poncet's Tuberculous Arthritis** (*Tuberculous Articular Rheumatism*, see page 250).—It was pointed out by Grocco in the early 80's that tuberculous patients may develop joint disease without tubercles, cold abscesses, or destruction of the joint. Poncet, of Lyons, has written extensively on this subject. He maintains that joint inflammation is often the first evidence of extra-articular tuberculosis or of a distant latent lesion of tuberculosis. The joint inflammation is due to toxins, the joint fluid does not contain bacilli, and tubercles do not develop in the joint structures.

Such a joint may eventually develop into a true tuberculous joint with bacilli and tubercles. In many cases the joint recovers, with some stiffness or perhaps even ankylosis. The process may be acute, subacute, or chronic. One joint only may suffer. Several or most of the joints may be simultaneously involved. It is most apt to arise after an operation on tuberculous glands or bone. Fever exists in Poncet's arthritis. Matas ("Southern Med. Jour.," Oct., 1911) thus describes tuberculous rheumatism:

"It may be a flitting arthralgia, a mere soreness, a dropsical synovitis, or it may persist as a hypertrophic or atrophic arthritis. It may present itself as a progressive polyarthritis deformans or as a dry senile arthritis (morbus coxarius senilis). The acute, as well as the chronic, form of this type of rheu-

[1] H. A. Wilson, in "Annals of Surgery," Feb., 1899.

matism may end in ankylosis. Many cases of spinal rigidity, spondylosis, scoliosis, painful flat-foot, genu valgum, coxa vara, and even certain cases of dry otitis media with ankylosis of the ossicles of the ear, and, in fact, all the osteo-articular affections of childhood and adolescence associated with defective osteogenesis, may owe their origin to the disturbing influence of a latent or obscure tuberculous toxemia.

"The most benign of the tuberculous rheumatic affections is a simple arthralgia—a sore or painful joint. There is pain without any objective signs, and yet this is often the precursor of a manifest visceral tuberculosis. The acute and subacute forms of the disease are usually confused at the bedside with the manifestation of ordinary acute rheumatism. Nevertheless, the clinical picture of acute tuberculous rheumatism is distinctly graver; there are fever, rapid pulse, profuse sweats, and frequent attacks of dyspnea. This is brought about by associate general infections involving the serous cavities, the pleura, peritoneum, pericardium, and the meninges. But in the majority the evolution of the disease is slow and more benign. The joint symptoms appear in successive stages separated by intervals of rest more or less long, and are distributed over various joints, ending ultimately in ankylosis. The disease is more often chronic, and many patients either recover altogether or remain permanently crippled or deformed; others die of marasmus or complications from associate visceral disease. Therapeutically, this form of rheumatism is not relieved by any of the usual antirheumatic remedies, and is improved or cured solely by treatment which aims at the cure of the tuberculous infection."

Fig. 382.—Tuberculous arthritis of wrist.

**Tuberculous Arthritis** (*White Swelling*, so christened by Richard Wiseman; *Strumous Joint; Pulpy Degeneration*).—*Pathology and Symptoms.*—The predisposing causes of tuberculous arthritis may be strains, blows, twists, or cold (see page 223). The real cause is the tubercle bacillus. A single joint is attacked. Other joints may subsequently become involved so that several suffer simultaneously, but it is rare that the process is active in more than one joint at the same time. During the course of tuberculous disease of a joint (except of the shoulder-joint) phthisis is not common, although it may develop after the joint gets well. The same is true of tuberculous glands. During the existence of phthisis or tuberculous glands tuberculous arthritis does not frequently arise.

The primary infection with tubercle bacilli is usually in the bone, though it may be in the synovial membrane, the joint-capsule, or the structures about the joint. The frequency of the bony origin of tuberculous arthritis is shown by John B. Murphy's statement that in 128 cases of tuberculosis of the knee it was demonstrated in all but 2 that the condition originated in the bone ("Jour. Am. Med. Assoc.," May 20–27, June 3, 1905). If the primary infective focus is in the bone, a portion of the cartilage undergoes destruction and the joint is opened, or a sinus forms and perforates the synovial membrane. When tuberculous inflammation attacks the synovial membrane granulation tissue is formed, and the capsule and periarticular structures soon become involved in the process; the parts thicken and soften from caseation, and they may be covered with tubercles, though but little fluid is usually effused into the joint. Some few cases present large joint effusions, but in most cases fluctuation is absent. Capsular thickening may or may not be manifest. Soon after tuberculous arthritis begins the joint becomes rigid, irritation having induced muscular spasm. This reflex rigidity fixes the joint more or less completely, and atrophy of the rigid muscles soon begins. There is usually some pain in tuberculous arthritis; it is seldom marked except on motion or when the epiphysis is involved, and it may be referred to a distant part. For instance, in hip-joint disease the pain is often referred to the inner side of the knee, and in Pott's disease of the spine the pain may be referred to the abdomen. A cardinal sympton of tuberculous disease of a joint is localized tenderness. In other types of infection the tenderness is widespread. Attempts at motion demonstrate the limitation of movement due to muscular rigidity and also produce pain. A child that suffers from a tuberculous joint is apt to be restless in sleep, moaning and tossing, and to wake at times crying out in terror (*night-cries* and *night-terrors*). In the ordinary form of tuberculous arthritis there occurs what is known as *gelatiniform degeneration;* the granulation tissue is formed in large amount as fungous growths; the structures are markedly edematous and softened; the relaxed ligaments yield under pressure; the natural contour of the joint is lost and it becomes spindle shaped; all the structures, articular and periarticular, are glued into one mass; the skin about the joint is white, thick, and adherent, and in it one or more large veins are seen; fluctuation or pseudofluctuation is noted when caseation has occurred; pain is not often severe, but it can usually be elicited by certain motions or by firm pressure, but the pain will always be severe when the epiphysis is involved; the temperature of the part is seldom elevated; deformity results from destruction of bone, cartilage, and ligament, from muscular spasms, and from the habitual assumption of certain attitudes to secure relief from pain. There is soon impairment of joint motions. When the products of a tuberculous arthritis caseate the thick liquid seeks exit by forming sinuses, and from them caseous pus flows. If a sinus becomes infected with pyogenic cocci, and the joint itself becomes their prey, acute suppuration arises in the joint, and constitutional involvement is pronounced and perilous to life.

In *pannous synovitis* a large effusion is formed, there is but little granulation tissue, though the tubercles are present in large numbers, and the ligaments and structures about the joint are slightly or not at all implicated.

*Diagnosis and Prognosis.—Tuberculous chronic synovitis* produces great swelling and distinct thickening of the capsule with obliteration of the outlines of the joint, no severe pain, and no tendency to early subluxation. Tuberculous arthritis rarely causes distinct fluctuation, does not thicken the capsule, causes reflex muscular spasm, rigidity of the joint, muscular atrophy, severe pain on movement, and eventually subluxation (Shaffer). In syphilitic arthritis there is usually some fluctuation, distinct enlargement of the joint, limitation of motion, no reflex spasm, trivial atrophy, but distinct pain on motion (James

K. Young, "Therapeutic Gazette," June 15, 1902). Acute rheumatism attacks more than one joint, is very rare in children under five, and produces high fever. The *x*-rays aid in the diagnosis of tuberculous arthritis and enable us to tell the extent of bone involvement.

The diagnosis of a tuberculous joint is often difficult, and sometimes impossible, and the prognosis is always grave. In only a very few cases, even when recognized early, is a cure obtained without some impairment of joint function. The best that can usually be accomplished is a cure with more or less ankylosis, fibrous or bony; and often ankylosis is complete. Long after the disease is apparently cured, it may break forth anew. Tuberculous lesions may arise in a distant organ or general tuberculosis may occur. Caseation is apt to produce severe constitutional disorder. Infection by pus organisms gives rise to grave danger of septicemia. Death is not unusual from exhaustion, from septicemia, from disseminated tuberculosis, from tuberculosis of an important organ, or from amyloid disease.

*Treatment.*—Conservative treatment is especially successful in children. According to Hoffa, in 75 per cent. of cases in children non-operative treatment will produce cure ("Die Bekampfung der Knochen- u. Gelenktuberculose in Kindesalter Tuberculosis," iv, 1, 1905). The conservative treatment consists in open-air life, if possible in a sanatorium, the following of the plans outlined under Tuberculosis, immobilization and extension of the joint, and injections of iodoform emulsion or formalin-glycerin (2 per cent.). Even when tuberculous pus forms, the same treatment may be followed unless there is violent pain or elevated temperature which does not quickly abate, in which case operation must be performed. Cases treated early by conservative methods may get well with a movable joint, but in most cases there is a stiff joint when the disease is arrested. Constitutionally, the treatment is directed against the tuberculous diathesis. The patient should be placed under good hygienic conditions. A change of climate is often of the greatest importance. Many cases do well at the seaside; others require high altitudes, and all should live in the open air. The value of sunlight is set forth on page 231. The Finsen light, too, is of service. Treatment by tuberculin is considered on page 235. Locally, rest is of the first importance, and fixation is maintained for many weeks. Rest is best secured by immobilization and traction, and traction is applied or maintained by splints, by Plaster-of-Paris bandages, or by extension appliances. The hot-air apparatus may be of some benefit. If it is employed, it should be used daily, the limb being immobilized during the remainder of the twenty-four hours. Fixation must be maintained as long as pain exists or muscular spasm is present. Fixation must be abandoned gradually, and mobility is to be slowly regained. During restoration to mobility, if pain arises, the part must be immobilized temporarily. Movements should never be violent, prolonged, or repeated at too close intervals. Forcible breaking up of an ankylosis is never advisable in the knee, ankle, or hip; it is seldom advisable in the wrist, elbow, or shoulder. Such an attempt may be followed by a fresh outbreak of the disease. Osteotomy or resection will more safely correct a faulty and disabling deformity. In a tuberculous joint injection of formalin-glycerin, as advised by Murphy, is a valuable procedure. Several injections or a number may be made. The joint is aspirated and the mixture introduced. About 4 drams are used in the knee, about 2 drams in the ankle. After injection extension is applied. The mixture used must be at least twenty-four hours old. In tuberculous joints intra-articular injections of iodoform are often of the greatest value. This drug strongly stimulates the formation of fibrous tissue. If sufficient fibrous tissue is formed the tuberculous foci will be firmly encapsuled and the case will be cured. Iodoform is particularly called for when no bone disease is shown by the *x*-ray or discovered

by incision. The joint is incised, adhesions are gently broken up, the capsule is sutured except for a small space, the nozzle of a syringe is inserted and a mattress suture draws the capsule tightly about the nozzle (Brackett's plan). The emulsion is injected under tension and then the capsule is closed tightly. In a large joint a 4 per cent. emulsion is used; in a small joint a 10 per cent. emulsion. The part must not bear weight for six months. Bier's plan of inducing passive hyperemia is often of great service (see pages 112–115). Aspiration or incision can be used for fluid accumulations. Caseous masses are often let alone, or an aspirator is used and the joint drained, washed out with saline solution, and injected with an emulsion of iodoform and glycerin (10 per cent.). From 1 to 2 drams are injected into the joint of a child, from 2 to 5 drams into the joint of an adult. Even surface lesions are not curetted. Bier's treatment should be associated with immobilization and systemic treatment. It is more serviceable in tuberculosis of the small joints than in disease of the large articulations. There are certain contra-indications to Bier's treatment, viz.: serious pulmonary involvement, extensive amyloid degeneration, and the existence of such an unfavorable position of the parts that cure by ankylosis would mean a less useful limb than cure by resection (Bier at Internat. Surg. Congress of 1905). One advantage of this treament is that we can employ active and passive motion early, except, according to Bier, when the foot or knee is diseased. Even in very serious cases cure may be obtained without any limitation of activity, and as the patient can get about it is not necessary to restrain him long in a hospital. Personally I believe that fixation should be the basis of treatment in most cases, and that passive hyperemia, compression, counterirritation, and intra-articular injections should be additions to fixation. Injections of balsam of Peru or of iodoform emulsion about the joint once a week are efficient in some prolonged cases, but are not to be used early. It is not wise to attempt to correct faulty position until the focus is well.

Fistulæ are frequently treated by the method of Beck, of Chicago, that is, by the injection of a paste containing bismuth. In early cases a bismuth-vaselin paste is used, in late cases a bismuth-wax-paraffin paste.

Beck described his method in the "Illinois Med. Jour.," April, 1908. The vaselin paste is composed of 30 parts of subnitrate of bismuth and 60 parts of vaselin, mixed and well stirred while boiling. The paste for later cases is composed of 30 parts of subnitrate of bismuth, 5 parts of white wax, 5 parts of soft paraffin, and 60 parts of vaselin, mixed while boiling. One per cent. formalin is often added to these pastes. The paste is injected cold before an *x*-ray picture is taken, and the picture shows all the ramifications of the fistula. The paste is left in for treatment. If a sequestrum exists it should be removed before the paste is injected. It is not necessary, but is advisable, to dry the fistula before injecting.

The paste is sterilized before using. It is sucked up into the syringe while still liquid and is cooled to the requisite temperature and hardened to the proper consistency by running cold water over the syringe. It is injected very slowly and the injection is continued until a sense of pressure annoys the patient. Then the syringe is laid aside, a bit of gauze is held for a time over the outlet of the fistula to keep the paste from running out, and an ice-bag is put over the region to quickly harden the injected material. There is no pain from such an injection. Beck employs the first paste until pus disappears and then uses the second.

The value of this paste is that it distends and fills the abscess-cavity and sinus, and affords a trestle or frame for granulations to grow upon. Considerable of the paste may run out of the fistula during the first twenty-four hours. In shallow sinuses it all runs out. In deep and tortuous fistulæ much of it remains for weeks and is slowly absorbed. In empyema and bone-cavities

it is slowly absorbed. Beck holds that the paste is bactericidal, astringent, and non-toxic. Cases of pigmentation of the lips, gums and cheeks, and cases of ulceration in the mouth from absorption of bismuth have been recorded, and at least 8 deaths are on record (Reich, in "Beiträge Zur klinischen Chirurgie," Nov., 1909). Beck limits the first dose to 100 gm., but increases the amount later. Because of possible danger of poisoning Blanchard uses a paste of white wax and vaselin without bismuth. Not over 4 oz. of bismuth paste should be injected. As Bell points out, if a large quantity is injected an opening must be left. This precaution may prevent poisoning. Some think the poisoning is due to bismuth, others believe it is due to arsenic held as an impurity. David and Kauffman ("Illinois Med. Jour.," Oct., 1909) point out that there may be acute cases of poisoning which are liable to be fatal, and chronic cases which tend to recovery. In an acute case of bismuth-poisoning there is pigmentation of the gums, ulcerative stomatitis, dyspnea, delirium, and albuminuria. David and Kauffman regard pigmentation of the gums, lips, or cheeks as an indication of toxic action and a sign to discontinue the drug. The use of bismuth paste is undoubtedly a valuable method. I have seen sinuses heal under it after they had resisted various other plans of treatment. It may be used in sinuses, cold abscesses, empyemata, tuberculous joints, and other conditions.

If these means fail, if the patient gets worse, if there is persistent fever or violent pain, if sequestra exist, if there is mixed infection, or if the condition of the sufferer renders dangerous the prolonged conservative course, operate, removing the entire diseased area by erasion, by excision, or possibly by amputation. If the *x*-ray picture shows extensive sequestrum formation, operation is indicated. If amyloid degeneration exists, conservative treatment is contra-indicated and so is resection. Amputation must be done. Always remember that an incomplete operation or a partial removal, unless it consists of simple drainage, is worse than no operation, as it opens the portals to systemic infection, and may be responsible for the development of general tuberculosis, septicemia, or pyemia. Simple drainage, as previously stated, is seldom advisable. Garré is of the opinion that the hip, wrist, and shoulder do best by conservative treatment; the knee, elbow, and ankle, by operative treatment (John W. Churchman, in "Am. Medicine," April, 1906).

**Tuberculosis of Special Joints; Tuberculosis of the Vertebræ** (see page 844).—**Tuberculosis of the Sacro-iliac Joint** (*Sacro-iliac Disease*).—This is an uncommon affection, and is especially rare before the age of fifteen. The disease may begin in the joint, may arise in adjacent bones, or may result from a cold abscess burrowing into the joint. In some cases it is associated with extensive disease of the pelvic bones. The disease, if undetected, may lead to dissemination of tubercle, to abscess, or even to death.

*Symptoms* are often obscure. The disease is frequently confounded with vertebral caries, hip-joint disease, or sciatica. The patient limps on walking, but can stand on either leg; there is pain in the sacro-iliac joint, about the hip, and down the thigh; tenderness is manifest on pressure over the joint and on pushing the ilia together; there is fulness over the sacro-iliac joint, but the hip is not flexed unless iliac abscess exists.[1]

*Treatment.*—Rest in bed for months, using also a felt case for the pelvis. Counterirritation by blisters and the actual cautery. In some cases injection of formalin-glycerin or of iodoform; in others, incision and curetting. I have operated on 9 cases, with 1 death. In 1 case in the Jefferson Medical College Hospital the abscess was pointing in both the back and groin. Both areas were incised, the diseased bone was removed, and the boy ultimately recovered. In another case the abscess pointed in the groin. The treatment was as previously set forth, and the patient, a woman, recovered. The best way to reach

[1] See A. G. Miller, "Edinburgh Med. Jour.," May, 1895.

the joint is to chisel or bore through the ilium above the great sacrosciatic foramen.

**Tuberculosis of the Hip-joint** (*Hip Disease; Morbus Coxarius; Morbus Coxæ; Hip-joint Disease*).—The primary lesion may be in the synovial membrane, but it is more often in the bone. It may begin in the acetabulum; it may begin in the femur. In 95 per cent. of cases it begins in the head of the femur. If it begins in the femur, it usually arises on "the distal side of the epiphyseal cartilage" (Senn). Sometimes primary tuberculosis arises in the trochanter major, and never involves the joint. When the synovial membrane becomes involved at any point, spread throughout the joint is rapid. In many cases the articular cartilages are attacked, and in some cases the epiphyseal cartilage is destroyed. It is commonest in children, but it may arise in adults and even occasionally in those of advanced years; 62 per cent. of cases arise in children under ten years of age and 80 per cent. of cases occur before the twentieth year (Bryant). Traumatism and cold may be predisposing causes. The disease strongly tends to caseation and the formation of sequestra.

*Symptoms.*—It has been usual to divide the disease into three stages: (1) the stage of microbic deposition and multiplication, the products of the bacilli causing irritation and new growth; (2) the stage of progression, with formation of masses of granulation tissue and effusion into the joint, and (3) the stage of caseation, with destruction of the joint and often of the structures about it. Bradford and Lovett[1] protest against this. They say: "It has been customary to divide hip-disease into stages, and to ascribe to these stages certain definite symptoms. Neither from a clinical nor a pathological point of view is it desirable to attempt such a division." As H. Augustus Wilson says: "Tuberculous bone and joint disease should be considered as the primary invasion or incipiency, and all other symptoms should be regarded as results and not as an integral and necessary part of the trouble."

The symptoms of incipient coxalgia are slight and may be overlooked entirely. In a child there are night-terrors; on getting about in the morning the child shows no lameness, but a limp develops during the day, and the little one soon grows tired while playing and lies down to rest. There is a slight limp; some muscle spasm may be noted, and pain may be complained of at night in the hip, in the front of the thigh, or at the inside of the knee. Tapping the sole of the foot, the thigh and leg being extended, may develop pain, just as it will develop pain in any inflammatory involvement of the joint, but the employment of this method is objectionable. It may injure a joint already damaged by the tuberculous process, and it gives no information which cannot be obtained by a safer mode of investigation. After all, pain on tapping the sole of the foot means only what muscular rigidity means, and muscular rigidity is always present and is easily demonstrable by careful manipulation. The diagnosis of incipient coxalgia is more or less problematical.

As the disease progresses more positive symptoms are observed. The limp grows worse; the hip is broadened by an effusion into the joint, and fluctuation may possibly be detected; the thigh muscles atrophy; the extremity is pushed forward, abducted, and everted. The position is described as flexion, abduction, and outward rotation (Fig. 383). This position may not be obvious; in fact, the limb may be extended by the side of the companion extremity. When it is, there is a forward curve of the lumbar spine (lordosis) and a lateral curve of the lumbar spine, which raises the pelvis on the sound side and depresses it on the diseased side. These lumbar curves serve to bring the femur toward the middle line, give the extremity the appearance of being lengthened, enable the sufferer to walk (Fig. 383), and cause him to

[1] "Orthopedic Surgery."

rest his weight on the sound limb. Apparent lengthening means abduction. In some few cases adduction exists rather than abduction. The abduction, which is usual, releases tension of the fascia lata, and thus abolishes pressure upon the joint through lessening of pressure upon the great trochanter, and also relaxes the outer portion of the Y-ligament and the ligamentum teres. The flexion relaxes the anterior portion of the capsule and the psoas muscle and prevents pressure of its tendon upon the front of the joint. Outward rotation relaxes the inner portion of the Y-ligament and the posterior portion of the capsule. Pain exists, often sudden or starting, and is located in the joint, on the front of the thigh, and to the inner side of the knee in the course of the obturator nerve; the pain is aggravated at night, and full extension and complete abduction are not possible. The gluteal muscles waste, and the gluteal crease is on a lower level than is that of the sound side. The gluteal crease may be nearly or quite effaced because of hypertrophy of the subcutaneous layer (Alexandroff), or from slight flexion of the leg (McClellan). As the disease progresses adductor spasm causes adduction, and the limb is flexed, adducted, and apparently shortened. This apparent shortening is accomplished by a lateral curvature of the spine, which keeps the limb from crossing its fellow and being useless. It does so by raising the hip of the

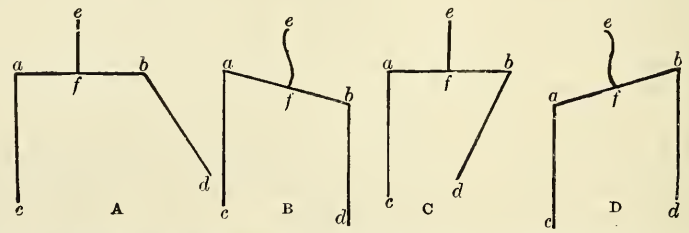



Fig. 383.—Positions in hip-joint disease. A: *e–f*, lumbar spine; *b–d*, limb fixed in flexion and abduction, useless for walking. B: *e–f*, lumbar spine. Patient corrects the condition in Figure A by curving the lumbar spine forward and rotating the pelvis on its transverse axis, thus making the femur point downward. The lumbar spine is curved laterally, the pelvis ascending on the sound side and descending on the affected side (apparent lengthening). C: *b–d*, limb fixed in flexion and adduction. D: *e–f*, curve of lumbar spine to correct condition in Figure C (apparent shortening). (After the plan of Howard Marsh and Treves.)

diseased side and drawing the femur outward (Fig. 383). This causes apparent shortening (Howard Marsh, in Treves's "Manual of Surgery"). The above symptoms arise chiefly from unconscious efforts to obtain ease, from joint-effusion, reflex irritation, and involuntary or spasmodic muscular contractions. There is an appearance of lengthening or shortening, but it is only apparent, not real. The real position is shown on Plate 8, Fig. 4. The fluid effusion may be absorbed or may find its way externally by means of sinuses. The latter condition is known as *abscess of the hip*. The absorption of the exudate or the rupture of the capsule permits the contracting muscles to bring the head of the femur into firm contact with the acetabulum or its brim; the bones are worn away and destroyed, real shortening results, there is adduction, and flexion is increased as shortening becomes more marked.

In advanced cases of coxalgia the head of the femur passes upward and outward upon the rim of the acetabulum, the thigh is flexed and fixed, and attempts at extension when the patient is recumbent cause the pelvis to tilt forward and occasion a marked lumbar curve (lordosis) (Pl. 8, Fig. 2), which is due to the pelvis moving with the femur as if ankylosed, and which disappears on flexion. In this condition adduction occurs because of the ascent and movement outward of the head of the bone. Shortening is marked. After a hip abscess finds an external outlet pyogenic infection is very apt to take place

and suppuration arises, which is followed by that state which is designated as "hectic." If a cure follows advanced coxalgia, partial or complete ankylosis takes place; if death ensues, it may be due to septicemia, tuberculosis of the viscera, exhaustion, or amyloid degeneration.

*Diagnosis* is very easy in well-established cases of hip-disease, but very difficult when the disease is incipient. Always make a systematic and thorough examination. Undress the patient and place him recumbent with his legs extended upon a table or a hard mattress. Note if the heels are level and if the iliac spines are on the same level (a depressed spine on the affected side means abducted extremity, the degree of which is determined by carrying the limb out until the spines are horizontal; elevation of the iliac spine on the affected side means adduction, the amount of which is determined by adducting the limb until the spines are horizontal, Fig. 383). The amount of flexion is ascertained by lifting the knee until the curved spine has become straight. Try all the movements belonging to the joint, to detect any limitations; observe if bringing down the knee produces lordosis; look for swelling and for muscular wasting; feel if the head of the bone is enlarged; determine if motion produces pain or if pressure develops tenderness; and always carefully elicit the history of the attack, of the person, and of the family.

Hip disease may be confounded with spinal caries in which a psoas or a lumbar abscess has formed, with sacro-iliac disease, with infantile paralysis, with congenital dislocation of hip, with lordosis from rickets, with gluteal abscess, and with bursitis of the gluteal bursæ. In hip disease there is always some lameness; pain may be severe, may be trivial, or may be absent entirely, and may be in the hip or be referred to the front of the thigh or the inner side of the knee. Always remember that the pain is not characteristic, and that pain in the same localities may arise from aneurysm of the femoral or iliac arteries, from abscess in Scarpa's triangle, from caries of the lumbar vertebræ, from sacro-iliac disease, and from cancer of the rectum. Altered position of the limb, limitation of movement in the hip-joint, muscular wasting, and swelling soon arise in hip-joint disease.

In disease of the sacro-iliac joint examination shows that the movements of the hip-joint are unlimited and produce no pain, and that pain is developed by pressure over the sacro-iliac articulation and by pressing the ilia together. In infantile paralysis there is no pain, but there is paralysis with great muscular atrophy, which comes on with considerable rapidity. In spinal caries with psoas abscess the evidences of disease of the vertebræ are clear and a collection of fluid is located in the groin external to the femoral vessels. The tuberculous pus of hip abscess generally gathers under the tensor vaginæ femoris muscle, but it may reach Scarpa's triangle by passing through the cotyloid notch or through the bursa under the psoas muscle; it may even appear under the glutei. Matter from a caseating acetabulum may reach the interior of the pelvis and appear above Poupart's ligament.

In gluteal bursitis the symptoms last for many months, and do not remit as the symptoms of early hip disease are apt to do. The pain is but moderate, and is aggravated by exercise, but passes away on going to bed, and is felt back of the hip and back of the knee. There is a certain amount of limitation of motion and there is a positive limp, which arises early. In marked cases fluctuation can be detected in the upper gluteal region.[1]

*Prognosis.*—If the case of hip disease is seen early the chances of cure are excellent in children, in whom the disease may be arrested at any stage. The longer the duration of the disease and the older the subject, the more unfavorable is the prognosis. Many months will be required to elapse before a cure

[1] See E. G. Brackett's important paper on "Gluteal Bursitis" in the "Transactions of the American Orthopedic Association," vol. x.

can be effected, and advanced cases only get well by means of ankylosis with shortening and deformity. Hip disease may recur years after apparent cure, and a person who has or has had hip disease runs a chance of developing visceral tuberculosis.

*Complications.*—The complications that may accompany hip disease are the following: *Abscess*, as above noted. *Tuberculous meningitis*, or the condition known as "acute hydrocephalus" or "water on the brain," may arise during the progress of the case or after apparent cure, and is apt to ensue upon incomplete operations. It is almost inevitably fatal. *Phthisis pulmonalis* is a rare complication, but a not uncommon sequence, perhaps arising, sooner or later, after the hip disease has been cured. *Amyloid, lardaceous*, or *waxy degeneration of viscera* (see page 239) follows upon profuse and long-continued suppuration and is apt to arise in the liver, spleen, kidneys, or intestinal mucous membrane. Tuberculosis is not the only cause of amyloid degeneration, syphilis being responsible for at least 30 per cent. of all cases. In amyloid disease of the liver the organ is much enlarged, smooth, painless, and of increased consistency; there is no jaundice, the spleen is apt to be enlarged, and albuminuria is the rule. In amyloid kidney large amounts of pale urine of low specific gravity are voided; albumin is usually present in large amount, but may be absent; globulin may often be found, as may also hyaline, fatty, or granular casts; the patient is anemic and dropsy usually exists. Test the hyaline casts with iodin for amyloid material. Amyloid changes are usually slow in onset, but they may be rapid; they are commoner in men than in women, and are most frequently encountered in individuals between the ages of ten and thirty. Slight amyloid change may be recovered from, but an extensive degeneration brings about a fatal result.

Fig. 384.—Thomas's posterior splint.

*Treatment.*—In most of these cases conservative treatment is advisable. Antituberculous treatment is used in all cases. In incipient hip disease the treatment usually advocated is rest. The patient is placed upon a solid mattress and extension is applied. In children under ten years of age a weight of from 3 to 5 pounds is used; in individuals between ten and twenty a weight of from 5 to 8 pounds is used. A long splint is often applied to the sound side to keep the patient recumbent and horizontal. A cradle is employed to hold up the bed-clothing. The extension is applied in the long axis of the limb, the extremity being placed in the line of the deformity due to disease and being properly supported. In lordosis from thigh-flexion the limb is raised until the iliac spine is straight (Pl. 8, Fig. 5). If the spine is depressed on the affected side, the limb is abducted (Pl. 8, Fig. 6); if the spine is elevated, the limb is adducted until the spines are horizontal (Pl. 8, Fig. 7). The object of extension is to overcome muscular spasm and so put the part in a condition of physiological rest. Muscular spasm is a great factor in destroying structures. Spasm presses the parts together, and as a result of pressure plus bacterial action destruction occurs. The extension and traction tire out the muscles and cause spasm to cease. Extension will remove flexion in two weeks in a recent case and in the course of some months in an older case. As flexion is relieved the pillows are removed and the leg lowered, but extension is maintained in the long axis of the thigh. Abduction and adduction cannot be removed by simple extension in the axis of the limb.

Abduction demands no special treatment. In a movable joint it will disappear, and in an ankylosed joint it is an advantage, compensating by apparent lengthening for the shortening due to bone-absorption or to stunted growth of the limb. Adduction requires an addition of several pounds to the extension weight, the use of a long splint on the sound limb, and the drawing up of the sound side by a rope and pulley toward the head of the bed. The weight used to pull the sound side toward the head of the bed is equal to that used to pull the diseased side to the foot of the bed. This expedient is used for a month or six weeks. In old cases, in which the weight will not bring about extension, the patient is anesthetized, the limb is gently straightened a very little, and the weight is reapplied.

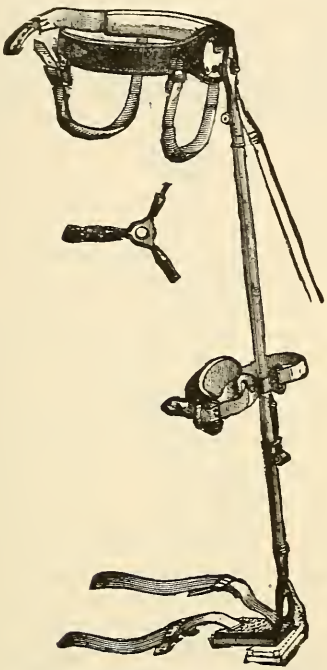


Fig. 385.—Sayre's long splint.

Fig. 386.—Wyeth's combination method.

Extension in a mild case must be continued for three months after the symptoms have disappeared, and in a severe case the period must be six months. The weight is gradually taken off; if symptoms recur, the weight is reapplied; if they do not recur, apply a traction splint or a plaster dressing, put a high-heeled boot on the sound limb, and send the patient out on crutches. In young children extension can be made while the child is in a wheeled carriage, thus enabling the patient to go out in the fresh air and sunlight. The general treatment is tonic and restorative. The joint is so deeply placed that external applications are useless. In the treatment of hip disease Thomas's splint (Fig. 384) is used by many, and it may be combined with weight extension; or Sayre's splint (Fig. 385) may be employed. Wyeth's apparatus (Fig. 386) is a favorite with many American surgeons.

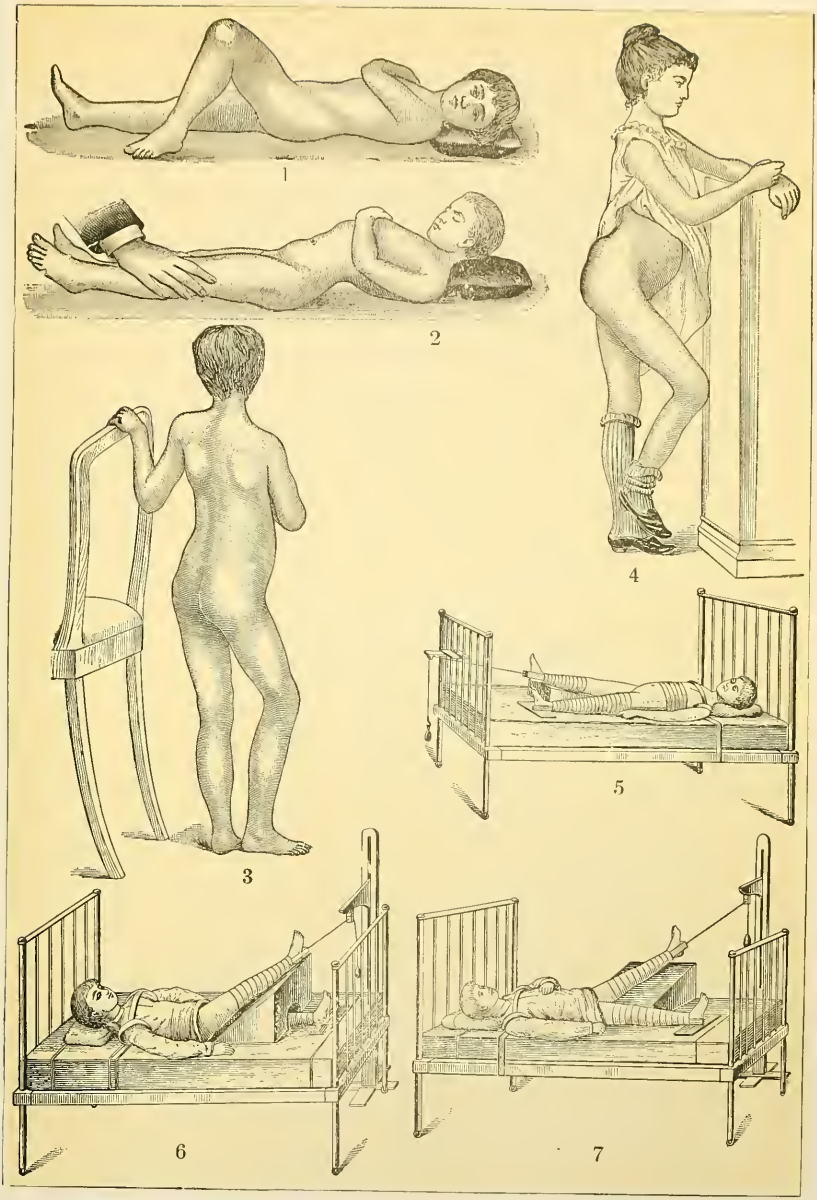
If the limb is in good position, or has been brought into good position, either by weight extension or straightening under ether, plaster-of-Paris is a useful dressing. It is applied from the toes up, and includes the entire extremity and also the pelvis. A patient wearing plaster may get about on crutches when the sole of the foot of the sound extremity is raised by the wearing of a thick-soled shoe.

*Treatment by Weight-bearing and Fixation by Hip Spica of Plaster of Paris (Lorenz's Method).*—This plan is based upon the principle that ankylosis is to be secured in every instance. To keep the patient in bed is to keep him from the open air and sunlight, which are so necessary in tuberculosis. Such confinement favors muscular atrophy, leads to anemia, and lessens vital resistance. The hip is placed in 20 degrees of flexion, 20 degrees of abduction, and 5 degrees of external rotation, and is fixed by a short plaster spica of the hip. The patient walks and so antagonizes muscular spasm. The joint surfaces bear weight, which leads to a useful increase of blood supply (curative hyperemia), but do not grind upon each other, hence, the spread of the disease is not favored. That muscles increase in size is shown by the fact that the cast gets tight and must be changed from time to time. The open-air life and exercise are of the greatest benefit to the patient and a gain in weight is the rule. Cases in a debilitated condition or those with discharging sinuses must not be treated by this plan "without the temporary use of crutches" (H. A. Wilson, in "Southern Med. Jour.," Dec., 1908). The plan is more suitable for incipient than for advanced cases. The average duration of treatment is claimed to be about ten months (H. A. Wilson, Ibid.). It is true that the method affords easier access to the open air than does the bed treatment, but why should every patient be doomed to ankylosis when we know that in many cases joint function may be retained by treatment looking to that end?

*Intra-articular Injections and Operation.*—If in spite of treatment the condition does not improve or if it becomes worse, use intra-articular injections of formalin-glycerin or iodoform. Always try these injections before doing a resection unless the *x*-rays show a large sequestrum. Sometimes they succeed, and if they do, resection is unnecessary. Asepticize the surface, carry a small aspirating needle into the joint, irrigate the joint with salt solution, and inject a sterile emulsion of iodoform and glycerin (see page 622). In one week, if reaction has ceased, repeat the injection. In another week repeat it again. It may be necessary to give from ten to twenty injections. The proper spot for puncture is thus determined: Draw a line from a point ½ inch outside of the middle of Poupart's ligament to the outer edge of the great trochanter. Puncture at the middle of the outer half of this line (De Vos). I have not attempted to remove the disease surgically early in any case and greatly doubt the wisdom of doing so. Huntington and some other surgeons advocate early operation in children instead of simply fixation, extension, and rest. Huntington ("Amer. Jour. Med. Sciences," July, 1905) recalls that when the lesion is in the head of the femur it tends to perforate into the joint, and he advises trephining at the lower border and outer aspect of the great trochanter and tunnelling the neck and head of the femur with a curet. Bradford objects to this method in most cases on the ground that unless the disease is localized and the cavity is well walled off and unless injury to the localizing barrier is avoided, the operation may be responsible for dissemination of the bacteria.

If an abscess forms, incise it with the most thorough antiseptic care, let the fluid drain away, irrigate the cavity with salt solution, remove any sequestra, inject with iodoform emulsion, sew up without drainage, and dress antiseptically. In some cases the sequestrum is extra-articular. In many cases no sequestrum is found. If this method fails, drainage must be employed. The old plan of not operating until rupture was seen to be inevitable would be wrong to-day. To open early and antiseptically often means rapid healing, the prevention of burrowing, a lessened danger of visceral infection, and an earlier cure. In contrast to what happens when a very large cold abscess is opened, hectic will rarely arise when a tuberculous joint is opened and drained with aseptic care.

Excision of the hip is to be performed when there is a large sequestrum or severe fistulæ (Garré, "Deutsch. med. Woch.," 1905, Nos. 47 and 48);




1, 2. Effects on the Lumbar Spine of Flexing and Extending the Diseased Leg in Hip Disease (Albert). 3, 4. Positions in Coxalg a (Albert). 5. Extension in Hip Disease (Treves). 6. Extension of the Limb in a Flexed and Adducted Position (Treves). 7. Extension of the Limb in a Flexed and Abducted Joint (Treves).

when the head of the femur is detached and lies loose in the joint; when profuse suppuration continues for a long time, and other methods fail to arrest it; when amyloid disease is threatening; or when very faulty position is inevitable without operation. Excision is an operation of considerable danger, and the older the person, the greater the danger. Schede advocates arthrectomy in some cases as a substitute for resection. Senn tells us that opinion as to resection has greatly changed of late, and it is now taught that the operation is advisable in all cases where fixation, extension, intra-articular and parenchymatous injections have failed to arrest the disease (Senn, on "Tuberculosis of Bones and Joints"). Resection of the hip does not give a very satisfactory functional result. When there is extensive disease of the femur, when excision has been tried and has failed, when the patient has not the recuperative power to withstand the long siege of illness following excision, or when there is amyloid disease, amputate.[1] Amputation of the hip-joint for tuberculous disease is a very successful procedure.

**Knee-joint Disease** (*White Swelling*).—After the hip, the knee is, of all joints, the commonest site for tuberculous disease. Knee-joint disease can begin as a synovitis, but oftener begins as tuberculous inflammation of the femoral or the tibial epiphysis. Tuberculous disease rarely attacks the bone on the diaphyseal side of the epiphyseal line; a single focus only exists, as a rule, and a sequestrum is seldom formed. In very rare instances the patella or the semilunar cartilage is primarily attacked. It may begin at any age, but is most common in children and young adults. If an acute synovitis ushers in the case, there may be a large effusion into the knee-joint and partial flexion, but swelling is usually slight in knee-joint disease. Pulpy degeneration of the synovial membrane occurs; the joint enlarges; the ligaments soften; the skin becomes edematous, and muscular spasm exists. The leg becomes flexed; the tibia displaced backward and outward; the foot is everted; and lameness arises, due chiefly to deformity. Muscle atrophy is marked above and below the joint. Pain may be absent, is often slight, and is rarely severe. Local tenderness is very common. When the disease begins in the bone or an epiphysis there are pain, tenderness, lameness, swelling, inability to extend the limb completely, sudden spasmodic muscular contractions, and final involvement of the joint. When an abscess forms, it may destroy the joint very rapidly or it may break externally.

*Treatment.*—In treating knee-joint disease conservative treatment is usually tried, but often fails. A plan of doubtful value is to make a mixture of guaiacol and olive oil (1 : 4). Once a day the surface of the knee is exposed by removing dressings, is painted with this mixture, and the painted surface is covered with cotton-wool. Rest is of the first importance, and may be secured by the application of splints (Figs. 387, 388), the use of extension (Fig. 389), or the employment of a plaster-of-Paris bandage. In any case the patient must be kept in bed for a few weeks; he may then be permitted to go out upon crutches, wearing a high-heeled shoe upon the foot of the sound limb. In cases in which treatment is begun early the

Fig. 387.—Sayre's knee splint applied.

Fig. 388.—Hutchinson's knee-joint splint.

[1] See the admirable article by Howard Marsh in Treves's "Manual of Surgery."

disease may often be arrested in from eight to twelve months. If the symptoms do not abate after a number of weeks, or if the condition grows worse and caseation occurs, aspirate, irrigate, and inject iodoform emulsion or formalin-glycerin. Intra-articular injections are not unusually curative. Insert the needle in the angle between the outer edge of the patella and the ligament of the patella (De Vos). Repeat the injection in one week if reaction has abated, and continue as directed for the injection of the hip-joint. If this plan fails, incise the capsule, remove all fragments and tuberculous foci, irrigate with normal salt solution, inject iodoform emulsion, and sew up without drainage (Neuber's plan). A more severe case requires drainage. If these means fail, or if the case is too far advanced to permit of their use, open the joint and perform an excision or an erasion (see pages 697 and 703). Excision gives a satisfactory result in most cases, although it leaves a rigid knee and marked shortening. Garré considers any shortening over 5 cm. a bad result, and he got such a bad result in 7.5 per cent. of his 117 cases. In children shortening follows even conservative treatment, and the shortening which follows excision is due in part to removal of bone and in part to impairment of the nutritive power of the epiphyseal cartilage. Some cases demand amputation, which, if the patient's health is much impaired or if amyloid disease exists, is to be preferred to excision. Amputation is preferred to excision in very young children and aged people.

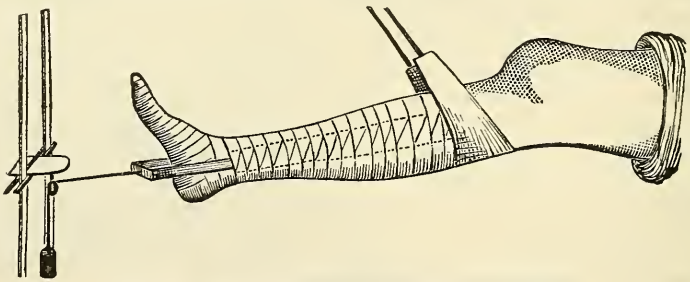


Fig. 389.—Sayre's double extension of the knee-joint.

**Ankle-joint disease** may begin in the synovial membrane, in the tibial epiphysis, or in the tarsus. The origin is frequently synovial.

The *symptoms* are pain, swelling, lameness, limitation of joint-movements, and atrophy of the calf muscles. Caseation often occurs and sinuses form.

*Treatment.*—Conservative treatment with injections of iodoform or of formalin-glycerin will cure many cases. Rest is obtained by means of splints or plaster-of-Paris bandages. Caution the patient to avoid standing upon the diseased extremity. In making an intra-articular injection insert the needle below the outer malleolus. When caseation occurs, it is advisable to open the joint, wash out with normal salt solution, inject iodoform emulsion, sew up the incision, and put up the ankle-joint in plaster. When there is considerable bone disease, when fistulæ exist, when adjacent joints or tendons are diseased, or when joint-disorganization occurs, perform an excision or an erasion. Some cases demand amputation (Syme's amputation being preferred by some, amputation above the ankle being approved by many). Osteoplastic resection is sometimes advised (Wladimiroff-Mikulicz operation). Operative treatment is more satisfactory in children than in adults (Garré).

**Shoulder-joint disease** is not common; it is rare in children and is commonest in adults; it may begin in the synovial membrane, but usually begins in the head of the humerus. The glenoid cavity is rarely attacked. Pain is slight, atrophy of the deltoid and other muscles is noted, the joint is stiff, and the scapula follows the motions of the humerus. Caries sicca is the usual

cause of destruction. In many cases swelling is not obvious, the joint shrinking because of destruction of the head of the bone and contraction of the capsule (Senn). Abscess-formation is unusual. If an abscess forms, it may open in the axilla, through the deltoid muscle, or at some far distant point. Shoulder-joint disease is frequently complicated by pulmonary tuberculosis.

*Treatment.*—A majority of cases recover by the use of conservative treatment, a stiff joint resulting. Put on a shoulder-cap, apply the second roller of Desault, and hang the hand in a sling. Maintain rest for at least four months. Arthur Gillette's plan is very efficient. It consists in placing in the axilla a wedge-shaped pad with the base up. The arm is allowed to hang. Aspiration and injection of iodoform emulsion or of formalin-glycerin are of great service in synovial tuberculosis. In making an intra-articular injection the needle is entered below the acromion, while the arm is held against the side and the forearm is at right angles to the arm and across the front of the chest (De Vos). If caseation occurs, open the joint, remove tuberculous foci, wash with hot saline fluid, inject iodoform emulsion, and close without drainage. In a decidedly severe case, drain. Caries sicca may occur. In rare instances dead bone will have to be gouged away. Excision is sometimes required, but the results are seldom satisfactory.

**Elbow-joint disease** may begin in the humerus or the ulna. The head of the radius is rarely the primary focus. In some cases the synovial membrane is first attacked. The disease is most frequent in young adults. The joint is swollen, its movements are somewhat limited, muscular wasting is pronounced, and pain is generally slight. Tuberculous pus may form.

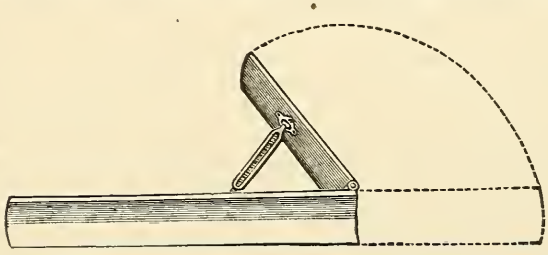


Fig. 390.—Stromeyer's anterior angular splint.

*Treatment.*—In treating early elbow-joint disease, especially in young children, conservative treatment is very successful. Rest is secured by means of an anterior angular splint (Fig. 390) and a triangular sling or a plaster-of-Paris dressing. Splints are to be worn for from four months to a year. Injections of iodoform emulsion or formalin-glycerin are usually employed. Insert the needle for injection by the outer side of the olecranon. In a cure by conservative methods a stiff joint will usually result. It may be necessary to perform resection because of extensive bone disease. Resection gives an excellent functional result.

**Wrist-joint disease** may arise at any age, and is sometimes met with in late middle life or even in old age. The joint presents a puffy swelling, loses its normal contour, and becomes spindle shaped. Hand-movements are impaired, pronation and supination cannot be performed completely or satisfactorily, the joint is stiff and partly flexed, the grasp is enfeebled, pain may be severe or slight, the skin is sometimes, but seldom, hot, and muscular atrophy is marked. This form of tuberculosis may begin in the synovial membrane, in the bones, or in the tendon-sheaths. The prognosis is better than in tuberculosis of any other joint. There is usually preservation of considerable function on recovery.

*Treatment* is usually conservative and very successful, giving, as a rule, a functionally useful joint and movable fingers. Garré recommends a trial of the method even when there are fistulæ and when there is necrosis of the carpus. Apply a Bond splint and sling or put on a plaster-of-Paris bandage and maintain strict rest for from four to six months. Aspiration and injection of iodoform emulsion or formalin-glycerin may be practised. Enter the needle at the dorsal edge of the radial styloid process, and again at the upper edge of the pisiform bone (DeVos). In some cases it is well to incise, wash with salt solution, inject iodoform emulsion, and close without drainage. Severe cases demand incision and drainage with the maintenance of rest. Resection is to be avoided if possible. It gives a bad functional result, the amount of bone removed leaving the tendons too long and contractures of muscles being common (Garré). It may be demanded because of extensive caries or sequestra formation. Amputation is occasionally necessary.

**Non-tuberculous Arthritis.**—Most of these cases, acute and chronic, are of infectious origin. Some of them are non-infectious. Among the non-infective forms are the joint lesions of locomotor ataxia and syringomyelia—constitutional conditions, as gout, purpura, hemophilia, and scurvy—functional derangements expressed by articular neuralgia or intermitting hydrops and traumatic states (Hoffa, in "Zentralbl. f. Chir.," xxxiv, 1907; and Bloodgood, in "Progressive Medicine," Dec. 1, 1907).

**Traumatic Arthritis.**—This may be due to a single injury (a sprain or a bruise) or to some continuing cause (genu valgum may cause arthritis of the knee; flat-foot may be responsible for arthritis of the knee or hip).

If a contusion or sprain causes relaxation of the capsule or fixing tendons, the joint becomes loose and injures itself again and again during movement. It does the same thing when there are loose bodies or enlarged synovial fringes. Traumatic arthritis usually involves but one joint. Recent traumatic arthritis is treated by protecting the joint, massage, hot air, and passive motions.

If a continuing cause is present it is to be removed. Distant causes may be removed by orthopedic apparatus or by operation. Causes within the joint may be sought for by arthrotomy, and when found they should be removed. For instance, in the knee, *inflamed synovial fringes* may be responsible for chronic inflammation. They get caught between the joint surfaces, are squeezed, and trip the victim. They should be removed. Lockwood ("Brit. Med. Jour.," July 3, 1909) points out that an overgrowth of fat may get between the tibia and femur and be squeezed. This surgeon calls attention to the fact that there are "adipose pads immediately above the articular surface of the femur, and on either side of the upper end of the patella" (Ibid.). He calls them the *pads of Malgaigne*, after the French surgeon who described them in 1859. If they are subject to repeated traumatism they should be removed.

**Infective Arthritis.**—In this condition the inflammation is due to bacteria. In some cases pus forms and pyogenic bacteria are demonstrable in fluid removed by aspiration. In other cases, perhaps exhibiting as acute symptoms, no pus forms and no bacteria are demonstrable in the fluid removed by aspiration. The latter cases are due to toxins or to bacteria of attenuated virulence. Secondary infection may occur. In most cases the disease is polyarticular, but if a wound is causal the arthritis will be monarticular. The bacteria may reach a joint by way of a wound from an adjacent focus of osteomyelitis, from a near or distant area of infection, from the genito-urinary tract, or by way of the tonsils. Bacteria not directly introduced into the joint reach it by way of the blood or lymph.

The disease may arise during the course of gonorrhea or any infectious process. It may arise when no area of infection can be discovered. It may

arise in the course of an acute infectious disease (such as erysipelas, typhoid fever, pneumonia, influenza, mumps, dysentery, diphtheria, measles, scarlatina, variola), and may be due to pyogenic cocci, to the specific micro-organism of the acute infectious disease, or purely to microbic products. Joint inflammation arising in the course of or as a sequel to an acute infectious disease may or may not suppurate.

*Symptoms.*—If no suppuration takes place, the *symptoms* of the attack resemble those of rheumatism; if suppuration occurs, the symptoms are the same as those of acute suppurative arthritis, with which disease this form of infective arthritis is identical. Suppuration rarely occurs. Ashby has well described the arthritis which sometimes follows *scarlatina*. It involves the wrists, finger-joints, tendons of the forearms, the knees, ankles, or spine. The joints are painful, but are rarely much swollen or discolored (Howard Marsh). We can distinguish infective arthritis from rheumatism by the fact that it does not migrate and is uninfluenced by antirheumatic remedies. Dislocation may follow the acute manifestations of infective arthritis. In the hip it may simulate congenital misplacement.

*Treatment.*—In every case in which we suspect the condition, diagnostic aspiration is performed. If the fluid obtained contains bacteria Murphy's formalin and glycerin should be injected (2 per cent.). The mixture must be at least twenty-four hours old. In many cases this treatment is of the greatest value. If aspiration and injection fail, arthrotomy and irrigation are indicated. In all clinically severe cases and in all prolonged cases, open and irrigate, first with corrosive sublimate solution (1 : 1000), then with normal salt solution. Recent cases which are not very acute and are free from pus may be closed without drainage.

**Pyogenic or Acute Suppurative Arthritis.**—This condition is a form of infective arthritis and is usually due to the Staphylococcus pyogenes aureus or to the Streptococcus pyogenes, which find entrance by means of a wound, by the spontaneous evacuation into a joint of the products of an osteomyelitis, by extension of suppurative inflammation through contiguous structures, or by the blood-stream. It is necessary to remember that causative bacteria may have entered the blood or lymph at a point near to or distant from the joint (tonsils, ethmoid cells, urethra, a focus of osteomyelitis, puerperal sepsis, etc.). Of course, a wound into a joint may be the open gateway for infection. A traumatism may create a point of least resistance and bacteria may be derived from the blood or lymph. It is not very unusual for traumatic arthritis to eventuate in pyogenic arthritis. Particularly in youths and young children the symptoms of arthritis may overlie and hide a causative osteomyelitis. Sometimes gonorrhea is the cause and in rare cases septicemia is causal. In pyogenic arthritis all the joint-structures are involved and suppuration rapidly appears. Synovial membrane is converted into granulation tissue and cartilage is destroyed by pus. The greater the intensity of the inflammation, the larger the amount of granulation tissue, hence, ultimately, the greater the amount of scar tissue and the greater impairment of joint function.

The *symptoms* of acute suppurative arthritis are usually a chill followed by fever and a rapid pulse. There are severe pain, which is aggravated by motion and is worse at night; discoloration, heat, and edema of the skin; partial flexion of the joint; fluctuation, and marked constitutional symptoms of sepsis. The joint tends to rapid disorganization, and fatal septicemia is very apt to occur. In pyemic arthritis several joints become infected.

*Treatment.*—In every suspicious case immediately aspirate. If bacteria are found in the aspirated fluid, at once open the joint (*arthrotomy*) and irrigate it with corrosive sublimate solution (1 : 1000) and then with salt solution. In early cases which are not very violent formalin-glycerin may be injected.

the wound closed, and the limb immobilized after the operation. In a late case or a violent case, drain by rubber tissue. Always be sure whether or not arthritis is the result of osteomyelitis. If osteomyelitis exists the area of bone disease must also be operated upon. Arthritis due to staphylococci and streptococci is often secondary to bone suppuration. If a periosteal abscess exists the joint condition is almost certainly secondary. If osteomyelitis exists it requires prompt and radical treatment (see page 507). In advanced cases involving the knee Allen and Alden ("Surg., Gynecol., and Obstet.," July, 1909) open the joint by a transverse incision below the patella, disinfect with pure carbolic acid followed by alcohol, and pack with iodoform gauze. I would only regard this as justifiable in advanced and very severe cases, as it is sure to be followed by ankylosis. Although in late cases which recover after arthrotomy and irrigation there is always more or less ankylosis, many cases treated early recover without serious impairment of joint function. Early arthrotomy is of the utmost importance, and if the aspirated fluid contains bacteria we should never postpone operation or hold it in reserve while we employ Bier's hyperemic or any other conservative method. Radicalism is here the course which promises the greatest safety, and the surest retention of joint function.

**Typhoid Arthritis.**—This disease is a form of infective arthritis. That the bacteria of typhoid may inflame the joints is proved, and it seems certain that they can cause suppuration, although their pyogenic power has been disputed. Some claim that mixed infection is the real cause of pus formation in a typhoid joint. The typhoid bacilli enter the bones in many typhoid cases and sometimes cause osteomyelitis. Joint-disease is more common than bone-disease. Typhoid disease of a joint begins when the fever is abating, and more than one joint may be involved. Typhoid joints may recover permanently, may become ankylosed, may dislocate, or the joint-disease may lead to fatal sepsis. In most cases the joints recover. In slight cases the synovial membrane only is involved; in more severe cases capsule, cartilages, ligaments, and even bones are involved. Some cases suppurate. Septic typhoid arthritis may result from a mixed infection with typhoid bacilli and pyogenic bacteria, and is identical in symptoms and progress with ordinary septic arthritis. Typhoid arthritis proper may be monarticular or polyarticular, the monarticular form being the most common, and the hip-joint being the articulation most liable to be attacked. In most cases typhoid arthritis causes little pain. The swelling is marked, although in the hip it is concealed. Pus rarely forms. Keen calls attention to the fact that in the 84 cases of typhoid arthritis which he collected spontaneous dislocation occurred in 43, nearly all in the hip.[1] Fluid from a typhoid joint may be sterile (bacteria having died), may show mixed infection, or may give a pure culture of typhoid bacilli (A. G. Ellis, in "Jour. of Infectious Diseases," April 1, 1909).

*Treatment.*—A mild case is treated as a simple synovitis. If diagnostic puncture obtains fluid free from bacteria, no more radical method than aspiration and irrigation is required. If the fluid contains bacteria, inject formalin-glycerin. If this fails, incision and drainage are demanded. In some cases an autogenous vaccine appears to be of service, in others it fails utterly.

**Gonorrheal Arthritis or Gonorrheal Rheumatism.**—During the progress of gonorrhea the development of a painful joint does not of necessity prove the existence of gonorrheal rheumatism, for ordinary rheumatism is just as likely to arise when a man has clap as when he has not this malady. Furthermore, the term is inaccurate, as gonorrheal rheumatism is not rheumatism at all, but is an infective disorder of the joints or of the synovial membranes, the infective material being contained primarily in the urethral

[1] Keen on "The Surgical Complications and Sequels of Typhoid Fever."

discharge. Gonorrheal arthritis is one of the forms of infective arthritis. Occasionally this form of arthritis arises from gonorrheal ophthalmia (Heiman's case); it sometimes, though rarely, arises during the height of a gonorrhea, but it is more frequently met with in chronic cases or when the intensity of the inflammation is abating in acute cases. Men suffer from gonorrheal arthritis far more frequently than do women, and the seizure is very apt to recur again and again. In some cases many joints are involved, but in most cases only a few joints suffer. Osler states that the knees and ankles are most apt to be involved in gonorrheal rheumatism, and that this form of arthritis is peculiar in often attacking joints that are usually exempt in acute rheumatism ("the sternoclavicular, the intervertebral, the temporomaxillary, and the sacro-iliac"). There are two forms of gonorrheal rheumatism—an acute and a chronic form. The poison reaches the joint by way of the blood. In some cases gonococci are found in the joint fluid; in other cases they are not found. I am inclined to believe that in the milder cases, which recover without genuine pus formation, only toxins are present in the joint. In the severe cases the organisms themselves exist in the articular fluid. Osler suggests that the non-suppurative cases are due to the action of toxins taken up from the area of primary infection, and that the suppurative cases are due to infection with pyogenic bacteria. Endocarditis may occur, and it is due to micro-organisms and not to toxins.

*Changes In and About the Joint.*—The inflammation of gonorrheal arthritis may be located around rather than in the joint, and especially in the tendon-sheaths. Suppuration is unusual, but it may occur in joints and in tendon-sheaths. Cultures of the exudate may or may not show the gonococci. Cover-glass preparations carefully stained may or may not show gonococci.

*Symptoms.*—The acute form attacks, as a rule, but a single joint, but may attack several joints. The joint trouble begins with great suddenness, and is often ushered in by chilly sensations or by a distinct chill. Moderate fever arises. The pain in the joint, severe from the first, becomes excruciating. If superficial joints suffer, the skin over them becomes red and hot, and periarticular edema soon presents itself. The fluid in the joint is in most cases serous, but may become purulent. If pus forms, the fever becomes very high and chills may occur.

A chronic condition may follow the acute, but the condition may be chronic from the start. The symptoms resemble those of the acute form, but are far milder, although acute exacerbations may occur. The joint fluid is usually serous.[1] In gonorrheal arthritis there may be transitory, intermittent, and wandering pain in and about the joint, without any other symptom; one or more joints may become swollen and painful, and moderate fever may develop. One joint, especially the knee, may swell to an enormous extent, pain, periarticular edema, redness, and fever being absent (hydrarthrosis, or dropsy of the joint). Suppuration in this form of the disease seldom occurs. The tendons, the tendon-sheaths, the bursæ, and the periosteum may inflame. Whether the joints are inflamed or not inflamed, the tendon-sheaths about the wrist and ankle and the retrocalcaneal bursæ may suffer. In some cases numerous bursæ are involved. It is often difficult and is perhaps impossible to check gonorrheal arthritis. It may last for a long period, and tends to recur again and again. Iritis, periostitis, pleuritis, endocarditis, and pericarditis have been observed as complications.

The *diagnosis* between gonorrheal arthritis and acute rheumatism rests chiefly on the great chronicity, the lesser degree of fever, the excessive tendency to recurrence, and the absence of profuse acid sweats in gonorrheal

[1] See Schuller, in "Aertztl. Pract.," No. 17, 1896.

arthritis; and on the shorter course, the higher fever, the profuse acid sweats, the lesser tendency to rapid recurrence, the greater proneness to symmetrical involvement, and the great liability to cardiac and visceral complications in rheumatic fever. Furthermore, in gonorrheal arthritis a gonorrheal infection (urethral or ocular) certainly exists or recently existed; in ordinary rheumatism a urethral discharge may, of course, happen to be present. Gonorrheal arthritis is apt to affect certain joints which acute rheumatism seldom attacks.

*Treatment.*—Because of the lingering character and dangerous nature of gonorrheal arthritis and because if unchecked it is liable to produce grave impairment of function, treatment should be prompt and radical, as advocated by Halsted many years ago. The joint should be aspirated and if the fluid obtained contains gonococci or any pyogenic bacteria, formalin-glycerin should be injected. If this fails, the joint should be opened and irrigated. If pus is absent and the case not very violent, the joint may be injected with formalin-glycerin and the wound can be closed without drainage. If pus is found by incision, irrigate, drain, and immobilize. In cases free from pus Bier's treatment is of value (see page 112). Drug treatment is of little value in gonococcal arthritis. The salicylates, the alkalis, and salol are useless; iron, arsenic, and strychnin are possibly of some benefit. Quinin is thought to be helpful in some cases. Large doses (1 dram) of syrup of iodid of iron are given by some clinicians, associated with tonic doses of corrosive sublimate. Iodid of potassium seems to be of a certain amount of value. The inflamed joints are usually wrapped in cotton and bandaged, and every day a little blue ointment is rubbed into the skin about them. If the inflammation lingers, it is customary to use the hot-air oven, massage, and gentle passive motion, to apply blisters, or to counterirritate with the hot iron. The object in this stage is to absorb infiltrations and adhesions and thus lessen stiffness. Early passive motion allays reflex muscular contractures, combats atrophy, and prevents adhesion (see Bier and Baetzner, in "Practitioner," June, 1912). It is thought by some competent clinicians that antigonococcic serum possesses distinct value, greatly alleviating pain and favoring the restoration of joint mobility. My experience with it is as yet too insignificant to justify me in expressing an opinion.

The value of vaccine treatment is estimated highly by some, but many have obtained no beneficial results.

**Pneumococcus Arthritis.**—This is a rare condition, although Herrick collected 52 cases ("Amer. Jour. of Med. Sciences," July, 1902). Examination of the blood may or may not discover pneumococci, and pneumococci may be found in the blood during pneumonia, when the joints are free from disease. The inflammation may attack any joint, but is most apt to arise in a joint weakened by previous injury or damaged by rheumatism or gout. Alcoholics are more prone to suffer than others. In a great majority of cases the disease is associated with lobar pneumonia, but Cole's case proves that the lung may be free ("American Medicine," May 31, 1902). As a rule, a single large joint is attacked, and the knee is most liable to suffer. The synovial membrane alone may be involved or cartilages may suffer and bone be attacked. The fluid may be serous, but is usually purulent (Herrick). I have seen 2 cases: in 1 case the knee only was involved; in the other, both knees, one elbow, and one shoulder were attacked. In Cole's series of 41 cases, 13 exhibited involvement of more than one joint. The inflamed joint is frequently completely destroyed. Pneumococcus arthritis develops, as a rule, soon after the crisis of pneumonia, but Herrick says it may arise as late as three weeks after the crisis.

The diagnosis is made by the history of pneumonia, the development of septic symptoms, and the signs of joint inflammation. It is confirmed

by aspiration and examination of the fluid. The disease is very fatal. In Herrick's series of cases over 65 per cent. were fatal. In Cole's series of cases there were 28 deaths and 13 recoveries. Even if the patient recovers, the convalescence is prolonged and more or less ankylosis is to be expected.

*Treatment.*—A non-purulent effusion may be treated by aspiration if bacteria are not found in the fluid. If the aspirated fluid contains bacteria, formalin-glycerin is injected. If this fails, or if pus is present, the joint should be opened and drained.

**Syphilitic Arthritis.**—(See pages 326 and 331.)

**Acute Rheumatic Arthritis; Rheumatic Fever or Acute Rheumatism.**—Acute rheumatism is a self-limited febrile malady whose characteristic features are polyarthritis, profuse acid sweats, and a tendency to heart involvement. There is some evidence to indicate that acute rheumatism is a form of infective arthritis, the bacteria being deposited in the synovial tissues and later perhaps entering into the joint cavity. Arthritis of many joints has followed intravenous injection into animals of diplococci obtained from the throat of a man suffering from rheumatic angina (Poynton and Paine at Manchester meeting of the Brit. Med. Assoc., 1902). John O'Conor[1] believes that acute rheumatism is a condition "something similar to gonorrheal arthritis and pyemia, the germ or toxin gaining admission to the body through the tonsil or other microbic trap-door, and that the joint invasion is promptly followed by a form of infective arthritis accompanied with general toxemia; and, furthermore, the infected joints serve as incubators, where the poison is elaborated and passed into the circulation and thus conveyed to other articulations and to the heart."

*Symptoms of Acute Rheumatism.*—In acute rheumatism the case begins with malaise and fever, and one or more joints become affected. The inflammation spreads from joint to joint, is apt to be symmetrical, and when it arises in fresh joints, usually disappears quickly in those previously affected. The temperature is high, the skin sweats profusely, the joints are red, swollen, hot, and excruciatingly painful, and the structures about the joints are edematous. After a short time the inflammation subsides in one joint and passes into another, the joint first attacked regaining its functions. Suppuration does not take place. Anemia is pronounced, exhaustion is profound, the sweat is sour, the saliva is acid; the urine is acid, scanty, high colored, often contains albumin, and is deficient in chlorids. Cardiac disease is apt to be produced (endocarditis, pericarditis, or myocarditis). Nodules may form upon fibrous structures, hyperpyrexia is not unusual, and cerebral or pulmonary complications may occur.

The *treatment* of acute rheumatism comprises the use of alkalis, salicylates, etc. (See a book upon practice of medicine.) O'Conor is a believer in incising and draining the inflamed joints; and if the theory of an infective origin is correct, this treatment is rational. I have never ventured to do it, but would consider the advisability of doing so if the ordinary treatment proved futile. O'Conor operates early and believes that this is the real way to arrest the disease and prevent complications, but his views have not met with general acceptance.[2]

**Chronic rheumatism** sometimes follows repeated attacks of acute rheumatism, but oftener arises insidiously in people who have been exposed to cold and damp, who have suffered from poverty, hardship, and privation, or have had much worry. The capsule and tendon-sheaths thicken, and there is usually but little effusion into the joint, but the articulation becomes stiff and painful. The joint-cartilages are occasionally eroded. Muscular atrophy occurs.

[1] "Lancet," Jan. 24, 1903. [2] Ibid.

*Symptoms of Chronic Rheumatism.*—In chronic rheumatism the affected joints are stiff and painful and are a little swollen, but not red. Dampness and cold aggravate the symptoms. One joint or many may be affected, but usually several are involved. Passive movements cause the joint to creak and develop crepitus in the tendon-sheaths. The muscles are wasted. Anemia is usually pronounced. The smaller blood-vessels become surrounded by fibrous tissue which progressively contracts and lessens the blood-supply of the synovial structures. The joints may ankylose. There is no fever and no tendency to suppuration, and the disease is incurable.

*Treatment.*—In chronic rheumatism maintain the general health of the patient, give courses of iron, arsenic, and strychnin, and an occasional course of iodid of potassium or a salt of lithium, and, if possible, send him every winter to a warm climate. Turkish baths give considerable temporary relief. The waters and regimen of Carlsbad and Vichy are of positive though temporary benefit, and the sufferer may obtain relief at the hot springs of Virginia. The patient must avoid damp and must wear woolens. Frictions, the douche, massage, flying blisters, counterirritation with the hot iron, ichthyol ointment, and mercurial ointment are of benefit. Subjecting the diseased joint to a very high temperature by placing it daily in a hot-air apparatus often does great good. The pains of chronic rheumatism may be greatly benefited by the plan communicated to me by Dr. J. T. Rugh. He makes a mixture of 14 min. of glycerin, 14 min. of sterile water, and 1 gr. of crystallized carbolic acid. This is injected at two points, about the articulation and in the extracapsular structures. As soon as an injection has been made the area must be vigorously rubbed to diffuse the fluid and prevent sloughing. The joint must be kept at rest for three days. Injections can be repeated at intervals of five days. In partial ankylosis it is proper in some cases to give ether and break up the adhesions.

**Gouty arthritis,** which appears especially in the smaller joints (as the fingers and the metatarsophalangeal joints of the great toes), is due to a deposition of urate of sodium in the joint and in the periarticular structures. The irritant urate of sodium causes inflammation, inflammation leads to the formation of granulation tissue, granulation tissue is converted into fibrous tissue, and the fibrous tissue contracts and thus deforms the joint and limits its mobility. A great mass of urates clinging to a joint constitutes a *chalk-stone.*

*Symptoms.*—The premonitory symptoms may be observed for a day or so, but the acute seizure usually occurs early in the morning, the patient, as a rule, being aroused by excruciating pain in the metatarsophalangeal articulation of one of the great toes. The joint swells, and the skin over it feels hot to the touch and becomes red and shiny. There is often considerable fever. After a few hours the intensity of the seizure abates, only to recur again with renewed violence early the next morning, these remissions and recurrences taking place during six or eight days, until the attack subsides. In patients with *chronic gout* many joints are stiffened and deformed as a result of repeated attacks. Chalk-stones form, and the skin above them may ulcerate. Such patients are chronic dyspeptics, have high-tension pulses, their hearts are hypertrophied, and their urine contains albumin and casts.

The *treatment* of gouty arthritis belongs to the physician, and not to the surgeon, although to the latter the symptoms of the disease should be known, so that it may be diagnosticated from other maladies.

**Osteo-arthritis** (*Rheumatoid Arthritis; Arthritis Deformans; Rheumatic Gout*).—In this disease, which is not a combination of gout and rheumatism, the synovial membrane and cartilages are affected, the periarticular structures are involved, and masses of new bone are formed.

Osteo-arthritis possibly has in certain cases, as John K. Mitchell long ago

pointed out, a nervous origin. It arises especially in persons who have been worried, driven, and harassed. There is apt to be muscular atrophy, trophic lesions of the hair and nails are likely to appear, and the symptoms are disposed to be symmetrical. The causative lesion has not been determined. The condition is now generally regarded as of toxic origin. The foci of infection may lie in any part of the body, but are most frequent in the tonsil, accessory sinuses of the nose and nasopharynx, the middle ear, the bronchial glands, the mesenteric glands, and the intestines, especially the large intestine. The disease is commoner in women than in men. The greatest liability exists between the ages of twenty and forty, but children may acquire the disease, and it may also be developed in people far beyond middle life. Apes in captivity may develop it. Arthritis deformans may attack the rich or the poor; it does not result from gout, nor does it often follow rheumatism; it is not caused by damp and cold, and only in rare cases does it arise after traumatism of a joint.

Osteo-arthritis differs from gout in the entire absence of urate deposit, and it differs from chronic rheumatism in the extensive alterations in the joint structures. The changes begin in the cartilage; the cartilage-cells multiply, the intercellular substance degenerates, the pressure of the bone causes thinning, and at length the cartilage is entirely destroyed and the bone exposed. The exposed bone is altered in shape, is hardened, and is worn away in the center, the periphery increasing in thickness by ossific deposit, the center deepening by absorption. The margins are not only thickened, but are bulged and lengthened by deposit. The fringes of the synovial membrane hypertrophy and multiply, and some of them are apt to break off (*loose cartilages*). The capsule and the ligaments of the joints, as a rule, become fibrous and contract; but they may soften, relax, and permit of dislocation. The joint usually contains no effusion, but in some cases there is great effusion (*hydrarthrosis*). The tendons about the joint may become fibrous and contracted, they may ossify, they may be separated from the bone, or they may be destroyed entirely. Deformity is marked and motion is limited. The fingers, when involved, show nodules on the sides of the joints (Heberden's nodules). The vertebræ may be involved. Almost all the joints may suffer. Suppuration does not occur.

*Symptoms.*—Charcot divides osteo-arthritis into three forms, and gives their symptoms, as follows:

1. *Heberden's nodosities*, which condition is commoner in women than in men, usually begins between the ages of thirty and forty, and is especially common in neurotic subjects. The interphalangeal joints become the victims of attacks of moderate swelling and of some tenderness, which attacks are not severe, but recur again and again. After a time small hard swellings (nodosities) appear upon the sides of the dorsal surface of the second and third phalanges, remain permanently, and slowly increase in size. The joints become stiff and creak on movement, the cartilages are destroyed, and contractions and rigidity develop, but there is no fever and the larger joints are not involved. The malady is incurable.

2. *Progressive rheumatic gout*, which may be acute or chronic. The *acute* form begins as does rheumatic fever. There are moderate fever and swelling, without redness, of a number of joints, of bursæ, and of tendon-sheaths; the joints are stiff and crepitate, and are apt to be symmetrically involved; muscular atrophy begins early and rapidly becomes decided; pain is slight. This acute form is apt to arise in young women after pregnancy, but is not unusual at the climacteric and in children. Anemia always exists. The case is apt to advance progressively until a number of joints are firmly locked, when it may become stationary. Another pregnancy will develop anew the acute symptoma. In the *chronic* form swelling and pain on movement are noted in certain joints. The involvement is apt to be symmetrical. Attacks of swelling

and pain alternate with periods of apparent quiescence, but the disease does not cease its advance. Articulation after articulation is attacked by the malady until almost all the joints are involved; deformity and stiffness become pronounced, and pain may or may not be severe. There is no fever. Muscular atrophy is marked.

3. *Partial rheumatic gout* or *monarticular rheumatism* attacks one articulation, and it is most often met with in old men. It may fix itself on the vertebral column, on the knee, on the shoulder, on the elbow, or on the hip. The joint grates and becomes stiff, swollen, and deformed; the muscles atrophy; there is usually pain, but fever is absent.

**Osteo-arthritis** or **partial rheumatic gout of the hip-joint** seldom occurs before the age of forty-five, but is occasionally, though very rarely, met with in persons under twenty-five. If the disease arises in an elderly person, it is often called *morbus coxæ senilis*. In some cases only the hip-joint is attacked; in many cases other joints are also diseased. Osteo-arthritis of the hip may follow an injury. Usually the disease is unconnected with traumatism, begins very gradually, and advances slowly. There is pain, often mistaken for sciatica, in and about the joint, and there is increasing stiffness. The pain and stiffness are worse when the patient first moves after resting. Lameness becomes noticeable, and grating can be detected in and about the joint. The symptoms become gradually worse, although at times they may seem to improve for brief periods. The lameness and the stiffness are greatly aggravated, and the pain becomes very severe, even when at rest. Shortening takes place, the great trochanter ascends above Nélaton's line, the limb is usually abducted, but in very rare cases is adducted, and finally ankylosis occurs.

Partial rheumatic gout of the vertebral articulations causing fixation is called *spondylitis deformans* (see p. 848).

*Treatment.*—Osteo-arthritis cannot be cured, but in some cases it remains stationary for many years. I have seen one case apparently arrested by removal of a diseased appendix. It is claimed by some that Lane's operation of extirpation of the large intestine may arrest it. As yet I would not feel justified in recommending a theoretical operation with the idea of directly benefiting osteo-arthritis. I would, however, remove, if possible, any *obvious* area of infection. The usual plan of medical procedure is to treat the anemia by iron, arsenic, nourishing food, and have the patient out in the fresh air as much as possible. Debility is met by the administration of strychnin. Hot baths of mineral water do good. It is claimed that the hot-air apparatus is of service. Douches improve these cases, but electricity is useless. Counterirritants do no good. Massage retards the progress of the case, relieves the pain, aids in the absorption of effusion, and delays fixation. During an acute exacerbation the joint should be put at rest for a time and evaporating lotions applied for a few hours. In an exacerbation in disease of the hip the patient should be put to bed and have extension applied. The patient is, unfortunately, liable to develop the opium-habit. If dropsy of a joint arises, try compresson by Martin's bandage, and, if this fails, aspirate and wash out the joint with a 2 per cent. solution of carbolic acid. Patients with rheumatic gout do best in a warm, dry climate. Cod-liver oil does good, as it improves nutrition and hence retards the progress of the disease. Do not be tempted to immobilize the joints beyond a day or two: fixation only hastens ankylosis. Howard Marsh[1] maintains that, as a rule, but little good comes from manipulation. He makes the following exceptions: when one joint only is affected; when the joint is very stiff but not very painful; when the patient is in good general health and is not beyond middle age. If only one joint is involved it may be proper to produce ankylosis by operation. When ankylosis occurs all symp-

[1] "Diseases of the Joints and Spine."

toms subside. Albee describes an operation for osteo-arthritis of the hip-joint. He removes a thin layer from the top of the head of the femur and a similar amount of bone from the roof of the acetebulum. The two surfaces are held in contact until union occurs. In the knee-joint Hibbs bridges the front part of the joint with the denuded patella. Later in such a case, after all symptoms have subsided, it may be justifiable to re-establish function by interposing a connective-tissue flap or chromicized pig's bladder (R. T. Taylor).

**Trophic Joint Affections** (*Arthropathies*).—It is well known that certain diseases and injuries of brain, cord, and nerves may be responsible for arthritic changes (hemiplegia, injury of the cord, locomotor ataxia, neuritis).

From three to six weeks after an apoplexy the joints of the palsied side are apt to suffer from inflammation. The condition following apoplexy is one of synovitis. Any joint may suffer, but the hip eldom does. A well-known arthropathy is Charcot's joint (Figs. 391, 392).

Fig. 391.—Charcot's joint.

**Charcot's Disease** (*Tabetic Arthropathy; Charcot's Joint; Neuropathic Arthritis*).—This condition is an osteo-arthritis due to trophic disturbance, arising in a sufferer from locomotor ataxia, and is anatomically identical with osteo-arthritis, which was described above. The knee is most apt to be attacked, and the hip suffers more often than any joint but the knee. The condition may develop in the shoulder or elbow. The smaller joints sometimes, though seldom, are involved. More than one joint may suffer. The disease in most cases begins acutely, often as a sudden effusion, which after a time may disappear. In most cases, however, the joint becomes rapidly disorganized. The swelling is usually very marked and is sometimes enormous. In the earliest stages it is due to periarticular edema and to articular effusion. Pain is slight or is absent, there is no constitutional involvement, and the condition is unconnected with injury. Some cases begin without this preliminay acute swelling, disorganization being manifest from the beginning. When disorganization has once begun, it continues inexorably. Bony masses form around the articular cavity, in the ligaments, and in the cartilages. The bones and cartilages are rapidly destroyed and absorbed; fracture is apt to occur; the joint creaks and grates; the softening and relaxation of the ligaments permit an extensive range of movement; great deformity ensues; dislocation is apt to occur; muscular atrophy is decided, and pus occasionally, though very rarely, forms. There is sometimes, but seldom, repair. Charcot's joint differs from rheumatoid arthritis in the usual acute onset and the painless course. The complete or nearly complete freedom from pain is one of the most striking features of the condition. In saying there is freedom from pain we mean freedom from pain in the joint, from the pain and tenderness in the regions in which we expect to find them in an inflamed joint. Usually these patients, though free from pain in the joint, suffer much from the lightning pains of locomotor ataxia. Gastric crises are not uncom-

mon (Bramwell). Charcot's joint is more common in female than in male tabetics. In saying that Charcot's joint is often of sudden origin, we mean that in a single night, as Charcot pointed out, swelling of a joint may arise. In a day or two the joint swelling becomes great, and if aspiration is performed, yellow serum is obtained. In a week or two the joint begins to creak on movement.

The *treatment* of Charcot's disease consists in the wearing of an apparatus to sustain the joint. Resection is recommended by some, but most surgeons do not advise its performance. Southam advocates amputation for certain cases of Charcot's joint. He has performed the operation on 4 patients. He amputated twice for ankle-joint disease and twice for disease of the tarsus. In every case the stumps healed quickly and without suppuration. Southam was led to perform amputation on his first case by the report of Jonathan Hutchinson's case of amputation of the leg for perforating ulcer and disease of the bones of the foot in a tabetic.

Fig. 392.—Charcot's joint.

**Osteo-arthropathie Hypertrophiante Pneumique** (*Marie's Disease*).—A condition associated with, and possibly springing from, pulmonary disease, and characterized by enlargement of joints, thickening of the finger-ends, and the formation of a dorsolumbar kyphosis. The joints are painful, the skin undergoes pigmentation, and profuse perspiration is often present. The head entirely escapes in this disease, which immunity marks a distinction from acromegaly.

**Hysterical joint** (*Brodie's joint*) is a condition mostly encountered in young women. The disease occurs most commonly in the knee and the hip, and often follows a slight injury which acts as an autosuggestion, a latent hysteria being awakened into action and localized, though severity of the injury does not determine the severity of the symptoms. The disease may ensue upon synovitis or arthritis, or may arise without apparent cause. The patient complains of pain in and stiffness of the joint, resists passive motion strenuously, and claims that it causes much pain. There is occasionally some muscular atrophy from want of use, and the joint is a little swollen. The skin is hyperesthetic, and a light touch causes more pain than does deep

pressure. The muscles may be rigid. The joint may be maintained either in flexion or in extension, but it is rarely in the exact degree of flexion assumed for ease in a true joint inflammation, and the position is apt to be changed from day to day or from hour to hour. The skin is usually pale and cool, but may be red and hot, because of hyperemia. A periodically developed heat may be observed, especially at night, accompanied apparently by much pain. The alleged pain in some cases is neuralgia, but in most cases is a pain hallucination. There is no effusion into the joint, and swelling does not exist, although occasionally there is slight periarticular edema. In some rare cases organic disease arises in a hysterical joint.

Hysterical phenomena are seldom isolated, but are associated with certain stigmata which may be latent. These stigmata are concentric contraction of the visual fields, pharyngeal anesthesia, convulsions, hysterogenic zones, globus hystericus, clavicus hystericus, zones of anesthesia, especially hemianesthesia, and hyperesthetic areas. Such patients are predisposed by inheritance, and have previously, as a rule, had nervous troubles. Hysterical phenomena, be it remembered, lack regularity of evolution, and are produced, altered, or abolished by mental influences and physical sensations which are without effect in causing, modifying, or curing organic disease. The general health, as a rule, is good, but neurasthenia may coexist. In examining these patients the observer will note that the symptoms disappear when the attention is diverted; that they are out of all proportion to the local evidences of disease; that there is no sign of joint destruction, and that a light touch may cause more pain than does firm pressure. If the patient is anesthetized, perfect joint mobility will be found without any evidences during function of joint changes.

The *treatment* for a hysterical joint comprises attention to the general health, the employment of nourishing and easily digested food, the prevention of constipation, and the administration of tonics if they are needed. The surgeon must dominate his patient's mind and make her realize that he is master of the case. He is to be an inexorable but just ruler—never a brutal or a cruel one. If possible, send the patient away from the harmful sympathies of her home and let her have the rest treatment of S. Weir Mitchell. Local remedies applied to the joint do harm, as a rule, by concentrating afresh the patient's attention upon the articulation, although the hot iron sometimes does good. Suggestion in the hypnotic state may be tried. The use of morphin should be avoided as being the worst of enemies. Never immobilize the joint, and always use massage, passive motions, and frictions.

**Neuralgia of the joints** as an independent, isolated affection is extremely rare, though as a complication of other diseases it is by no means uncommon. Neuralgia is more common outside of the joints than in them, and periarticular neuralgia is especially frequent about the knee and the ankle. Joint-neuralgia may arise in any person, but it is more commonly present in young neurotic females. The pain may be persistent, or it may occur in periodic storms, and it is often associated with neuralgia in other parts. The pain may be dull and aching, but it is more often sharp and shooting. Joint-neuralgia is associated with tenderness on pressure, soreness on motion, often with transitory swelling without redness, and sometimes with numbness of the extremities. The *diagnosis* depends on the temperament of the patient, the sudden onset of the pain, the absence of constitutional symptoms, and the free mobility of the joint, especially under ether. Articular neuralgia may depend upon disease or injury of the central nervous system, upon malaria, syphilis, neurasthenia, rheumatism, gout, hysteria, and neuritis, and may be due to reflected irritation, especially from the ovaries, the uterus, or the rectum.

The **treatment** to be observed in joint-neuralgia is to maintain the general health. Examine for a possible exciting cause, and, if found, remove it. Give a long course of iron, quinin, and strychnin or arsenic. In rheumatic or gouty subjects administer suitable drugs and insist upon the use of a proper diet. During the attack use phenacetin. Morphin must occasionally be given in severe cases, but be sparing of it, and never tell the patients they are taking it, as there is a possibility of their forming the opium-habit. Locally, employ frictions, ointment of aconite, heat, and keep upon the part a piece of flannel soaked in a mixture of soap liniment, laudanum, and chloroform (Gross). Never allow the joint to stiffen; any tendency to stiffness should be met by daily massage, frictions, passive motion, and hot and cold douches. In some rare cases nerve-stretching or neurectomy becomes necessary.

**Articular Wounds and Injuries.**—A *penetrating* wound is very serious, and it may be due to a compound fracture, to a compound dislocation, to a gunshot-wound, or to a stab. If a bursa near a joint be opened, secondary penetration may occur as a result of suppuration. In a penetrating wound, besides pain, hemorrhage, and swelling there is a flow of synovial fluid. A small amount of synovia flows from an injured bursa, a large amount from an open joint.

**Treatment.**—If a joint is opened aseptically (as when incised by the surgeon) the wound heals nicely under rest and asepsis. If a joint is opened by a septic body, suppurative arthritis is apt to arise, and the surgeon endeavors to prevent it by asepticizing the surface, irrigating the joint, draining, applying antiseptic dressing, and securing rest. Normal salt solution is the best agent for irrigation, as it does not injure joint endothelium. Active antiseptics may lessen tissue resistance, and thus may actually favor infection. In gunshot-wounds inflicted by pistol bullets or sporting rifle bullets, if antisepsis is not employed, suppuration is inevitable; hence military surgeons in the past, as a rule, have advocated amputation or excision in gunshot-splinterings of large joints. Recent experience shows that the wound of a large joint produced by a hard-jacketed and small-caliber bullet may heal with little trouble. In articular wounds the surface is sterilized, and usually the wound is enlarged, the finger is introduced to discover and remove foreign bodies, through-and-through drainage is secured, a tube is inserted, the joint is irrigated, antiseptic dressings are applied, and the extremity is placed upon a splint. Very severe joint injuries demand resection or even amputation. Ankylosis, more or less complete, often follows a gunshot-wound of a joint. If the joint suppurates, the drainage must be made more free, sinuses must be slit up and packed, sloughs must be cut away, dead bone must be gouged out, and the patient must be placed upon a stimulant and tonic plan of treatment. The above remarks do not apply to wounds inflicted by the modern military projectile. Such wounds are not of necessity infected, and recovery may be prompt and uneventful if the surface is sterilized and antiseptic dressings and splints are applied.

**Sprains.**—A sprain is a joint-wrench due to a sudden twist or traction, the ligaments being pulled upon or lacerated and the surrounding parts being more or less damaged. A sprain is often a self-reduced dislocation (Douglas Graham). The joints most liable to sprains are the knee, the elbow, and the ankle. The smaller joints are also often sprained, but the ball-and-socket joints are infrequently sprained, their normal range of free movement saving them; they do occasionally suffer severely, however, as a result of abduction. In a severe sprain the ligaments are torn; the synovial membrane is contused or crushed; cartilages are loosened or separated; hemorrhage takes place into and about the joint; muscles and tendons are stretched, displaced, or lacerated; vessels and nerves are damaged; the skin is often contused; and portions of

bone or cartilage may be detached from their proper habitat, though still adhering to a ligament or tendon (sprain fractures). Sprains are commonest in young persons and in adults with weak muscles. They happen from sudden twists and movements when the muscles are relaxed. A large part of the support of joints comes from muscles, and when muscles are suddenly caught unawares they do not properly support the joint, and a sprain results. A joint once sprained is very liable to a repetition of the damage from slight force. Sprains are common in a limb with weak muscles, in a deformed extremity in which the muscles act in unnatural lines, and in a joint with relaxed ligaments.

**Symptoms.**—There is severe pain in the joint, accompanied by general weakness. Nausea, vomiting, and even syncope may occur. There is impairment or loss of ability to move the joint. The above-described condition is succeeded by a season of relief from pain while at rest, numbness being complained of, and pain on motion being severe. Swelling arises very early if much blood is effused. In any case swelling begins in a few hours. Extensive effusion, by separating joint surfaces, produces slight lengthening of the limb. Movements of the joint become difficult or impossible; the tear in the ligament may sometimes be distinctly detected by the examining fingers; pain and tenderness become intense; joint-crepitus will be manifested, and in a day or two discoloration becomes marked. Moullin and others have pointed out that when a muscle is strained the skin above it becomes sensitive, especially at tendinous insertions over joints. As muscles are invariably strained when a joint is sprained, there is always some cutaneous tenderness. There is also tenderness over a sprained joint due to capsular injury, bands of adhesions, etc. Tenderness is apt to arise at certain reasonably fixed points: in a hip-joint injury it is found behind the great trochanter, in a knee-joint injury by the side of the patella, in an ankle-joint injury to the inner side of the external malleolus (Culp). When the vertebral articulations are sprained the muscles of the back are rigid, the skin is often sensitive, pain may be awakened by pressure or by certain movements, but there is no sign of cord injury in an uncomplicated case.

**Diagnosis and Prognosis.**—Sprain-fractures can be diagnosticated with certainty only by the *x*-rays. In the *diagnosis* of a sprain, fracture and dislocation must be considered. In fracture, crepitus and mobility exist; in dislocation, rigidity. The diagnosis of sprain should be made by a consideration of the joint involved, of the age, of the nature of the force, of the length of the limb, of the fact that the patient could use the joint for at least a short time after the accident, and of the local feel and movements of the part. In some cases examine under ether, in some apply the *x*-rays. Many injuries about the ankle which we would have formerly regarded as sprains, are shown by the *x*-rays to be fractures. The *prognosis* depends on the size of the joint, on the extent of laceration, on the amount of intra-articular hemorrhage, and on the age of the patient. The danger is ankylosis. A sprained joint is a point of least resistance to tubercle bacilli. In rare cases after a sprain of the hip-joint osteo-arthritis arises. In some few cases after a sprain of the hip the head of the bone undergoes absorption.

**Treatment.**—In a mild sprain apply at once a silicate, rubber plaster, or plaster-of-Paris dressing. The first indication after the infliction of a severe sprain is to arrest hemorrhage and limit inflammation. For the first few hours apply pressure and an ice-bag. Wrap the joint in absorbent cotton wet with iced water, apply a wet gauze bandage, and put on an ice-bag. After some hours place the extremity upon a splint and to the joint apply flannel kept wet with lead-water and laudanum, iced water, tincture of arnica, alcohol and water, or a solution of chlorid of ammonium. These evaporating lotions produce cold. Instead of them an ice-bag may be used for a day. Leeches

around the joint do good. Constitutionally, employ the remedies for inflammation. Morphin or Dover's powder is given for the pain. Judicious bandaging limits the swelling.

After twenty-four hours, if the symptoms continue or if they grow worse, use hot fomentations, the hot-water bag, plunge the extremity frequently in very hot water, or apply heat by Leiter's tubes. When the acute symptoms begin to subside rub stimulating liniments upon the joint once or twice a day and employ firm compression by means of a bandage of flannel or rubber. Frictions should be made from the periphery toward the body. Many cases do well at this stage under the local use of ichthyol and lanolin (50 per cent.), tincture of iodin, or blue ointment. Later in the case use hot and cold douches, massage, frictions, passive motion, and the bandage. Passive motion and massage are begun a day or so after swelling ceases. If they cause the swelling to return, abandon them for several days and then try them again. Blisters are used when tender spots persist and stiffness is manifest. If stiffness becomes marked, move the joint forcibly. Give iodid of potassium and tonics internally, and insist on open-air exercise. If the person is gouty or

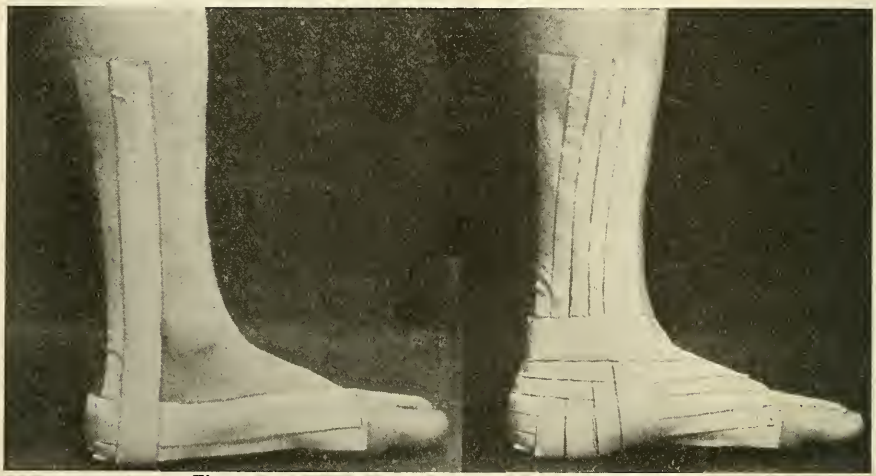


Fig. 393. Fig. 394.

Figs. 393, 394.—Gibney's method of strapping in sprains of the ankle.

rheumatic, use appropriate remedies. Van Arsdale treats sprains by massage almost from the start. Gibney treats them by strapping with adhesive plaster. Gibney's dressing is of great service in a sprain of the ankle (Figs. 393, 394). Many sprains may be put up in an immovable dressing the first day or two after the accident. If the joint contains much blood, aspiration should be practised before the dressing is applied.

The hot-air oven is a very valuable method for treating recent sprains, and the swelling, pain, and stiffness which follow sprains of the extremities. The sprained extremity is placed in an oven, and the part is subjected to heat for an hour. The next day the treatment is repeated, and on as many subsequent days as may be necessary. In an acute sprain the pain often disappears during the first application of heat. In the intervals between the use of the oven the extremity should be at rest, perhaps upon a splint.

**Sprain of the Sacro-iliac Articulation.**—This condition was first described by Goldthwait. A fall, lifting a heavy weight, a blow, or a twist may injure the articulation. Normally there is slight motion, rupture or stretching of ligaments may lead to increased motion, and any considerable

range of motion at the synchondrosis means lack of solidity and want of support. A sprain may arise from long-continued standing, bending, lying, or sitting. A sprain of this articulation may be caused by parturition, and also, as shown by Dunlop, it may develop during anesthesia because of obliteration of the normal lumbar curve by lying on a flat table without a support under the lumbar region ("New York Med. Jour.," July 10, 1909). Dunlop thus explains, and I think truly, the severe backache which is so common after anesthesia.

A sprain causes severe pain, greatly aggravated by standing, by rising up from recumbency, by movements of the ilia which jar the joint, and frequently by direct pressure upon the synchondrosis. There is often lateral spinal curvature due to spasm, and the concavity is toward the injured side.

There is pain in the injured articulation, but there may be a general backache, and, just as in sacro-iliac tuberculosis (see page 624), there may be pain in the sciatic nerve, in the groin, and in the hip-joint.

When some of the sacro-iliac ligaments are ruptured or relaxed we get the chronic condition described by John Dunlop (Ibid.) and which he calls *sacro-iliac relaxation*. When this exists the individual may injure the articulation again and again because of its unsteadiness, he may now and then have trouble, he may be in a constant condition of helplessness, with backache, groinache, pain in the hip and over the ischial tuberosity, lumbar rigidity producing lateral curvature, etc. I believe in the reality of the condition.

**Treatment.**—A recent sprain is treated by rest in bed and adhesive-plaster strapping, reinforced by a canvas roller around the pelvis. In a chronic case (sacro-iliac relaxation), after securing by manipulation normal relations in the articulation, insist on rest and apply a spica bandage of plaster of Paris. If there is marked tendency to displacement, ankylosis may be secured by operation. Fixation by a bone-graft from the tibia should give the most rapid and permanent result (Rugh).

**Rupture of the Crucial Ligaments of the Knee.**—This is a rare injury. Rupture of both ligaments is unusual except in very grave injury, such as complete dislocation, and then other ligaments are also torn or destroyed.

The anterior crucial ligament instead of rupturing from force may cause avulsion of the tibial spine (see page 610). When this portion of bone is not torn off, the ligament itself tears off from the femur rather than from the tibia. The posterior ligament, too, tends to tear off from femur rather than tibia. Pagenstecher ("Deutsch. Med. Wochenschrift," Bd. xxix) believes that the anterior ligament may be ruptured by forced flexion of the knee and by blows applied to the posterior part of the head of the tibia when the knee is flexed. The same surgeon maintains that the posterior ligament may be ruptured by blows applied to the front of the head of the tibia when the knee is flexed. Pringle ("Annals of Surgery," August, 1907) maintains that the anterior ligament may be ruptured by "flexion, abduction, and internal rotation of the leg at the knee." If the ligaments are ruptured there will probably be abnormal freedom of anteroposterior movement between the femur and tibia.

Pringle (Ibid.) states that if, after an injury, the knee-joint becomes distended with blood, the inference should be that one or other crucial ligament has been injured or that the tibial spine has been torn off, unless some other lesion is obviously present; that if internal rotation of the extended leg is possible at the knee, or if the head of the tibia can be brought forward on the femur, or if there is unnatural abduction, the indications are that the anterior crucial ligament has been injured or the tibial spine torn off; that injury of the posterior crucial is suggested by the possibility of pushing the head of the tibia backward during flexion of the knee-joint.

In most cases exploratory incision is required to make the diagnosis.

The **treatment** is to open the joint and suture the torn ligament.

**Ankylosis.**—When a joint-inflammation eventuates in the formation of new tissue in and about the joint, contraction of this tissue limits or destroys joint mobility, producing the condition known as "ankylosis." Ankylosis may be *complete* (bony) or *incomplete* (fibrous); it may arise from contractures in the joint (*true* or *intra-articular ankylosis*) or from contractures in the structures external to the joint (*false* or *extra-articular ankylosis*).

There are qualifying terms to indicate the extent of stiffness—viz., false, spurious, true, bony, ligamentous, partial, complete, or incomplete ankylosis. The significance of the above terms will be better appreciated if ankylosis is considered as meaning a stiff joint. It may be stiff without being rigid. Fibrous adhesions produce stiff joints and they become rigid only when bony union takes place between the bones forming a joint.

*Spondylitis deformans* is bony ankylosis of vertebræ due to osteo-arthritis.

*Arthritis ossificans* is a progressive bony ankylosis in which numerous joints are involved and are finally completely obliterated. It is an ossifying arthritis.

**Etiology.**—There are various causes—viz., traumatism, eruptive fevers resulting in acute or suppurative synovitis or arthritis, gonorrheal arthritis, tuberculous arthritis, syphilitic affections of joints, bony fixation when a fracture is near or extends into a joint, and osteitis deformans. Simple fixation of an uninflamed joint cannot cause true ankylosis.

**Pathology.**—In complete—*i. e.*, bony—ankylosis the bones forming a joint become united by callus in much the same manner as bone-fragments are united after a fracture, or osseous bridging takes place at one or more places around the joint. Osseous ankylosis is preceded by a more or less prolonged stage of fibrous or partial ankylosis. In fibrous ankylosis, bands of fibrous connective tissue unite the bones forming a joint, thereby limiting the motion. In cases of joint stiffness produced by extra-articular fibrous, tendinous, or cicatricial contracture the joint proper remains free from adhesions for years, provided it is not and has not been involved in inflammatory action.

**Diagnosis** of bony ankylosis is usually made without difficulty except where there are several joints near together, as the carpus, tarsus, and the spine. When there are several joints near together the limitation of motion in one joint is generally compensated for by the excess in mobility of another, thereby rendering the associated parts capable of closely approaching normal function. Fibrous ankylosis is more difficult to recognize, especially if pain accompanies manipulative measures. It is most apt to be confused with fibrous, ligamentous, or cicatricial contractures of soft parts outside of a joint, but having more or less direct functional relations therewith.

Extra-articular thickening may usually be detected by the existence of resistance to free joint motion in one direction only, *i. e.*, that produced by the fibrous contracture while the joint moves freely in other directions. Muscular contracture, whether voluntary or involuntary, is but temporary, and is easily detected by the preternatual rigidity of surrounding parts. In bony ankylosis no voluntary muscular action can be detected, inasmuch as in the process of the formation of the callus uniting the bones the muscles have become atrophied from disuse. Conversely, voluntary muscle action about a joint always indicates that joint mobility is not entirely destroyed.

As muscular rigidity is one of the most important and reliable symptoms of joint inflammation and tuberculous invasion, it is a serious error to anesthetize a patient for examination of a joint until the full significance of the muscular action has been carefully studied.

Anesthesia removes the pain and abolishes muscle fixation and thus leaves

the unguarded joint free for manipulative movements, which are generally prejudicial and rarely beneficial. Where no muscle fixation is present, much may be learned by the careful study of a joint while the patient is anesthetized. We thus determine the character of the adhesions, whether they are extra-articular or intra-articular, whether they are fibrous, cicatricial, or osseous, and if bony union exists, whether it involves the entire joint or only a portion of it.

Skiagraphs are invaluable helps in making an accurate diagnosis, especially when stereoscopic plates are made. Definite information can thus be obtained as to the character of the uniting material, its extent, and definite location. Positive information will be given as to the relationship of the bones composing the joint, whether there is luxation, subluxation, flexion, or other abnormal position that may influence decision as to the therapeutic measures to be adopted. It is important to remember that a joint very rarely becomes ankylosed in the position of extension (the elbow may if treated in extension for intra-articular fracture). The almost invariable rule is that flexion is the posture of such joints, and the tendency is toward increase of the flexion until bony ankylosis occurs. In the steady progress of the flexion subluxation is apt to be induced.

**Treatment of Intra-articular Ankylosis.**—It is most important to prevent the occurrence of ankylosis, or in the event of its becoming inevitable, to avoid postures that will render the parts unfit for future usefulness when ankylosed.

The most useful position for a stiff joint is a matter of individual opinion; no definite rules have been accepted. In ankylosis of the elbow the flexed position is more useful in certain occupations than the fully extended arm. In other occupations the extended arm is most useful. In walking when the knee is ankylosed and does not tend toward flexion the extended leg is more useful than the flexed leg, but it is more difficult to manage when sitting.

The hip, when ankylosed at various angles, is made useful by the increased latitude of motion of the other hip and by the compensatory motion of the lumbar spine. To such an extent is the lower spine reciprocal when one or both hips are abnormal that it has been termed the third hip-joint. For general usefulness the best position for an ankylosed hip is 10 to 15 degrees of flexion, 10 to 15 degrees of abduction, and 5 degrees of external rotation. In this position, when supplemented by reciprocal action of the other hip and by the lower spine, a gait very closely approaching normal carriage may be obtained in walking, and the sitting posture may be possible.

Each individual joint has its own peculiar requirement and must become a subject for careful study in determining the most useful posture if ankylosis is to be permanent. The trend of modern surgery is to greatly reduce the time of fixation of a fractured bone in order to avoid joint stiffness and prevent muscular wasting. Early passive motion when judiciously employed does not interfere with the efficient treatment of a fracture, but does lessen the joint stiffness that is often a serious and painful sequel.

*Stereo-arthrolysis* "is that branch of arthroplasty whose object is to loosen stiff joints and produce new joints with mobility, following ankylosis" (R. Tunstall Taylor, "Surgery, Gynecology, and Obstetrics," April, 1912).

*Brisément forcé* or *redressement* are terms applied to the use of such manipulative force as the surgeon can judiciously employ in freeing a joint from fibrous adhesions. It is important to keep constantly in mind the danger of breaking the shaft of a bone or of separating the epiphysis when unguarded leverage of the entire shaft of the bone is used. It is of paramount importance to avoid brisément forcé in all cases where the ankylosis has resulted from tuberculosis. The plan is said to have been suggested by Louvrier, of Paris. It was advocated by Langenbeck. Brisément forcé is only curative when the adhesions

are limited to the synovial membrane. When portions of that membrane have been destroyed or when there are dense fibrous adhesions the ankylosis invariably recurs after manipulative force and often becomes worse than before. In bony ankylosis the method is out of the question.

In applying manipulative force it is not always advantageous to have the patient profoundly anesthetized. If he is profoundly anesthetized we may be tempted to apply too violent force. Severe lacerations of fibrous adhesions produce painful joints which necessitate fixation or rest for several days to permit the reunion of the torn structures. A little gain, care being taken to maintain the motion gained, mobility being gradually increased by short progressive steps, is always better than attempts to do a great deal at once. Of course the patient's co-operation is necessary.

When pain is great, much can be accomplished during primary ether anesthesia or while the patient is under influence of nitrous oxid. Bromid of ethyl and chlorid of ethyl are used by many because of their rapid action and brief effect. As stated elsewhere (see page 1209), I seldom use either of these drugs.

Fixation appliances of any kind are contra-indicated during corrective manipulations, as increased freedom of motion is essential rather than fixation. Voluntary efforts are needed to maintain the joint motion already secured as well as to increase the muscle function controlling the affected joint.

Among the operative procedures applicable to intra-articular ankylosis are excision to obtain a false joint (pseudo-arthrosis), excision to obtain a better position for the usefulness of the limb, breaking the bone after partially cutting it with an osteotome (osteotomy), and breaking the bone without any incision (osteoclasis). These several procedures have special advantages in different joints.

Lexer ("Zeit. f. Chir. Med. Orth.," Oct., 1908, p. 476), after resecting a knee-joint ankylosed at a right angle, transplanted the entire knee-joint apparatus from a freshly amputated limb. Complete union resulted. Muscle training was subsequently practised to improve the weak muscular control. Three and a half years later the result was excellent.

Weglowski ("Zent. f. Chir.," April, 27 1907) resorted to cartilage transplantation in a case of ankylosis of the elbow. After freeing and re-forming the ends of the three bones, two plates of cartilage (one-half thickness of rib cartilage) with perichondrium were taken from the sixth and seventh ribs and placed between the newly formed joint surfaces, the perichondrial surface being turned toward the humeral epiphysis. No special fixation was used. No drainage was employed. Active and passive motion was begun on the tenth day. In a month the patient had 60 degrees of free flexion and extension and full pronation and supination. Death from pleuropneumonia of three days' duration occurred in five weeks. Postmortem showed that the perichondrial surface of the cartilage was united to the humerus, while the opposite side was smooth, even shining; the cartilage was enlarging and passed without definite margin into the surface of the humerus.

Microscopic examination showed newly formed blood-vessels between the cartilage and bone; the cartilage was well preserved in its entire extent, the cells and nuclei staining well.

Huguier and Murphy revive a suggestion fifty years old. They interpose soft tissue between the bone ends after freeing the ends from ankylosing material ("Traitement des Ankyloses par la Résection Orthopedique et L'interposition Musculaire," par Le Dr. Alphonse Huguier). The plan was first suggested by Verneuil in 1860 ("Archives de Medicine," 1860). He cured temporomaxillary ankylosis by the interposition of temporal muscle and fascia. In 1899 Helferich suggested inserting a portion of the vastus internus muscle between the patella and femur, and two years later Cramer reported some

successful cases. Hoffa followed this method. Helfereich, in 1893, mobilized the temporomaxillary joint by the insertion of a flap from the temporal muscle. This was repeated by Lentz, Henle, and others. Mikulicz, in 1895, used a flap from the masseter muscle. Similar procedures were employed by Bilezguski, Hoffa, and Kusnetzow. Rochet and Schnudt with Glück, in 1902, used a skin-flap and had previously employed ivory joints to take the place of the excised structures. In 1901 Murphy operated by Verneuil's plan.

J. B. Murphy ("Jour. Amer. Med. Assoc.," May 20–27, June 3, 1905) reviews the literature of the attempt to produce new joints and says: "In our work we have been able, by the interposition of fascia and muscle, covered with a layer of adipose tissue, to produce normal movable joints with capsules and collagen intra-articular fluid." In cases of synovitis with adhesions he resects the capsule and replaces it by aponeurosis or muscle, and it is desirable, when possible, that the replacing piece contains fat, which, under pressure, will form a hygroma or artificial synovial cavity. In bony ankylosis he operates, separates the bones, removes adjacent bony prominences or processes, frees the soft parts, prevents the bones coming again in contact, and interposes between them tissue which will remain fibrous or will form a hygroma or artificial synovial surface. After wound healing has taken place, passive motion, active motion, and forcible extension are required.

W. S. Baer ("Amer. Jour. Orthop. Surg.," 1907, p. 234) advocates the use of sterilized oil injected into joints to prevent the formation of adhesions and thus increase the arc of motion. It may also be poured into a joint after arthrotomy. After using oil in the manner described, Baer summarizes as follows: "That injection of oil into joints under proper precautions is a harmless procedure; that the joint will apparently tolerate as much as it will hold.

"It is most useful in cases where adhesions have followed some acute infectious process, or in those stiff joints which are classified as arthritis deformans of the infectious type.

"Passive motions are made with less pain where the joint contains oil.

"It plays some part in preventing adhesions, so that the mobility of the joint is increased more rapidly."

W. S. Baer (Ibid., August, 1909) reviews the literature of the operative treatment for mobilizing joints, and advocates the use of chromicized pig's bladder as the most satisfactory material to place between the surfaces of the recently separated bones.

Chlumsky, disappointed with the results of muscle flaps, used non-absorbable materials, zinc, rubber, celluloid, silver, and layers of colloidin, but no permanent mobility was obtained. He then employed absorbable plates of decalcified bone, ivory, and magnesium, with somewhat better results. Baer's method of inserting chromicized pig's bladder followed the unsuccessful employment by him of Cargile membrane. The pig's bladder is tightly stitched by catgut sutures around the recently denuded bone and the wound is closed. A hip-joint two months after this procedure showed a voluntary flexion of 35 degrees; abduction of 15 degrees; adduction of 10 degrees; rotation of 25 degrees. Seven months after operation the mobility was better than that recorded at two months. Similar results are recorded of the knee and elbow.

Baer emphasizes the necessity of having the pig's bladder absorbable in thirty to forty days. It should be pliable enough to be adapted to the contour of the joint. Every raw surface should be absolutely separated by it from that with which it would tend to come in contact.

Thorn, after moblizing the joint, transplants a free flap of fascia to between the bone ends ("Zeitschr. f. Chirurgie." Bd. cviii). Hauer, of Baltimore, has also done this successfully. R. Tunstall Taylor has experimented elaborately in the endeavor to find a liquid and absorbable animal substance which, when

injected by a syringe between the denuded ends of the bones, would immediately solidify and prevent contact of bone ends for six or eight weeks. He finally selected yellow wax 1 part and lanolin from 2 to 6 parts (this melts at from 120° to 135° F.). Taylor has had some excellent results from this method ("Penna. Med. Jour.," Jan., 1913). He cautions us not to operate by any method for ankylosis due to infective arthritis until joint inflammation has been quiescent for a year or more.

**Treatment of Extra-articular Ankylosis.**—The treatment of false ankylosis depends upon the case. Recently contracted muscles or tendons require motion, massage, frictions with stimulating liniments, hot and cold douches, and the use of the hot-air apparatus. Violent breaking up is not satisfactory, neither is tenotomy nor myotomy. Old contractions of tendons require tendon lengthening by tendoplasty or myoplasty. Chronic inflammation of tendon-sheaths with adhesion of tendons requires excision of the sheaths. Whenever possible, excise a cicatrix that causes false ankylosis, and fill the gap with sound cutaneous tissue and fat. When the fixation is due to adhesive synovitis of the capsule, excise the capsule and attached ligaments; "the head and neck of the bone should then be surrounded by an aponeurosis or muscle to prevent the re-forming of adhesions" (John B. Murphy, in "Jour. Amer. Med. Assoc.," May 20–27 and June 3, 1905). Bony deposits are gouged away and tumors are removed. Contractures in cases of paralysis require electricity, passive motions, frictions with stimulating liniments, the hot-air bath, and general treatment. Constant and graduated pressure by means of splints and braces (with ratchet or screws) will, in many instances, restore function. The patient can be taught to alter the pressure frequently, making it as powerful as he is able to bear it.

**Loose Bodies in Joints (Floating Cartilages).**—The knee is the joint affected in 90 per cent. of cases, but the elbow, shoulder, hip, wrist, lower jaw, and ankle may suffer. There may be but one loose body in a joint, there may be two or more, there may be many, or even hundreds. More than one joint may be involved. The condition is commonest in adult men. These bodies may be free or each may have a stalk or pedicle; they may move about and occasionally block the joint, or may lie quietly in a joint-recess or diverticulum. They may be flat or ovoid, smooth or irregular, as small as peas or as large as plums, and may be composed of fibrous tissue, of cartilage, or of bone. There are numerous different modes of origin of these bodies, many being "detached ecchondroses or pieces of hyaline cartilage hanging by narrow pedicles" (Sir J. Bland-Sutton), and they result from enlargement and chondrification of the villi of the synovial membrane.

**Symptoms.**—Some bodies give rise to no symptoms for a long time and others merely cause synovitis. A loose body may produce pain and interfere with joint-function. The joint is weak and a little swollen, and the patient can perhaps feel the body and can even push it into a superficial area of the joint, where it may be felt by the surgeon. From time to time the body may get caught, thus suddenly locking the joint and producing intense and sickening pain, extension and flexion being impossible until the body slips out. It may slip out in a moment, but may not for hours or even for many days. A rather small body seems more apt to cause locking than a very large one, but if a large one does cause locking, it is more difficult to dislodge than is a small one. Locking of a joint by a loose body is followed by inflammation and effusion. If the loose body is dense and large, the *x*-ray may disclose it. Sesamoid bones in the gastrocnemius muscle must not be confused with loose bodies in the knee-joint. In some cases of loose body in the knee the diagnosis is impossible from dislocation of a semilunar cartilage, inflamed semilunar cartilage, and synovitis with proliferation of villi.

**Treatment.**—To relieve locking, employ forced flexion and sudden extension. Cure can be obtained only by operation. Let the patient bring the foreign body to a point where it can be felt by the surgeon, so that he can determine where it lodges. Asepticize the knee with the utmost care. Operate if possible under cocain; if not, give ether. If the body is felt before operating, fix it with a pin. The joint is now opened, explored with the finger, the foreign body extracted, and an exploration made to see that no other bodies are present. If a body has an attachment the pedicle is snipped through by scissors. The wound is sutured in two layers and the leg is placed upon a splint. Asepsis must be most rigid. The operation does not cure the causative process, and these bodies are apt to form again. When the knee is involved, some surgeons saw the patella transversely, open the joint widely, remove all foreign bodies, and seek to cure any causative process.

## Luxations or Dislocations

A dislocation is the persistent separation from each other, partially or completely, of two articular surfaces. A self-reduced dislocation is called a sprain (Douglas Graham). There are three forms of dislocations: (1) traumatic; (2) spontaneous or pathological; (3) congenital.

**1. Traumatic dislocations** are due to injury. They are divided into—*complete* dislocation, in which the two articular surfaces are entirely separated and the ligaments are torn; *incomplete* or *partial* dislocation or *subluxation*, in which the two articular surfaces are not completely separated and the ligaments are rarely lacerated; *simple* dislocation, in which there is no wound leading from the surface to the articulation; *compound* dislocation, in which a wound leads from the surface to the joint; *complicated* dislocation, in which, besides the dislocation, there is a fracture, extensive damage of the soft parts, an opening which makes the case compound, or damage of a nerve or blood-vessel; *primitive* or *primary* dislocation, in which the bones remain as originally displaced; *secondary* dislocation, in which the dislocated bone assumes a new position, for instance, a subglenoid luxation of the humerus is primary, and it may become secondarily a subcoracoid luxation because of muscular contraction or attempts at reduction; *recent* dislocation, in which the displaced bone is not firmly fastened by tissue changes in its new situation, and its old socket is not obliterated; *old* dislocation, in which the displaced bone is firmly fastened by tissue changes in its new habitat, and the old socket is to a great extent obliterated (whether a dislocation is old or new depends on the state of the parts rather than on the time which has elapsed since the accident); *double* dislocation, in which corresponding bones on each side are dislocated; *single* dislocation, in which only one joint is dislocated; *unilateral* dislocation, in which one articulation of one bone is out of place; *bilateral* dislocation, in which symmetrical articulations are dislocated; and *relapsing* or *habitual* dislocation, which recurs frequently from slight force because of relaxed ligaments or lack of complete repair after the ligamentous rupture of a first dislocation.

**2. Spontaneous, Pathological, or Consecutive Dislocations.**—Spontaneous dislocation arises from such very slight force that the cause may not be identified, and it acts on a joint rendered lax by disease. It may arise in the course of chronic synovitis, tuberculous joint-disease, or rheumatoid arthritis. In Charcot's joint a spontaneous dislocation will occur sooner or later. In typhoid fever spontaneous dislocation is not uncommon. The hip-joint is most often the one attacked. *Dislocation of the hip in typhoid fever* follows a severe joint inflammation, is usually upon the dorsum of the ilium, and is frequently not noticed until convalescence has set in. If a typhoid dis-

location is seen early, reduction is easily effected, but if seen late, is impossible. The treatment for irreducible typhoid dislocation is the same as for any other irreducible dislocation. Dislocation may occur in the acute infectious arthritis of scarlatina, pneumonia, etc., from distention of the joint cavity with septic products or exudates. In infantile palsy muscular atrophy may be so great that a shoulder or hip may be easily dislocated.

**3. Congenital Dislocations.**—A congenital dislocation is due to a congenital joint malformation which renders it impossible for the bone to maintain a normal position, or is due to external violence during the period of uterine gestation. Congenital dislocations should not be confounded with dislocations produced during delivery. The hip is the joint most often involved. The shoulder suffers occasionally. Lannelongue maintains that congenital dislocation of the hip is due to atrophy of the muscles and of the acetabulum following spinal-cord disease. Verneuil thinks the dislocation is paralytic. Broca says that in view of the fact that the head of the bone is larger than the cavity in which it belongs, it is useless to attempt reduction by manipulation or extension, but many successful cases by the Lorenz bloodless method prove Broca's condemnation to have been too sweeping. Lorenz and Hoffa have each devised an operation for this condition (see page 711). Congenital dislocation of the shoulder requires incision, possibly excision, or the paring down of the head to fit the glenoid cavity (Phelps).

**Traumatic Dislocations.**—In the succeeding pages the traumatic form of dislocation will be particularly considered.

The causes of traumatic dislocations are divided into *predisposing* and *exciting*.

*Predisposing causes* are: (1) *age;* dislocations are commonest in middle life, the usual lesion of the young being green-stick fracture, and that of the old being fracture; dislocations of the radius are not uncommon in youth; (2) *muscular development*, dislocations being commonest in those with powerful muscle; (3) *sex*, males being more predisposed than females, because of their occupations and muscular strength; (4) *occuption* predisposes as a cause according as it demands the employment of muscular force, as in the carrying of burdens; (5) *nature of the joint*, ball-and-socket joints being more liable to luxation than are ginglymoid joints, because of their wide range of motion; (6) *joint-disease* predisposes by relaxing the ligaments; (7) *situation of the joint*, some joints being more exposed to injury than others.

*Exciting causes* are divided into—(1) external violence and (2) muscular action. *External violence* may be *direct*, as when a blow upon one of the bones forces it directly away from the other; or it may be *indirect*, as when force applied to a distant part of a bone is transmitted to its end and drives the bone out of its socket. *Muscular action* is a cause when sudden and violent muscular contraction occurs during the maintenance of a position of the joint which gives the muscles full sway, and throws the head of the bone against the weakest part of its retaining ligaments.

**Pathological Conditions.**—In a recent complete traumatic dislocation the ligaments are damaged, and may perhaps exhibit extensive laceration, or may show only a buttonhole laceration through which a bone projects. External force produces much laceration and little stretching of the ligaments; muscular action produces little laceration and much stretching of the ligaments. In some cases of dislocation due to external violence the structures about the joint are bruised or otherwise damaged; the old socket is filled with blood, and the bone in its new situation lies in a bloody area. Large vessels and nerves are rarely torn, though they are not unusually compressed.

If a dislocation is not soon reduced, inflammation arises in the old joint cavity and about the displaced bone, and the whole area becomes glued together, first,

by coagulated exudate, and finally by fibrous tissue. After a time, in ball-and-socket joints, the old socket fills with fibrous tissue, contracts, becomes irregular, and may even be obliterated; the head of the dislocated bone is altered in shape, its cartilage is destroyed or converted into fibrous tissue, and the pressure of the head of the bone forms a hollow in its new situation, which hollow becomes surrounded by fibrous tissue or even by bone. A new joint may form, the surrounding tissue becoming a compact capsule, and a bursa forming between the head of the bone and its new socket. In a dislocated hinge-joint the ends of the bone alter greatly in shape and their cartilage is converted into fibrous tissue. In an unreduced dislocation the muscles shorten or lengthen or undergo atrophy or fatty degeneration, as the case may be. An unreduced dislocation of a ball-and-socket joint may give a fairly movable new joint, but an unreduced dislocation of a hinge-joint rarely allows of much motion.

**General Symptoms of Traumatic Dislocation.**—In general, traumatic dislocations are indicated—(1) by *pain* of a sickening, nauseating character; (2) by *rigidity*, voluntary motion being impossible except to a slight extent in the direction of the deformity. (For instance, in dislocation of the inferior maxillary the jaw can be opened a little more, but it cannot be closed.) This rigidity brings about loss of function. When the surgeon attempts to move the joint he finds it very rigid; (3) by *change in the shape of the joint* (as flattening of the shoulder after dislocation of the humerus); (4) by *alteration in the mutual relations of bony prominences about a joint* (as the alteration of the relation between the olecranon and humeral condyles in dislocation of the elbow backward); (5) by feeling the displaced bone in its new situation; (6) by missing the head of the bone from its proper situation; (7) by alteration in the length of the limb (in dislocation of the femur into the thyroid foramen the limb is lengthened, but in dislocation on to the dorsum of the ilium it is shortened); and (8) by alteration in the axis of the bone (in dislocation upon the dorsum of the ilium the axis of the injured thigh would, if prolonged, pass through the lower third of the sound thigh); (9) by seeing the dislocation with a fluoroscope or looking at a skiagraph of it.

**Diagnosis of Traumatic Dislocation.**—A dislocation may be mistaken for a fracture. In dislocation there is rigidity, in fracture there is preternatural mobility; in dislocation there is no true crepitus (there may be tendon- or joint-crepitus), in fracture there usually is crepitus; in dislocation the deformity does not tend to recur after reduction, in fracture it does recur after extension is relaxed. In a sprain the movements of the joint are only limited, not abolished, by the almost complete rigidity encountered in dislocation. The change which a sprain may cause in the shape of a joint is due to effusion or to bleeding; there is no alteration in the relation of the bony prominences to one another; there is no notable alteration in the length of the limb (a slight increase in length may arise from joint-effusion, or the head of the bone may subsequently be absorbed and thus produce shortening after some weeks); there is no alteration in the axis of the bone; the bony head is not felt in a new position, and it is found in its normal place. Always remember that a fracture may exist with a dislocation. In any doubtful case—in fact, in most cases—give ether, for a dislocation should be reduced while the patient is anesthetized (except in dislocation of the jaw, of a finger, of the carpus, etc.). In some cases swelling renders the diagnosis difficult or impossible. Always compare the injured joint with the corresponding joint of the sound side. The *x*-rays constitute an invaluable method of diagnosis.

**Treatment of Traumatic Dislocation.**—*Recent Simple Dislocation.*—Reduce a simple dislocation under ether, as a rule. Try *manipulation*, a procedure which seeks to make the bone retrace its own pathway. If this pro-

cedure fails, employ extension and counterextension. If considerable force is needed, an assistant makes counterextension, and the surgeon fastens to the extremity a clove-hitch, which he ties about his waist, and thus secures powerful extension. Counterextension may be obtained by bands, or, in some instances, by the foot of the surgeon. The *clove-hitch* is used because it will not tighten by traction; a tightening band would lacerate the soft parts (Fig. 395). If great power is needed, compound pulleys may be employed, such as the Jarvis adjuster or some similar appliance, but at the present day pulleys are rarely used (see page 668). If these means fail, cut down upon the bone and restore it to position; operation is much safer than the application of great force. After reducing a dislocation, immobilize the joint for a time, which varies for different joints, and for the first few days combat swelling and inflammation by rest of the part and the use of evaporating lotions or an ice-bag. If there exists a fracture of the dislocated bone, apply splints and then try to reduce by manipulations, grasping the limb and the splints with one hand below and, if possible, the head of the bone with the other hand above the seat of the fracture. Allis believes that a dislocation can be reduced even when a fracture exists. It is possible to pull the dislocated head down to the joint, because a portion of periosteum and possibly tendinous material and muscle still hold the two fragments as a strap might unite two sticks. The head may be forced into place by the fingers while traction is being made. If the fracture is near the joint and the fragments cannot be fixed, try to reduce the dislocation, first striving to press the bone into place. This attempt can be greatly aided by traction upon the lower fragment. In some cases with fracture reduction can be much aided by making a small incision, screwing a gimlet into the head of the bone, and using this tool as a handle. McBurney incises, drills a hole in each bone, inserts hooks into them, and pulls the dislocated bone into position (see Figs. 277, 278). When the dislocation has been reduced, the bone-fragments should be wired or plated together.

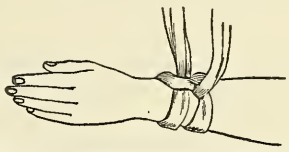


Fig. 395.—Clove-hitch knot applied above the wrist. In dislocation of the shoulder this knot is put above the elbow (after Erichsen).

*Compound Traumatic Dislocation.*—The opening in the soft parts may be due to external violence or to projection of a bone. Compound dislocations are very serious. Hinge-joints are more liable to these injuries than are ball-and-socket joints. Many cases require excision; some, amputation; one that does not demand excision or amputation should be treated by sterilizing the parts, restoring the dislocated bone, making a counteropening, draining, dressing antiseptically, and immobilizing. Considerable ankylosis generally ensues, except sometimes in the small joints. It is scarcely ever necessary to cut away any portion of the protruding bone to effect reduction. If a joint is badly splintered, or if the soft parts are extensively damaged, it may be necessary to excise or amputate; if the main vessels of a limb are seriously injured, amputation must be considered. If the patient is so old or so feeble that it is perilous to force him to combat a long illness, amputation should be performed.

*Old Traumatic Dislocation.*—The problem always presented in an old dislocation is, Shall reduction be tried or shall the bones be let alone? Sir Astley Cooper laid down this rule: "Do not attempt to reduce a shoulder-dislocation after three months, nor a hip-dislocation after two months"; but this rule was put forth before the days of ether. Do not select any fixed period of time to determine what action is advisable. In dislocation of a ball-and-socket joint considerable motion may become possible and a new joint may

form. If movement does not produce pain, a useful new joint may be obtained by the persistent employment of active and passive movements; if movement of the limb does produce pain, enough motion will not be attempted by the patient to produce a useful joint. In the former case it may be best to try to obtain a useful new joint, and in the latter case the surgeon should endeavor to reduce the old dislocation. Always remember that dislocation of a hinge-joint, if left unreduced, will never eventuate in a useful new joint.

In trying to reduce an old dislocation give ether, make movements to break up adhesions, and persist in making these motions until the head of the bone is felt to move; then try at once to reduce by manipulation or extension and counterextension, not waiting for two days, as some suggest. If the head of the bone cannot be made to move, the Dieffenbach plan has been advised, which is to cut the tense restraining bands with a tenotome. Lord Lister, being much impressed with the danger inevitably linked with forcibly dragging old dislocations into place, preferred to cut down and restore the bone, employing, of course, the strictest asepsis, and surgeons in general have adopted this view. In some old dislocations excision of the head of the bone is the proper operation.

**Special Traumatic Dislocations.—Mandible.**—A dislocation of the lower jaw, when there is no fracture, is almost invariably forward. Backward dislocation without fracture is extremely rare, and some have maintained that it cannot occur. Croker King reported a case in 1858. Theim has observed it seven times in five women. The condyle passes under the lower surface of the auditory canal.[1] The common dislocation is forward, and this is the form meant when we simply speak of dislocation of the jaw. There are two forms of forward dislocation—the *unilateral*, which is rare, and the *bilateral*, which is common. Dislocations of the jaw are commonest in women and during middle life. When the mouth is open, contraction of the external pterygoid muscle may pull the condyle over the articular eminence; this contraction may be brought about by yawning, vomiting, scolding, etc. When the mouth is open, dislocation of the lower jaw may be caused by a blow upon the chin; it may also be caused by forcing the mouth more widely open by pushing a bulky body between the teeth.

*Symptoms of Lower-jaw Dislocation.*—In the *bilateral* form the mouth is open and fixed, and it cannot be closed, though it can be opened a little more. The condyles are in front of the articular eminences, and are fixed by the action of the masseters and internal pterygoids, the coronoid processes being wedged against the malar bones. The lower jaw is advanced in front of the upper jaw and the face looks longer than natural. The lips cannot close, the saliva dribbles, swallowing and speech are difficult, there is a depression in front of each ear, the condyles are recognizable in their new abodes, the coronoid processes are detected by a finger in the mouth, and the masseters and temporals stand out in a state of rigidity. Pain may be severe, may be moderate, or may be absent. In the *unilateral* form the chin goes toward the sound side, and the mouth is not so widely open as in the bilateral form, neither is the jaw so fixed. The symptoms are similar to those of a bilateral luxation, but are not so pronounced. The hollow in front of the ear and the abnormal situation of the condyle are detected upon one side only. In an unreduced dislocation the patient may after a time establish some movements of the jaw, but the power of mastication will always be seriously impaired.

*Treatment of Lower-jaw Dislocation.*—In reducing a dislocation of the lower jaw the patient is usually placed with his head against the back of a chair or against the body of an assistant. The surgeon, after wrapping up his thumbs to protect them from being bitten, stands in front of the patient, puts his

[1] Theim, in "Rev. de Chir.," vo. viii, 1888.

thumbs upon the last molar teeth, and grasps the chin with his free fingers. He now presses downward and backward on the jaw, and as soon as the condyle is loosened, closes the jaw over the thumbs by pushing up the chin, using his thumbs as levers. For the last year I have followed the excellent suggestion of W. J. Young ("Brit. Med. Jour.," March 23, 1913), which is to stand behind the patient, with the patient's head against the surgeon's chest, place the right thumb far back in the right side of the patient's mouth and grasp the chin with the left hand. The right hand easily depresses the jaw and the left guides the condyle into place. Then the procedure is reversed and the left side reduced. If reduction by the hands fails, wedges should be put between the molar teeth and the chin should be pushed up either by the hands or by a tourniquet, the band of which surrounds the head and chin. In a unilateral dislocation the wedge should be used only on the injured side. In difficult cases Sir Astley Cooper pushed a round wooden ruler between the molar teeth, used the upper teeth as a fulcrum, and raised the end of the ruler as the handle of a lever. The forceps used by an anesthetist may depress the condyle from its point of fixation, whereupon the chin may be pushed up and back. Nélaton advises that the surgeon place his thumbs in the mouth of the patient and push the coronoid processes backward. After reduction a Barton bandage should be applied and worn for over two weeks. The dressing should be renewed once a day, and passive motion be begun in the second week. The bandage may be discarded at the end of the third week. Liquid diet is advisable for three weeks after the accident. In an old dislocation reduction is always attempted, at least up to a period of six or seven months after the accident. An irreducible dislocation requires osteotomy of the neck of the bone if the part cannot be restored after incision.

**Dislocation of the Clavicle.—Sternal End.**—There are three forms of dislocation of the sternal end of the clavicle, namely: (1) forward; (2) backward, and (3) upward.

**Forward Dislocation of the Sternal End of the Clavicle (Presternal Dislocation).**—The *causes* of forward dislocation of the clavicle are blows, falls, or pulls which drive or draw the shoulder backward.

*Symptoms and Treatment of Forward Dislocation of the Sternal End of the Clavicle.*—The symptoms manifest in dislocation of the clavicle are: prominence in front of the sternum; the acromion is nearer to the sternum on the injured than on the sound side; the clavicular origin of the sternocleidomastoid muscle is rigid; movement is difficult and painful. To reduce a dislocation of the clavicle, pull the shoulders back against the knee of the surgeon, which is placed between the scapulæ. Dress with a posterior figure-of-8 bandage (Fig. 833) or a Velpeau bandage (Fig. 835), the dressing to be worn for three weeks. After removal of the dressing apply a truss, the pad of which is put over the head of the clavicle, and which instrument is to be worn for a month. Dislocation of the clavicle is difficult to keep reduced, but even if it becomes fixed in deformity, the motions of the arm will not be impaired permanently. It can be reduced and fixed by incision and wiring.

**Backward dislocation of the sternal end of the clavicle** is very rare. The *causes* are direct violence and indirect force, such as falls or blows which drive the shoulder forward and inward.

*Symptoms and Treatment of Backward Dislocation of the Sternal End of the Clavicle.*—The symptoms are: pain, loss of function in the arm; inclination of the head toward the injured side; stiffness of the neck; the shoulder passes forward and inward, and often falls downward; a depression exists over the sternoclavicular joint; the head of the clavicle cannot be felt, or is found back of the sternum. The displaced clavicle may press upon the trachea, the esophagus, or the great vessels, inducing dyspnea, dysphagia, obliteration of

pulse in the arm of the injured side, or great venous congestion of the head (see Pick). The usual method of treatment is to pull the shoulders backward and apply a posterior figure-of-8 bandage (Fig. 833), which must be worn for three weeks. If pressure-symptoms are urgent, it is the rule to incise, restore the bone to place and wire it, or resect the displaced head.

**Upward dislocation of the sternal end of the clavicle** is very rare. The *cause* is indirect force, which carries the shoulder downward, inward, and backward (Smith).

*Symptoms and Treatment of Upward Dislocation of the Sternal End of the Clavicle.*—The chief symptom is impaired function of the arm; the shoulder passes downward and inward, the clavicular axis is altered, and the displaced head is felt. Dyspnea may or may not exist. To treat this dislocation, put a pad in the axilla and press the elbow to the side (in order to throw the bone outward), and try to push the head into place. Apply a Desault bandage (Fig. 836) and place a firm pad over the sternoclavicular joint. The deformity is apt to recur, but a useful limb will nevertheless be obtained. The best method of treatment is to wire the bones in place.

**Dislocation of the acromial end of the clavicle** is almost always upward, but it may be below the acromion. The *cause* is violent force, which, if so

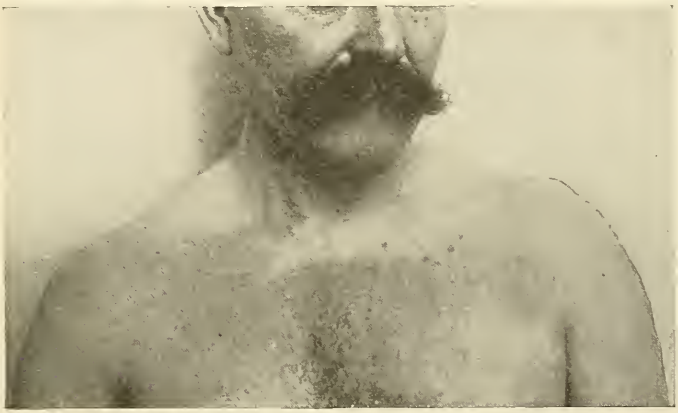


Fig. 396.—Dislocation upward of acromial end of clavicle.

applied to the scapula as to drive the shoulder forward, may produce a dislocation upward. A dislocation downward is due to blows upon the upper surface of the outer end of the clavicle.

*Symptoms and Treatment.*—In dislocation of the acromial end of the clavicle upward there are noted: prominence of the clavicle upon the top of the acromion; impaired function of the arm (it cannot be lifted over the head); the shoulder falls downward and passes inward; there is apparent lengthening of the arm; the head is bent toward the injured side, and the clavicular origin of the trapezius is strongly outlined (Pick). In *dislocation downward* both the acromion and the coracoid are very prominent, the clavicular axis is altered, and there is depression over the sternoclavicular joint. The surgeon usually endeavors to reduce a dislocation upward by placing the patient supine on a hard table, pulling the shoulder back, and pushing the bone into place. After reduction the old method of treatment was to apply a Desault bandage, which was kept on for three weeks, and decided deformity, enduring pain, and disability were looked for as inevitable. Stimson used to apply dressings of adhesive plaster. The author has seen several cases treated by the apparatus of Thomas Leidy Rhoads. The apparatus completely corrected the deformity, and the patients made a most satisfactory recovery.

The essential element of Rhoads's apparatus is a trunk-strap applied after reduction of the dislocation, as shown in Figs. 397, 398. If the deformity can be completely corrected, Rhoads's apparatus will serve a good purpose, but in many cases it is impossible really to reduce the deformity or after apparent

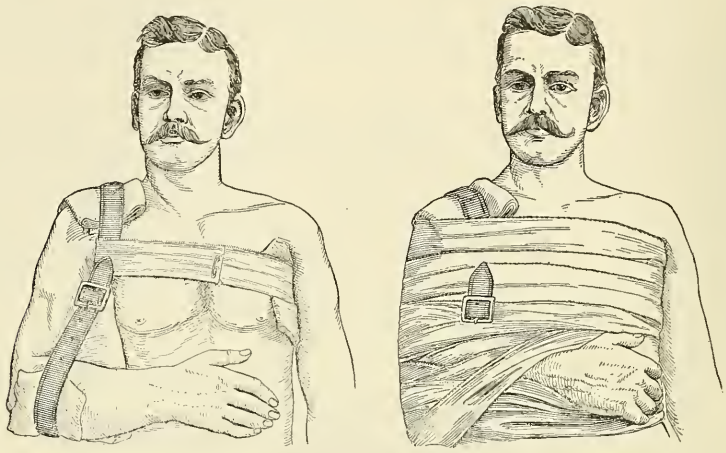


Fig. 397. Fig. 398.

Figs. 397, 398.—Rhoads's apparatus for treating dislocation upward of the acromial end of the clavicle.

reduction the deformity at once returns. This is due, as Moore[1] points out, to the fact that the superior acromioclavicular ligament is torn from the clavicle, but remains attached to the scapula, and when reduction is attempted, is pushed under the clavicle and nothing remains to hold the clavicle "in place but the skin and superficial fascia." I agree with Moore that the best treatment is incision, replacement, and suturing the acromion to the outer end of the clavicle. The bones are sutured with silver wire or kangaroo tendon, the acromioclavicular ligament is sutured with catgut, the wound is closed with sutures of silkworm-gut, and the patient is kept supine in bed for three weeks. I have operated successfully on 5 of these cases.

Fig. 399.—Subcoracoid dislocation of shoulder.

Dislocation downward is reduced and treated in the same manner as dislocation upward.

*Simultaneous dislocation of both ends of the clavicle* is a very rare injury. It is treated as is single dislocation.

The so-called **dislocation of the lower angle of the scapula** is not, as was long taught, a dislocation at all. The lower angle and vertebral border deviate from

[1] "Annals of Surgery," May, 1902.

the chest. This condition was thought to be due to the bone slipping from under the latissimus dorsi muscle, but it is now known to be due to *paralysis* of the *serratus magnus muscle*, the bone being acted upon by the trapezius, pectoralis minor, levator anguli scapulæ, the rhomboid muscles. Examination shows that the scapula will not rotate normally forward. This is demonstrated by extending the arms in front to a right angle, the gliding forward of the scapula upon the sound side being marked, but upon the diseased side being slight or absent.

*Treatment* of paralysis of the serratus magnus muscle comprises massage, electricity, passive motion, and deep injections of strychnin.

Katzenstein advocates operation for serratus palsy. He makes an incision near to the midline of the back, exposes portions of origin of the trapezius and rhomboideus major, divides them, carries the cut muscles downward and outward, and sutures them to the periosteum of the seventh, eighth, and ninth ribs and to the latissimus dorsi. He then makes an "incision along the inner surface

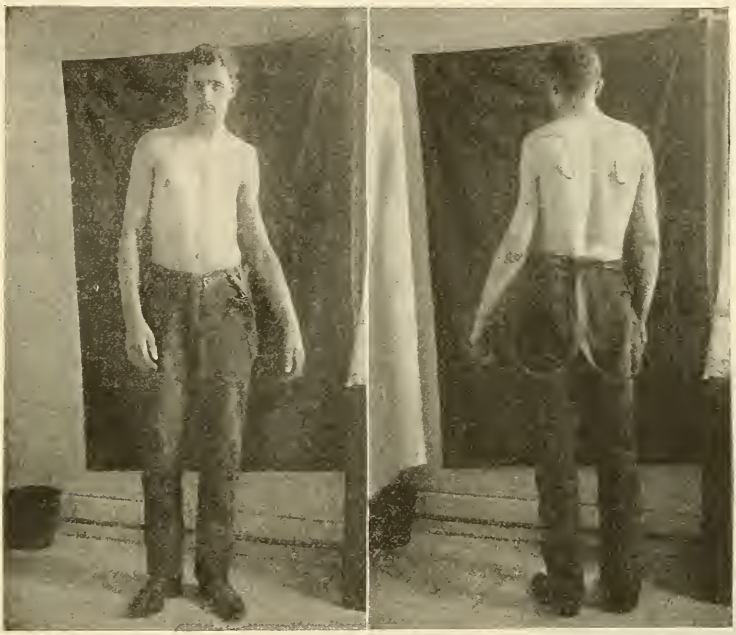


Fig. 400.—Subcoracoid dislocation of the left humerus (St. Joseph's Hospital case; photographed by Dr. Nassau).

of the arm from the middle up through the axilla to end on the thoracic wall." He divides the humeral insertion of the great pectoral and sutures its tendon to the axillary border and the anterior scapular muscles (Binnie's "Operative Surgery").

**Dislocation of the Humerus (Shoulder-joint).**—This injury is quite frequent because of the free mobility of the shoulder-joint, its anatomical insecurity, and its exposed situation; it rarely occurs in the very young and in the aged, and is oftenest encountered in muscular young adults. Shoulder dislocation is produced by throwing the arm into abduction. In this position the head of the humerus presses against the lower and front part, that is, against the thinnest and most poorly supported portion of the capsule. In almost all cases the tear in the capsule occurs between the tendon of the subscapularis and the triceps. Hence, most dislocations are *primarily* subglenoid, although the bone usually moves to some other position, being dragged or

driven there by the injuring force or being pulled there by muscular action. Dislocation forward is much more common than dislocation backward because the long head of the triceps keeps the head of the bone from going posterior and because the anterior are stronger than the posterior muscles. Four chief forms of shoulder-joint dislocation exist, namely: (1) forward, inward, and downward, under the coracoid process—subcoracoid; (2) downward, forward, and inward, beneath the glenoid cavity—subglenoid; (3) backward, inward, and downward, under the spine of the scapula—subspinous; and (4) forward, inward, and upward, under the clavicle—subclavicular.

A very rare form of shoulder-joint dislocation has been described, which is known as the *supracoracoid*. Another rare form is the *luxatio erecta*.

*Subcoracoid Luxation* (Figs. 399, 400).—The subcoracoid variety of dislocation embraces three-fourths of all shoulder-joint luxations. It may be caused by direct force driving the head of the humerus forward and inward, or by indirect force, such as falls upon the hand or the elbow. In this dislocation the head of the bone lies against the anterior surface of the scapular neck below the coracoid process. A part of the anatomical neck of the humerus lies upon the anterior margin of the glenoid cavity, and the head of the bone is above the tendon of the subscapularis muscle.

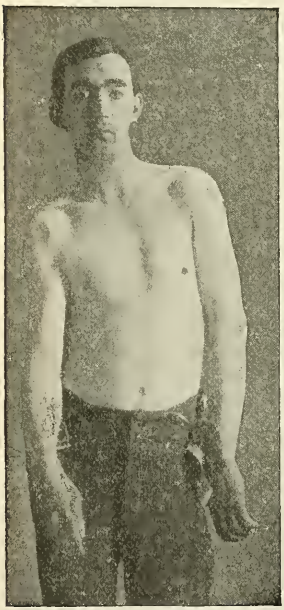


Fig. 401.—Axillary dislocation of the right humerus.

*Subclavicular luxation* is very rare. It is caused by the same sort of violence which produces subcoracoid luxation. The head of the bone rests upon the thorax, below the clavicle, and underneath the pectoralis major muscle.

*Subglenoid or Axillary Luxation* (Fig. 401).—It may be produced by contraction of the great pectoral and latissimus dorsi muscles when the arm is at a right angle to the body, but it is usually due to falls upon the hand or the elbow when the arm is raised and the head of the bone is against the lower portion of the capsule. In this dislocation the head of the bone rests upon the border of the scapula, below the tendon of the subscapularis, in front of the long head of the triceps, and above the teres muscles. Most dislocations of the shoulder are primarily subglenoid, the position perhaps being subsequently altered by muscular action.

*Subspinous luxation* is a rare injury. Pick met with this accident in a man who, while having his hands in his pockets, fell upon the front of the point of the shoulder. The head of the bone reposes beneath the scapular spine, between the infraspinatus and teres minor muscles.

*Supracoracoid luxation* is seldom encountered. The head of the humerus rests upon the coraco-acromial ligament or upon the acromion process, and the acromion or the coracoid is always fractured.

*Luxatio Erecta.*—In this injury the arm is markedly abducted and in some cases the elbow is actually raised above the patient's head. As a rule, the forearm rests behind the occiput, sometimes on the top of the head. The patient holds the forearm to the occiput or vertex to avoid pain. It is, in reality, a form of subglenoid luxation. In such an injury the head of the bone has passed under the subscapularis muscle and also under the teres major or the lower border of the great pectoral. Judd, Hulke, Cleland, and others have reported cases.

*Symptoms of Dislocation of the Shoulder-joint.*—Dislocation is diagnosticated by—(1) pain of a sickening character; (2) flattening of the shoulder, the head of the bone having ceased to bulge out the deltoid muscle; (3) apparent projection of the acromion through sinking in of the deltoid; (4) hollow beneath the acromion, over the empty glenoid cavity, and the bone missing from its normal habitat. This hollow may be easily appreciated by the finger, especially when the extremity is somewhat abducted; (5) rigidity (some movement is possible in the direction especially of an existing deformity, but mobility is strictly limited and attempts at motion produce great pain); (6) Dugas's sign: the elbow cannot touch the side when the hand is placed upon the sound shoulder, and the hand cannot be placed upon the sound shoulder if the elbow is to the side (this is due to the rotundity of the chest. In a dislocation the head of the bone is already touching the chest, and the bone, being approximately straight, cannot touch it in two places at the same time. If the elbow can be placed against the chest with the hand on the sound shoulder there cannot be dislocation; if it cannot be so placed, there must be dislocation); (7) finding the head of the bone in a new situation; (8) examining by means of the *x*-rays. Symptoms 1 to 5 inclusive may be grouped as Erichsen's list of signs. The form of dislocation is made out by a study of the direction of the axis of the limb, the existence and extent of lengthening or of shortening, and the situation of the head of the bone.

In a shoulder-joint dislocation the head of the bone may press upon the brachial plexus and produce pain and numbness, and occasionally traumatic neuritis or paralysis; sometimes pressure upon the axillary vein causes intense edema, and pressure upon the axillary artery diminishes or obliterates the pulse. The axillary vessels may be torn and the muscles may be lacerated badly. The capsule is torn and considerable blood is usually effused. Swelling is due first to hemorrhage, and secondly to inflammation. Partial dislocation sometimes, though rarely, occurs. What is usually spoken of as "partial dislocation" or "subluxation" is a condition in which the head of the humerus passes forward under the coracoid because of rupture of the long head of the biceps or because this tendon slips out of its groove, the ligaments of the shoulder-joint being intact.

The following table from T. Pickering Pick's work on "Fractures and Dislocations" makes the above points clear:

| | Direction of the Axis of the Limb. | Alteration in the Length of the Limb. | Presence of the Head of the Bone in New Situation. |
|---|---|---|---|
| Subcoracoid. | The elbow is carried backward and slightly away from the side. | Very slight lengthening. | The head of the bone cannot easily be felt; it is found at the upper and inner part of the axilla. |
| Subglenoid. | The elbow is carried away from the trunk and slightly backward. | Very considerable lengthening. | The head of the bone can easily be felt in the axilla. |
| Subspinous. | The elbow is raised from the side and carried forward. | Lengthening intermediate in degree between the subglenoid and the subcoracoid. | The head of the bone can be felt and be grasped beneath the spine of the scapula. |
| Subclavicular. | The elbow is carried outward and backward. | Shortening. | The head of the bone can readily be seen and be felt beneath the clavicle. |

*Diagnosis of Shoulder-joint Dislocation.*—In *fracture of the neck of the scapula* the acromion is prominent, a hollow is detected below it, and a hard

body is felt in the axilla; but the coracoid process descends with the head of the humerus, which it does not do in dislocation. Furthermore, in fracture there is mobility; in dislocation, rigidity. In fracture crepitus is present; in dislocation it is absent. In fracture the deformity is easily reduced, but it at once recurs; in dislocation the deformity is with difficulty reduced, but does not recur. In fracture the elbow can be made to touch the side when the hand is upon the sound shoulder; in dislocation it cannot be so manipulated. In *fracture* of the *anatomical neck of the humerus* deformity is slight; the head of the humerus is found in place, does not move when the shaft is rotated, and is not in line with the axis of the bone. Crepitus exists in the fracture if impaction is absent. In *paralysis of the deltoid muscle* there is distinct flattening, but the bone is felt in place and there is no rigidity. The *x*-rays are invaluable in diagnosis.

*Treatment of Shoulder-joint Dislocation.*—Reduction by manipulation is usually readily accomplished in a recent case of shoulder-joint dislocation. If a simple trial without ether fails, an anesthetic should be administered. Ether is given, but not chloroform, for chloroform seems to be particularly dangerous to life when given to enable the surgeon to reduce a dislocation of the shoulder. *Forward dislocations* (subcoracoid, subclavicular, and axillary) are reduced by *Kocher's method* (Fig. 402). This method was introduced by Kocher in 1870 ("Sammlung klin. Vorträge," No. 83). Reduction by this

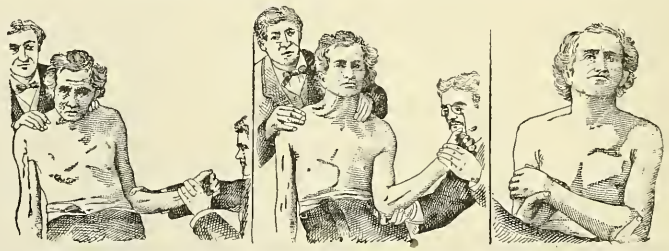


Fig. 402.—Kocher's method of reduction by manipulation: *a*, First movement, outward rotation; *b*, second movement, elevation of elbow; *c*, third movement, inward rotation and lowering of the elbow (Ceppi).

method can frequently be effected without the aid of ether. The patient should be recumbent. Slowly but forcible adduct the abducted elbow and get it finally against the side. At the same time draw it slightly backward. The forearm is flexed. If there is much muscular resistance, follow Keetley's advice, and not only bring the elbow to the side, but push it backward and inward toward the spine. Grasp the elbow with one hand, and the wrist with the other, and slowly make external rotation until the forearm points outward from the body. We thus carry the head of the humerus to the margin of the glenoid cavity. External rotation must be done slowly and gently. When we first try it there is much muscular resistance. If enough force is used to overcome the resistance the surgical neck of the bone may be broken. By gently and gradually persisting in external rotation the muscles are finally tired out and relax. Next lift the elbow anteriorly as far forward as it will go, so as to bring the head of the humerus to the glenoid margin just opposite the capsular tear (Keetley). Then throw the bone into place by gradually swinging the forearm inward across to the other side of the chest, that is, by internal rotation. The formula is, flexion of the forearm, external rotation, lifting the elbow forward, internal rotation of the arm, and lowering the elbow. The motions to unlock the bone and start it to retrace the steps it took when emerging should be gentle, not forcible, slow, not sudden, and rigid muscles should be tired out and made to relax by steady traction upon them. Sudden

and violent motions increase rigidity. Adduction stretches the upper portion of the capsule and presses the head against the glenoid. External rotation opens the tear in the capsule. Elevation relaxes the untorn part of the capsule and coracohumeral ligament and stretches the torn portion. On this fulcrum the head, which is the end of a lever, is forced into place. If in trying Kocher's plan external rotation of the humerus does not take place, abandon the method, as persistence will fracture the humerus. Another method of manipulation is as follows: if the *right* shoulder is dislocated, the surgeon stands behind the patient (who is sitting erect); if the *left* shoulder is dislocated, he stands in front of the patient. The surgeon holds the forearm flexed upon the arm with his right hand and makes external traction and rotation, and with the fingers of his left hand he tries to force the bone into place.

In *Henry H. Smith's method* for forward dislocation the surgeon stands in front of the patient. If the *left* shoulder is dislocated, the surgeon grasps it with his left hand; if the *right* shoulder is dislocated, he grasps it with his right hand, the thumb resting on the head of the bone. With his disengaged hand the surgeon grasps the elbow, abducts it, makes traction and external rotation, and suddenly sweeps the elbow inward, aiming it at the sternum, and tries with his thumb to push the bone into place. In *subspinous luxations* reduction may be effected if the surgeon stands behind the patient, makes abduction, traction, and internal rotation, sweeps the elbow inward toward the spine, and with the thumb aids the bone in its return into position. Raising the elbow far above the head and sweeping it inward will reduce some dislocations. As the head of the bone slips back a distinct jar is felt and a snap is heard, the motions of the joint are again obtainable, and with the hand on the opposite shoulder the elbow may be made to touch the side.

*Reduction by Extension.*—Before attempting the reduction of a dislocation of the shoulder-joint by extension the patient should be anesthetized and placed upon a low bed or upon the floor. The surgeon then places his foot, covered only by a stocking, in the axilla. Place the sole of the foot, not the heel, against the chest high up, the instep being made to touch the humerus and the heel the border of the shoulder-blade, a towel being first put into the axilla to rest the foot against (Fig. 403). If the left arm is dislocated, use the left foot, and vice versâ. The elder Gross approved of making extension while sitting between the patient's limbs. Make steady extension, which will in many cases bring about the reduction. If it fails to cause reduction, bring the patient's arm across the chest and use the foot as the fulcrum of a lever. If the humerus is pretty firmly fixed in its abnormal position, make counterextension with a foot in the axilla and make extension by fixing a clove-hitch (see Fig. 395) *above the elbow* and fastening to it bands which go over one shoulder and under the other shoulder of the surgeon. The back may thus be used for extension, the hands being left free for manipulation (Allis's and Pick's plan). Lateral extension is used by some surgeons. The patient lies down, a large piece of canvas is split, the arm is passed through the split, and the body is thus fixed. The arm is pulled to a right angle with the body and traction is applied.

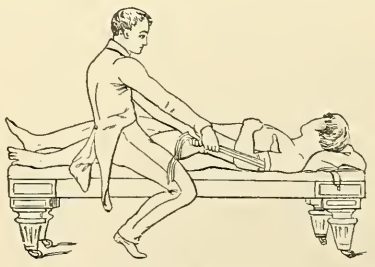


Fig. 403.—Reduction of shoulder-joint dislocation by the foot in the axilla (Cooper).

The late Prof. Joseph Pancoast favored *Sir Astley Cooper's method* of placing the unanesthetized patient in a chair and using the knee as a fulcrum,

pushing the elbow to the side (Fig. 404). Brunus, in the thirteenth century, devised the method of *upward extension.* In applying this method the surgeon takes his place behind the patient, steadies the scapula with his hand, and carries the patient's arm upward and backward above his head, making extension and external rotation (Fig. 405). *La Mothe's method* is applied with the patient supine upon the floor. The surgeon places his foot upon the shoulder to make counterextension, and makes extension as in Brunus's method. It is a useful expedient, when either of these plans is applied, to have an assistant make the traction while the surgeon manipulates the head of the bone. Cock advises, when reduction fails, that an air-pad be placed in the axilla and the arm be bound to the side—a method by which reduction will sometimes take place after two or three days.

Fig. 404.—Reduction of shoulder-joint dislocation by the knee in the axilla (Cooper).

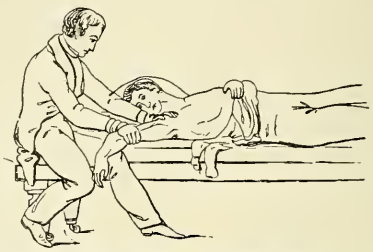


Fig. 405.—Reduction of shoulder-joint dislocation by upward extension (Cooper).

Pulleys should not be used to pull the bone into place, as they develop a dangerous force. In a dislocation irreducible by ordinary force, antiseptic incision is safer and better than the pulleys. After incision try to restore the bone to place.

In reducing a dislocation the axillary artery or vein may be ruptured, fracture of the neck of the humerus may take place, injury to the brachial plexus may occur, or the soft parts may be badly damaged. After reducing a dislocation apply a Velpeau bandage, keep the shoulder immobile for one week, then make passive motion daily, reapplying the dressing after each séance. The patient may wear a sling alone during the third week, after which period he may use the arm. (For Compound Dislocations, see page 658.)

**Unreduced and Irreducible Dislocations of the Shoulder.**—In some cases where we find there is considerable movement without pain we can, by manipulation and active motion, increase the range of movement and the usefulness of the new joint.

As a rule, in a youth or a middle-aged person we attempt bloodless reduction if the head of the bone is movable and there is no prospect of a useful new joint. Give ether, break up adhesions by forced flexion and extension, and try Kocher's method, and, if this fails, the other methods, but never use violent force. In reducing an old dislocation we may fracture the surgical neck of the humerus. I have seen this happen twice. The proper treatment is incision and pulling the head into place by McBurney's hooks. In attempting reduction of an old dislocation by force the brachial plexus may be lacerated or one or both of the axillary vessels may be torn. If an axillary vessel is torn, it must be at once exposed by incision. A large tear in either vessel requires a ligature about the vessel on each side of the tear. A small tear may be sutured (Keetley, in "Lancet," Jan. 23, 1904). Rather than use sufficient force to endanger the vessels in attempting to reduce an old dislocation, practice

incision. In some cases after incision the head of the bone can be pulled and pushed into place. In other cases the head must be resected. After reduction of an old dislocation immobilize for three weeks, and begin passive motion after seven days.

If a *dislocation* is *complicated* by a *fracture of the humerus*, try to pull the head of the bone opposite the joint. This may be possible if the two fragments are held partly together by a fair amount of periosteum and muscle. Traction is exerted upon the arm, and an attempt is made to manipulate the head into the socket (Allis's plan in the hip). McBurney incises, fixes a hook in the scapula and a hook in the head of the humerus, pulls the head into place, and wires the fragments (see Figs. 277, 278). In an emergency gimlets may be used instead of the hooks. In some cases it is necessary to excise the head of the bone.

**Habitual or Recurrent Dislocation.**—Habitual or recurrent dislocation of the shoulder, following an original traumatic dislocation, results usually from a slight or trivial force. It is apt to take place when the arm is in abduction, slight rotation frequently being necessary. In some cases rotation will produce a dislocation while the arm is near the side of the body. Little is known of the frequency with which these cases occur, but they are probably much more frequent than is generally supposed. The frequency of the recurrences in the individual cases varies widely. In some they occur more or less regularly every two or three years, while in others they have been known to take place daily and even several times a day. In most cases in the intervals between the recurrences the joint functionates normally without difficulty, although the patient fears abduction because of its influence in favoring a recurrence. In rare cases pain persists a long time after each dislocation, so that if the recurrences are frequent, the patient may be compelled to give up work.

*Cause.*—The essential cause is a relaxation of the capsule at the site of the original tear, produced by the addition to the old or original portion of capsule of a new or cicatricial portion bridging over the gap between the margins of the tear produced by the first dislocation. The failure of these margins to unite closely is due to the repeated emergence of the humeral head forcing them apart before union is complete. The defects in the head of the humerus which have been found at autopsy and operation have, probably, only a slight and secondary causal importance, while the fractures of the greater tuberosity of the humerus sometimes occurring in dislocations of the shoulder are probably not followed by recurrent dislocations.

*Treatment.*—Excision of the head of the humerus has been abandoned in these cases. Capsulorrhaphy for the shortening of the relaxed anterior portion of the capsule has given excellent functional results. Dawbarn, of New York, did this operation in my clinic, upon a city fireman, and the result was a perfect success. The capsule may be exposed through the usual resection incision along the anterior margin of the deltoid. This may be modified by an additional incision outward at right angles to the first, and the insertion of the pectorlais major may be partially divided. T. Turner Thomas makes an axillary incision along the inner border of the coracobrachialis, passing between this muscle and the axillary vessels and nerves, and avoiding particularly the circumflex and musculocutaneous nerves. The subscapularis muscle is partially divided to give a freer exposure of the capsule. This route exposes, by a small incision, the site of the original tear in the capsule. It avoids division of the deltoid and gives dependent drainage if drainage is necessary.

The relaxed portion of the capsule may be shortened by taking up a reef with catgut or silk sutures, without opening and exploring the joint for loose pieces of bone; the capsule may be incised transversely to its longitudinal

fibers and the margins of the incision overlapped; an oval piece may be excised and the edges united by sutures; or the margins of the original tear may be found and sutured together.

**Dislocation of the elbow-joint** is not infrequent, and is commonest in children. Both bones or only one bone of the forearm may be dislocated, and the dislocation may be partial or complete.

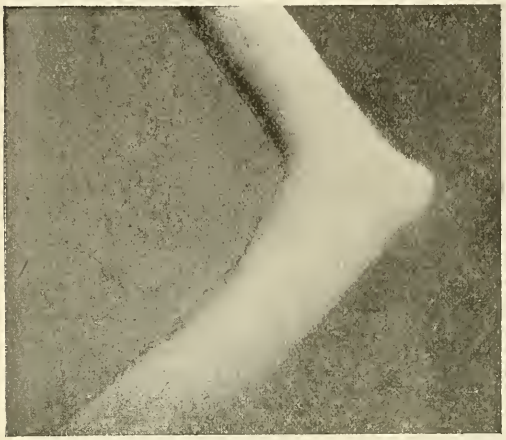


Fig. 406.—Dislocation of both bones of the forearm backward.

**Dislocation of Both Bones Backward** (Fig. 406).—The *causes* of backward dislocation of both bones of the forearm are falls upon the extended hand or twists inward of the ulna (Malgaigne). The coronoid process lodges in the olecranon fossa of the humerus.

*Symptoms of Backward Dislocation.* — In complete dislocation of both bones of the forearm the olecranon is very prominent. The distance between the point of the olecranon and the apex of the inner condyle is notably greater than on the sound side; the forearm is flexed, supinated, and shortened; the lower end of the humerus projects in front of the joint, below the skin-crease; the head of the radius is found back of the outer condyle; and there are the general symptoms of dislocation. Fracture of the coronoid rarely occurs with backward dislocation, but if it does occur, there will be crepitus and mobility. Fracture at the base of the condyles is distinguished from dislocation of both bones of the forearm backward by the following points: in fracture there are found the ordinary symptoms; measurement from the condyles to the styloid processes does not show shortening; there is no alteration of the normal relation between the olecranon process and the condyles; and the projection in front of the joint is above the crease of the bend of the elbow.

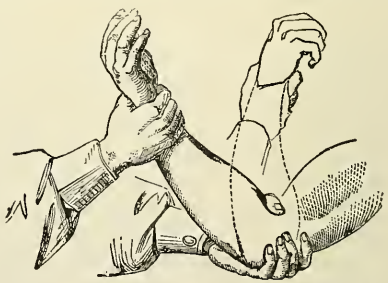


Fig. 407.—Reduction of elbow-joint dislocation.

*Treatment of Backward Dislocation.*—Reduction must be effected early in dislocation of both bones of the forearm, because it will soon become impossible, and an unreduced dislocation means a limb without the powers of flexion, pronation, and supination. The surgeon may place his knee in front of the elbow-joint, grasp the patient's wrist, press upon the radius and ulna with his knee, and bend the forearm with considerable force, the muscle pulling the bones into place (Sir Astley Cooper's plan). Forced flexion, traction, and extension may be tried (Fig. 407). Put the arm in Jones's position for two weeks, and make passive motion daily after the first few days.

**Dislocation of Both Bones Forward.**—The *cause* of forward dislocation of both bones of the forearm is a blow on the olecranon when the arm is flexed. It is an unusual accident.

*Symptoms and Treatment.*—The *symptoms* of forward dislocation of both bones of the forearm are: the forearm is flexed and lengthened; some slight motion is possible; the olecranon is on a level with the condyles if unfractured, hence its prominence is gone; the humeral condyles are felt posteriorly, and the radius and ulna are felt anteriorly. The *treatment* of this injury consists in early reduction, which is accomplished by means of forced flexion, extension, and pressure, placing the part in Jones's position for two weeks, and making passive motion daily after the first few days.

**Lateral dislocation of both bones of the forearm** is usually incomplete.

*Symptoms and Treatment of Outward Dislocation.*—The *symptoms* of outward dislocation of both bones of the forearm are: the forearm is flexed, fixed, and pronated; the joint is widened; the head of the radius projects externally and has a depression above it; the inner condyle projects internally and has a depression below it; the olecranon is nearer than normal to the external condyle and further than normal from the internal condyle. Reduction is effected by extension of the forearm and pressure inward upon the head of the radius. Apply an ascending spiral reversed bandage of the forearm, a figure-of-8 bandage of the elbow-joint, and a sling. Make passive motion after a few days. The bandages must be worn for two weeks.

Fig. 408.—Forward dislocation of the radius.

*Symptoms and Treatment of Inward Dislocation.*—In dislocation inward of both bones of the forearm the position of the forearm is the same as that in dislocation outward; the sigmoid cavity of the ulna projects internally, and the external condyle projects externally. Reduction is effected by extension of the forearm and pressure outward on the ulna, subsequent treatment being the same as that employed in the preceding form.

**Dislocation of the ulna alone** is very rare, and can take place only backward.

*Symptoms and Treatment.*—Dislocation of the ulna alone is indicated by the forearm being flexed and pronated. The head of the radius is found in place, and the olecranon projects posteriorly. The *treatment* of this injury is the same as that for dislocation of both bones.

**Dislocation of the radius forward** (Fig. 408) is the commonest form of dislocation of the elbow. This injury is caused by a fall upon the hand with the forearm in pronation and extension, or is produced by blows on the back of the joint; forced pronation alone will not cause it.

*Symptoms and Treatment.*—The *symptoms* in dislocation of the radius forward are: the forearm is midway between pronation and supination, and is semiflexed; attempts to increase flexion cause the radius to strike against the humerus with a distinct blow; the head of the radius is felt in front of the outer condyle and is missed from its proper abode. Reduction is effected by flexion over the knee, extension, and manipulation. The subsequent treat-

ment is Jones's position and passive motion. Deformity is apt to recur after reduction because of rupture of the orbicular ligament. If permanent dislocation exists, resection of the head of the radius is necessary. Passive motion should be begun in two weeks after the resection. The results as to function and strength are usually excellent.

**Dislocation of the radius backward** (Fig. 409) is caused by falls on the hand or by blows on the front of the joint.

*Symptoms and Treatment.*—Backward dislocation of the radius is indicated by the forearm being slightly flexed and fixed in pronation, by some impairment of flexion and extension, and by the head of the radius being felt behind the outer condyle. Reduction is effected by flexion over the knee, extension, and manipulation, and the subsequent treatment is the same as that given for the preceding dislocation.

Fig. 409.—Dislocation of the radius backward.

**Dislocation of the radius outward** is very rare. In this injury the head of the radius is distinctly felt. Reduction is effected by extension and pressure; the subsequent treatment is the same as that for the above-mentioned dislocations.

**Subluxation of the Head of the Radius.** — This name is given to an injury which is very frequent in children between two and four years of age. It results from traction upon the hand or the forearm, and often arises when the nurse or the mother pulls upon a child's arm to save it from a fall or to lift it over a gutter. Some writers hold that pronation as well as extension is required to produce the injury; many surgeons claim that extension and adduction are the causative forces. Hutchinson asserts that supination may cause subluxation. Bardenheuer assigned falls as causes.

The *symptoms* are very characteristic. The history points to the injury. Pain and perhaps a click may be felt about the elbow, and pain and a click may also be felt in the wrist at the time of the accident. The arm hangs by the side, with the elbow-joint slightly flexed and the forearm midway between pronation and supination. Flexion to an angle of less than 60 degrees and complete extension are resisted and are very painful, but movements between 60 and 130 degrees are free and painless.[1] The movements of the wrist-joint are free and painless. The elbow-joint presents no deformity. Pressure over the head of the radius causes pain. Strong pronation is painful; strong

[1] See the instructive article by W. W. Van Arsdale, in "Annals of Surgery," vol. ix, 1889.

supination is very painful, and there seems to be a mechanical obstacle to its performance. Forced supination develops a distinct click at the head of the radius, and causes pronation and supination to become natural and free from pain. The condition will be reproduced if the parts are not immobilized for a time. The nature of the lesion is not understood, and various conditions have been thought to exist by different observers. Among them may be mentioned the following: a slight anterior displacement of a head of the radius; a slight posterior displacement; locking of the tuberosity of the radius behind the inner edge of the ulna; dislocation of the triangular cartilage of the wrist; intracapsular fracture of the radial head; painful paralysis from nerve-injury; displacement by elongation, the return of the bone being prevented by collapse of the capsule; and the slipping up of the margin of the orbicular ligament over the rim of the head of the radius.

*Treatment.*—In order to reduce, place the forearm at a right angle to the arm and make forcible supination. Apply an anterior angular splint, and have it worn for four or five days, or put the part in Jones's position for an equal period.

**Dislocation of the wrist** is very uncommon and is caused by a fall upon the hand.

**Backward Dislocation of the Wrist.**—*Symptoms.*—The deformity in backward dislocation of the wrist (Fig. 410, A) resembles that of Colles's fracture (Fig. 410, B). The fingers are flexed, the wrist is bent backward, the radius projects on the front of the wrist, the carpus projects on the dorsal

Fig. 410.—Deformity in dislocation of the wrist backward (A) and in Colles's fracture (B) (Stimson).

surface of the forearm, the relation of the styloid process of the radius to the styloid process of the ulna is unaltered (it is altered in Colles's fracture), there is rigidity, and crepitus is absent.

**Forward dislocation of the wrist** is very unusual and is caused by a fall upon the back of the hand.

*Symptoms and Treatment.*—In forward dislocation of the wrist the radius and ulna project posteriorly and the carpus projects in front. The *treatment* in both of these dislocations is reduction by extension and manipulation, the use of a Bond splint for ten days, and the employment of passive motion after five or six days.

**Dislocation at the inferior radio-ulnar articulation** is rare and is caused by twisting.

*Symptoms and Treatment.*—In *forward* dislocation at the inferior radio-ulnar articulation the forearm is pronated, the space between the styloid processes is diminished, and the ulna forms a projection posteriorly. In *backward* dislocation the forearm is supinated, the space between the styloid processes is diminished, and the ulna projects in front. Reduction is accomplished by extension and manipulation. Two straight splints (as in fracture of both bones) are to be applied for four weeks, and passive motion is to be made in the third week.

**Dislocation of Individual Carpal Bones.**—Pick says there is one weak spot, which is "between the head of the os magnum and the scaphoid and semilunar bones," and the os magnum may be forced up. The lesion is called

by some dislocation of the os magnum backward. Codman and Chase ("Annals of Surgery," March and June, 1905) regard the injury as really *dislocation of the semilunar forward*, a dislocation which may be associated with fracture of the carpal scaphoid. The injury is caused by forcible overextension or by twisting of the wrist. According to Codman and Chase, the injury is most frequently met with in men between the ages of thirty and forty, results from violent force, immediately produces severe pain, soon followed by tenderness and ecchymosis. On examination a silver-fork deformity is observed, the posterior projection being the os magnum, this projection being separated from the radius by a groove which marks the former situation of the dislocated semilunar. The dislocated bone is felt under the flexor tendons of the wrist, the palm seems shorter than its fellow, the fingers are partly flexed, active or passive motion causes pain, and the *x*-rays exhibit the dislocated bone (Ibid.).

*Treatment.*—According to Codman and Chase, recent dislocations (even after the fifth week) may be reduced by hyperextension followed by hyperflexion over "the thumbs of an assistant held firmly in the flexure of the wrist or the semilunar" (Ibid.).

If bloodless reduction fails, the author advises palmar incision and reduction, and if this fails, excision of the bone. If in excising the semilunar the scaphoid is found to be fractured, the proximal part or the entire scaphoid must also be removed.

**Dislocation of a metacarpal bone** is seldom encountered. The first metacarpal bone is most liable to dislocation.

*Symptoms and Treatment.*—Dislocation of a metacarpal bone is obvious because of projection. It is reduced by extension and manipulation, a straight splint and large pad for the palm are applied (as in fracture of the metacarpus), and the splint is worn for three weeks.

**Dislocation at a metacarpophalangeal articulation** is uncommon. Backward dislocation is the most common. The *cause* is a fall upon the hand.

*Symptoms and Treatment.*—A dislocation at a metacarpophalangeal articulation is obvious. Reduction is easily effected by extension and manipulation, except in the case of the thumb. A splint must be worn for three weeks.

**Dislocation of the Metacarpophalangeal Joint of the Thumb.**—In this dislocation the phalanx usually passes backward. In some cases the long flexor of the thumb gets to the ulnar side of the head of the metacarpal bone and hinders reduction (J. Hutchinson, Jr., in "Brit. Med. Jour.," Jan. 15, 1898). The chief impediments to reduction, as demonstrated by Farabeuf, are the sesamoid bones and glenoid ligament, which accompany the base of the phalanx in the dislocation. It is not probable that the catching of the metacarpal bone between the two heads of the flexor brevis, which often happens, is an important impediment.

The *symptoms* of *backward* dislocation are as follows: The base of the first phalanx rests upon the metacarpal bone; the head of the metacarpal bone projects forward and buttonholes the muscles of the thumb; the first phalanx of the thumb is strongly extended, and the terminal phalanx is semiflexed. The *symptoms* of *forward* dislocation are as follows: The base of the first phalanx is felt in the palm, and the head of the metacarpal bone is felt posteriorly.

*Treatment.*—In treating *backward* dislocation of the metacarpophalangeal joint of the thumb reduction is difficult. Always give ether. Keetley's directions are to adduct the metacarpal bone into the palm (this relaxes the flexor muscles) and to have an assistant hold it; bend the thumb strongly back, extend, pull the thumb toward the fingers, and suddenly flex. To get a firm enough grasp for these manipulations use the apparatus of Char-

rière or of Levis (Figs. 411, 412). If the above maneuvers fail, incise freely on the dorsum and reduce. Tenotomy is seldom of service. After reduction of this dislocation a splint must be worn for three weeks. In *forward* dislocation reduction is easily effected by strong extension and forced flexion. A splint is to be worn for three weeks.

A **dislocation of a phalanx** may be complete or may be partial. It is most common between the first and second phalanges.

*Symptoms and Treatment.*—Dislocations of the phalanges are obvious. In reducing such dislocations employ extension and manipulation. Use a splint for one week.

**Dislocations of the Ribs and Costal Cartilages.**—The ribs may be dislocated from the vertebræ. This accident is seldom uncomplicated, and cannot be

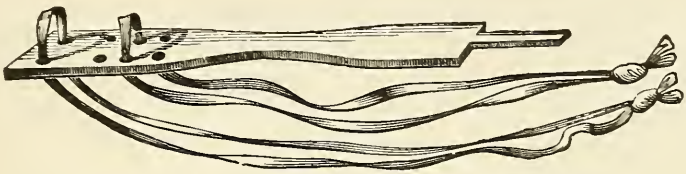


Fig. 411.—Levis's splint for reducing dislocation of phalanges.

differentiated from fracture without a skiagraph. The diagnosis is rarely made, and the injury is treated as a fracture. The ribs may be dislocated from their cartilages, one or more ribs being displaced. The end of the rib forms an anterior projection, there is a depression over the cartilage, and crepitus is absent. *Treatment* is the same as that employed for fractured ribs. The costal cartilages may be displaced from the sternum, forming an anterior projection upon this bone. Reduction is brought about by placing the patient upon a table, with a sand-pillow between the scapulæ, pushing back the shoulders and chest, and forcing the cartilage into place. The dressings are the same as those used for fractured sternum. The cartilages of the lower ribs (sixth, seventh, eighth, ninth, and tenth) may be separated. The inferior cartilage goes forward and can be felt. Pick states that reduction is brought about by causing

Fig. 412.—Levis's splint applied.

the patient to hold the chest full of air while efforts are made to push the cartilage into place. The injury is dressed as are fractured ribs (see page 544).

**Dislocation of the Sternum.**—In dislocation of the body of the sternum the manubrium is separated from the gladiolus. The injury is a rare one, is usually associated with fracture, and is most common in the young. It is due in most cases to violent direct force inflicted by a fall or heavy blow; it may be due to indirect force and arose in a reported case of acute tetanus. The *symptoms* and *treatment* are the same as those of fracture (see page 546). Dislocation of the ensiform process is one of the rarest of injuries. It is usually due to direct force, but Polaillon reports a case caused by tight lacing.

**Pelvic dislocations** are almost always complicated by fracture. A pubic bone can be dislocated by falls from a height or by the application of violent force

to the acetabula. The condition may happen from accident while riding a horse. The dislocation may be up or down, front or back, and it may damage the urethra or the bladder. The patient cannot stand; there are great pain and recognizable deformity. Treat by molding the bones into place, by applying a pelvic girdle, and by rest in bed for four weeks. Pure separation or relaxation of the symphysis is seldom the result of injury and is usually due to childbirth. In this condition there is mobility, pain on pressure and movement, and pain on abduction of the thighs. The treatment is rest in bed and the application of a pelvic girdle. Occasionally in traumatic and also in post-obstetric separation it is necessary to wire the bones. Dislocation of the sacro-iliac joint is produced by falls. Movement on the part of the patient is diffi-

Fig. 413.—Thyroid dislocation of the femur eight weeks after the accident. Reduced by open section (Rugh).

cult or impossible; there is violent pain, and often paralysis (from pressure upon nerves). In dislocation backward there is apparent shortening of the leg, eversion of the foot exists, and the ilium moves posteriorly and upward. In dislocation forward the anterior superior iliac spine projects and the pelvis is broadened. Sacro-iliac dislocation is reduced by holding the pelvis firm and making extension by means of a pulley. The patient stays in bed for four weeks and wears a pelvic belt as in fracture.

**Dislocation of the coccyx** is considered on page 549.

**Dislocation of the Femur (Hip-joint).**—Dislocation of this joint is not often encountered, as the hip-joint is very strong. It is most apt to occur in a young adult. In forcible extension the head of the femur presses against the capsule of the joint, but the capsule here is very thick, and certain muscles, the rectus,

psoas, and iliacus, are pulled tight and serve to strengthen it. The head of the bone cannot go directly upward because of the acetabulum (Edmund Owen). The weak point of the acetabular rim is below; the weak part of the capsule is also below; hence forced abduction is apt to push the head of the bone through the lower part of the capsule, a dislocation occurring primarily into the thyroid foramen. The signs of the dislocation depend upon the untorn portion of the capsule. The anterior portion of the capsule, including the Y-ligament, usually escapes laceration. Vessels are rarely injured. Muscles are often torn. In some cases the sciatic nerve is lacerated, bruised, or caught up on the neck of the femur during the circumduction of attempted reduction. Four forms of hip-joint dislocation are usually described: (1) upward and backward, on the dorsum of the ilium; (2) backward, to the border of the sciatic notch; (3) downward, into the obturator foramen, and (4) inward, on the pubes.

Fig. 414.—Dislocation of femur upon the dorsum of the ilium (Dr. Ohnesorg's case).

All dislocations are primarily inward or outward. From these initial positions the head may be shifted to any region about the socket within reach of the remnant of untorn capsule (Oscar H. Allis). Allis rejects the old classification and suggests the following:

Low thyroid, Mid- " High " } All present abduction and outward rotation.

Reversed thyroid:

Low dorsal, Mid- " High " } All present adduction and inward rotation.

**Dislocation upon the dorsum of the ilium** (Fig. 414) is the commonest form. One-half of all hip dislocations are of this variety. It is *caused* by a fall or a blow when the limb is flexed and abducted (as in carrying a weight upon the shoulder), by a fall upon the knees or feet, by a weight striking the back while bending, etc. Allis says rotation inward is the chief element in its production. In this dislocation the head of the femur goes upward and backward, rests upon the ilium, and is always above the tendon of the obturator internus muscle. This dislocation is secondary to thyroid dislocation, muscular action shifting the bone from its initial seat of displacement.

*Signs.*—Dislocation upon the dorsum of the ilium is indicated by the following symptoms: the buttock appears flat and broad; the great trochanter is above Nélaton's line and is deeply placed; the head of the bone can be detected in its new situation; deep pressure in front of the joint finds a hollow; the leg is shortened by about 2 or 3 inches; the fascia lata is relaxed; in some thin people the socket can be outlined; when the patient is recumbent the injured extremity can be brought to the perpendicular without flexing the leg (Allis); the knee is somewhat flexed; the thigh is slightly flexed,

inwardly rotated, and adducted (Fig. 415) (this is shown by the fact that the axis of the thigh of the injured side, if prolonged, would pass through the lower third of the sound thigh); when the capsule is extensively lacerated there may be no adduction and may be eversion (Allis); the heel is raised, and the great toe of the foot of the injured side rests upon the front of the instep or the ankle of the sound side (Fig. 415); rigidity exists; voluntary movement is impossible, though some passive motion is possible in the direction of the deformity (the deformity can be made more marked). If a patient is recumbent and the knees vertical, the foot of the sound extremity is free of the bed, but the foot of the injured extremity touches the bed (*Allis's sign*).

*Diagnosis.*—Examine first without anesthesia. The *x*-rays are invaluable in diagnosis. If the *x*-rays are not obtainable, examine again while the patient is anesthetized. Dislocation is distinguished from intracapsular fracture by noting the inversion, the great shortening, the absence of crepitus, the age of the subject, and the nature of the force. The nature of the force, the inversion, and the absence of crepitus mark the diagnosis from extracapsular fracture.

Fig. 415.—Hip-joint dislocation upon the dorsum of the ilium (Cooper).

*Treatment.*—The chief obstacle to reduction in dislocation upon the dorsum of the ilium, Bigelow states, is the untorn portion of the capsule, especially the Y-ligament. The iliofemoral, Y, or Bigelow's ligament resembles an inverted Y, arises from the anterior inferior spine of the ilium, is inserted into the anterior intertrochanteric line, and is incorporated into the front of the capsule. To reduce a dislocation this ligament must be relaxed by manipulation or be torn by extension. Manipulation makes the head of the bone retrace its steps over the same route it took in emerging. Give ether; place the patient supine upon a mattress on the floor; flex the leg on the thigh (to relax the hamstrings), flex the thigh on the pelvis; increase the adduction over the middle line; strongly abduct; perform external rotation and extension. This treatment may be summed up as flexion, adduction, external circumduction, and extension; or, as Pick puts it, "bend up, roll out, turn out, and extend." Allis's advice is to fix the pelvis to the floor, lift the head of the bone to the level of the socket, rotate outward by carrying the leg toward the pubis, and extend the femur. If extension and counterextension are employed, make extension in the axis of the dislocated limb and obtain counterextension by a perineal band. The extension band is fastened to the thigh by a clove-hitch. After reduction put the patient to bed and use sand-bags (as in fracture of the hip) for four weeks. We may tie the knees together instead of using the sand-bags. Passive motion is made in the third week. The pulleys must not be used in reduction. They may inflict great or even fatal injury. If the surgeon fails to reduce the deformity, there are two courses open to him. He may let it alone. He may operate. If he lets it alone, the limb will almost certainly become ankylosed, though probably useful. If he determines to operate, he must recognize that tenotomy is useless. It is necessary to make a free incision in order to restore the bone.

**Dislocation Onto the Border of the Sciatic Notch.**—In this dislocation the head of the bone passes backward and a little upward, and rests upon the ischium at the margin of the sciatic notch (not in the notch), below the tendon of the obturator internus muscle. The *causes* are the same as those given for the previous dislocation.

The *signs* in dislocation by the sciatic notch are like those of dislocation upon the dorsum of the ilium, but they are not so marked. There

are flattening and broadening of the hip; ascent of the trochanter above Nélaton's line; shortening to the extent of an inch; relaxation of the fascia lata. If the knee of the injured side is vertical, the sole of the foot touches the bed. Flexion, inward rotation, and adduction exist, but the axis of the femur of the injured side passes through the knee of the sound side, and the ball of the great toe of the injured side rests upon the great toe of the sound side (Fig. 416). Other symptoms are identical with those of dislocation upon the dorsum of the ilium, but are less pronounced. Allis's signs of this dislocation are of value: if, with the patient recumbent, the thighs are brought to a right angle with the body, shortening on the affected side is materially increased; if the dislocated thigh is extended, the back arches as in hip disease.

Fig. 416.—Hip-joint dislocation onto the sciatic notch (Cooper).

*Diagnosis and Treatment.*—The signs of dislocation on the border of the sciatic notch are similar to, but are less marked than, those of dorsal dislocation, and, being a backward dislocation, the reduction and treatment are the same as for dislocation backward upon the dorsum of the ilium.

**Dislocation Downward Into the Obturator Foramen** (Fig. 417).—Downward dislocation is the primary position of most dislocations of the hip, the bone rarely remaining in the thyroid foramen, but usually mounting up as a result of muscular action or of the initial violence. The *cause* is a violent abduction by falls or by stepping from a moving car.

*Signs.*—Dislocation downward into the obturator foramen is indicated by flattening of the hip; the head of the bone is felt in its new position and is missed from the acetabulum; rigidity exists; passive motion is only possible in the direction of deformity, and that to a slight extent; a hollow is noted over the great trochanter, which process is well below Nélaton's line and nearer than normal to the middle line. The gluteal crease is lower than is the crease of the opposite side; there is lengthening to the extent of 1 to 2 inches; the body is bent forward by the traction upon the psoas and iliacus muscles, and is also deviated to the side, thus causing great apparent lengthening; the limb is advanced partially flexed and abducted, and the foot is pointed straight ahead or is a little everted (Fig. 417); when the patient is recumbent extension is impossible, the knees cannot be pushed together without great pain, and the abductor muscles are hard and rigid. Allis's signs are absent. Unreduced dislocations do well, the patient obtaining a very useful hip-joint (Sédillot).

Fig. 417.—Hip-joint dislocation into the obturator or thyroid foramen (Cooper).

*Treatment.*—In treating dislocation downward into the obturator foramen give ether and effect reduction, if possible, by manipulation, and, if this fails, by extension. To reduce by manipulation, flex the leg on the thigh and the thigh on the pelvis, and then perform, in the following order, abduction, internal circumduction, and extension. Allis's rule of reduction is as follows: fix the pelvis to the floor; pull the head of the femur outward and above the socket; fix the head; push the knee toward sound knee and extend the femur. If extension is made, make traction in the axis of the limb by means of muslin fastened around the thigh by a clove-hitch. Do not use pulleys; incise rather than use them.

**Dislocation upon the pubis** is a very uncommon accident. The head of the bone usually rests just internal to the anterior inferior spine of the

ilium. The primary position of the bone is in the thyroid foramen; the pubic dislocation, when it occurs, is always secondary, and is due to the initial force and to muscular action.

*Symptoms.*—In pubic dislocation the head of the bone can be felt and seen in its new position; the hip is flattened; there is a hollow over the great trochanter, this process being found below the anterior superior spine of the ilium; there is shortening to the extent of 1 inch; the limb is in abduction with eversion (Fig. 418), and the knees cannot be approximated without great pain.

In the *treatment* of pubic dislocation give ether and employ manipulation as for thyroid dislocation. If this fails, employ extension. The limb is well abducted, extension is made downward and backward, and the head of the femur is pulled outward "by a towel around the thigh, just beneath the groin" (Keetley). The after-treatment is the same as that for the previous forms.

Fig. 418.—Dislocation on pubis (Cooper).

**Central or Internal Dislocation** (Fig. 419).—By this term we mean that the head of the femur has been displaced and perhaps forced through a fractured acetabulum into the pelvis. It is not a genuine dislocation. There is neither tearing nor stretching of the capsule and the acetabular floor may remain in contact with the femoral head. Skillern and Pancoast ("Annals of Surgery," Jan., 1912) point out that "the injury varies from a slight depression of the floor of the acetabulum . . . to the passage of the femoral head into the pelvic cavity." For the first injury they suggest the term *fractura acetabuli perforans;* for the second, *fractura acetabuli perforata.* This injury is due to violent force. The usual cause is a fall upon the great trochanter. The symptoms vary with the degree of depression or fracture of the floor of the acetabulum. If there is only slight depression of the floor, there may be no appreciable shortening and very slight approach of the trochanter toward the symphysis pubis. The *x*-rays may be needed to make a diagnosis. If the head has perforated, shortening will be evident, there will be marked approach of the trochanter toward the midline, and vaginal or rectal palpation will discover the displaced femoral head. In both injuries there are pain, tenderness, and impaired mobility. There may be serious damage within the pelvis. Intrapelvic hemorrhage is common. The peritoneum may be lacerated, the bowel may be damaged, the bladder injured, or the obturator nerve bruised (Skillern and Pancoast, Ibid.).

*Treatment.*—If complications such as are mentioned above exist, at once open the abdomen and repair the injury. Reduce the deformity by extension and counterextension. Treat by "extension in the axis of the limb in conjunction with lateral traction upon the femoral neck" (Ibid.). After a week, if there is no contra-indication, apply a plaster-of-Paris dressing from the toes to the chest.

Skillern and Pancoast (Ibid.) state that the injury was first described by Callisen in 1788, and that, with their 4 cases, 55 have been reported.

**Anomalous Dislocations of the Hip.**—In *supraspinous dislocation* the dislocation of the hip is backward, the head of the femur resting upon the ilium above or even anterior to the anterior superior spine. In *ischial dislocation* the dislocation is downward and backward, the head of the femur resting on the ischial tuberosity or in the lesser sciatic notch. *Monteggia's dislocation* is a supraspinous dislocation with eversion of the limb. In *perineal dislocation* the head of the femur is in the perineum. In *suprapubic dislocation* the head of the femur passes above the pubes. In *subspinous dislocation* the femoral head rests on the horizontal ramus of the pubes.

**Dislocation with Catching Up of the Sciatic Nerve During Reduction.**—This accident causes severe pain. The leg is flexed on the thigh and the thigh is flexed on the pelvis. Allis tells us that the task of reduction is very unpromising. We must strive to put the neck of the femur in such a position that the nerve will "drop off," and yet often the nerve cannot drop off because it is held by adhesion to the injured muscles. Allis attempts reduction by the following plan:

1. Place the patient upon his back and redislocate the femur.
2. Extend the thigh.
3. Flex the leg on the thigh.
4. Turn the ankle out until the leg is horizontal (this causes the head of the bone to look downward).
5. "Shake, shock, jar, adduct, and abduct," to disengage the nerve.
6. Rotate into socket without flexing leg (without making nerve tense).
7. If this fails, make an incision above the popliteal space, and draw the nerve out of the wound. Detach the head of the bone from its entanglement and rotate it into the socket.[1]

Fig. 419.—Dislocation of left hip. Fracture of acetabulum, ischium, and pubes (right), with central dislocation of hip. Separation of pubic arch.

**Dislocation of the Head of the Femur with Fracture of the Shaft of the Bone.**—We may incise, replace, and plate the fragments. We may use McBurney's hooks as in the shoulder. We may be forced to do a resection of the head.

Allis maintains that it is possible to reduce it by manipulation. He states that the upper fragment is the entire lever, and the lower fragment "is only the agent through which we apply our force." The fragments are not completely separated, but are connected at one side by material which is "partly periosteal, partly tendinous, and partly muscular." This connecting material enables us to make traction upon the upper fragment, but does not allow "rotation, circumduction, and leverage through the agency of the lower frag-

[1] Allis's views will be found in "An Inquiry Into the Difficulties Encountered in the Reduction of Dislocations of the Hip," by Oscar H. Allis, M. D. This highly original and valuable treatise received the Samuel D. Gross Prize of the Philadelphia Academy of Surgery in 1895.

ment." Hence "the only agency at our command is traction." If the dislocation is inward (forward), draw the head outward and have an assistant make direct pressure upon the head of the bone. If this fails, the assistant holds the head of the bone to prevent its slipping into the thyroid depression, and the surgeon makes traction inward or inward and downward. If the dislocation is outward (backward), make traction directly upward to lift the head of the bone to the level of the socket, and try to place the head over the socket by traction obliquely upward and inward. During all these manipulations an assistant presses upon the trochanter to prevent the head of the bone slipping back. Traction is now made downward and inward, and the tightened ligament may drag the head of the bone into place.

**Dislocation of the Knee.**—It is a rare injury. There are four forms—forward, backward, outward, and inward. Any one of the four may be complete or incomplete; the commonest dislocations are lateral. The *cause* is violent force, such as a fall, or in jumping from a moving train, or in being caught by the foot and dragged.

Fig. 420.—Old dislocation of the patella outward.

**Dislocation Forward of the Knee-joint.**—In the *complete* form of forward dislocation the deformity is obvious. The limb is usually extended, but it may be flexed. Much shortening exists; the condyles are felt posterior and below; the head of the tibia is felt anterior and above; the patella is movable and the quadriceps is lax; pressure of the condyles upon the contents of the popliteal space arrests the tibial pulse and causes edema and intense pain. In *incomplete* dislocation the symptoms are identical in kind, but are less pronounced.

*Treatment.*—Compound dislocation of the knee-joint often demands excision or amputation. In simple dislocation give ether, have one assistant extend the leg while another makes counterextension on the thigh, and the surgeon pushes the bone into place. Reduction is easy because of ligamentous laceration. Place the limb on a double inclined plane, and combat inflammation by the usual methods (see Synovitis, page 617). Begin passive motion in the third week. The patient must wear a knee-support for months. Very extensive laceration of ligaments calls for incision and suturing. If the popliteal vessels are much damaged, gangrene will supervene and amputation will be demanded.

**Dislocation Backward of the Knee-joint.**—In the *complete* form of backward knee-joint dislocation displacement is not so great as in dislocation forward. The head of the tibia projects posteriorly and above, the femoral condyles anteriorly and below; the leg is, as a rule, partly flexed, but it may be extended, and there is moderate shortening. In *incomplete* dislocation the symptoms are less marked.

The *treatment* of backward dislocation of the knee-joint is the same as for forward dislocation.

**Dislocation outward of the knee-joint** is usually incomplete. The [inner tuberosity of the tibia in outward dislocation lies upon the outer condyle of the femur (Pick); the inner condyle of the femur projects internally; the outer tibial tuberosity and fibular head project externally, the former having a depression below it, and the latter above it; the leg is semiflexed, but shortening is absent.

**Dislocation inward of the knee-joint** is usually incomplete. The outer tuberosity of the tibia in inward dislocation lies upon the inner condyle of the femur; the outer condyle of the femur forms an external prominence, and the inner tuberosity of the tibia forms an internal prominence. Pick cautions us not to mistake a separation of the lower femoral epiphysis for lateral dislocation (the former is reduced easily, the deformity tends to recur, and there is soft crepitus).

*Treatment.*—In treating lateral dislocation of the knee-joint, effect extension and counterextension as in anteroposterior dislocations. The leg is moved from side to side and attempts are made at rotation. The after-treatment is the same as that for anteroposterior luxations.

**Dislocation of the patella** is seldom congenital. There are 35 congenital cases on record (Bajardi). There are three forms of dislocation of the patella: outward, inward, and edgewise. The so-called dislocation upward is, in reality, rupture of the ligamentum patellæ (see page 718).

Fig. 421.—Dr. Morris Booth Miller's case of outward dislocation of the patella from direct force six months after the fall. Failed of reduction under ether anesthesia.

**Dislocation of the patella outward** (Fig. 420) may be due to muscular action or to direct force, and occurs during extension of the leg. It occasionally happens in a person with knock-knee. If dislocation is complete the bone lies upon the external surface of the external condyle; if incomplete, the patella rests upon the anterior surface of the external condyle. The leg is extended, flexion is impossible, and attempts at flexion produce great agony. In the patient shown in Fig. 420 flexion became possible in an unreduced dislocation, but not until months after the accident. The knee is wider than normal. There is a hollow in front of the joint. The bone is felt in its new position.

**Dislocation of the patella inward** is very rare (Fig. 421). The signs are like those of dislocation outward, except that the patella rests upon the inner condyle.

*Treatment of Lateral Dislocations of the Patella.*—Give ether. Raise the

body upon a bed-rest and flex the thigh. Grasp the patella and depress the margin which is farthest from the center of the joint (Pick). The muscles may pull the bone into place. Extend the extremity and immobilize for three weeks, and then begin passive motion. Incision may be necessary in order to effect reduction.

**Dislocation of the Patella Edgewise.**—The patella rotates vertically, one edge resting between the condyles. As a rule, the outer border is in the intercondyloid notch. This condition is produced by direct force when the extremity is partly flexed. Twisting and muscular action have been assigned as causes. The condition is obvious at a glance.

*Treatment.*—Give ether. Pick recommends "sudden and forcible bending of the knee." In some cases the bone can be pushed into place, the limb being extended and flexed as in the reduction of a lateral dislocation. In some cases incision will be necessary.

**Dislocation of the Semilunar Cartilages of the Knee-joint** (*the Internal Derangement of Hey; Subluxation of the Knee-joint*).—The condition was described by Hey, of Leeds, in 1803. The interarticular cartilages of the knee-joint are attached in front of and behind the tibial spine, and the convexity of each cartilage is attached to the edge of the corresponding tibial tuberosity by means of the coronary ligament. The internal cartilage is fastened to the internal lateral ligament and has a moderate freedom of movement. The outer cartilage is not connected with the external lateral ligament and is not freely movable. It has been stated that the outer cartilage is more frequently dislocated than the inner, but modern experience indicates that this is not true, and that the internal cartilage is the one most apt to suffer. In 17 cases operated upon by Barker the internal cartilage was involved in every case ("Lancet," Jan. 4, 1902). Those persons whose occupations force them to pass considerable time upon their knees are predisposed to this accident (Annandale). The derangement of the cartilage is usually caused by a sudden external rotation of the tibia while the knee-joint is in partial flexion; for instance, when the patient stumbles over an obstacle, the knee-joint being partially flexed, the tibia is twisted outward. When the joint is flexed a normal cartilage moves backward, and when it is extended moves forward again. When the cartilage is thrown out by the sudden eversion and flexion of the tibia it is caught and does not move into place readily when the leg is extended. The tear takes place in the direction of the fibers of the cartilage.

*Symptoms.*—The first indication of interarticular cartilage displacement is a sudden, violent, sickening pain in the knee, which may be so severe as to cause the patient to fall to the ground. The knee is in a position of fixed semiflexion. Further flexion is possible, but extension is impossible. In some cases the patient can voluntarily make further flexion; in others, the pain is so severe that he either cannot or will not do it; but increase of flexion can be obtained by passive motion. The joint is, however, blocked both to passive and to voluntary extension. Attempts at passive motion are productive of fierce pain. If either cartilage is displaced away from the tibial spine, a prominence may be found on one or the other side of the knee-joint. If the displacement takes place toward the tibial spine a prominence may be found on one side of the ligament of the patella. Subluxation is rapidly followed by inflammation of the synovial membrane of the joint and inflammation of the cartilage itself; and swelling quickly masks the projection of the cartilage. This accident is frequently mistaken for the blocking of the joint by a floating cartilage; but a dislocated cartilage always remains in the same position, and a loose cartilage changes its position from time to time (Turner). Loose bodies in a joint produce pain of a shifting character and interference with both flexion and extension, or with either flexion or extension in an

irregular way (Cotterill). In regard to the diagnosis, Cotterill points out that in a sprain of the joint extension is not painful, but flexion is interfered with; whereas, in the dislocation of a cartilage of the joint, flexion is still possible, but extension cannot be carried out ("Lancet," Feb. 22, 1902).

*Treatment.*—To reduce a displaced semilunar cartilage I have used with satisfaction a method described by Henry W. Jacob ("Brit. Med. Jour.," March 7, 1908). It is not followed by severe synovitis unless the patient has walked or has made repeated efforts to reduce the displacement, and as it is a painless method ether is not required. Jacob describes it as follows:

"The patient lies on a bed or couch, the surgeon standing on the outer side of the limb affected, with his face toward the patient's foot; the patient then raises his leg off the couch in the semiflexed position, the surgeon grasps the patient's leg in both hands, and using his own thigh as a fulcrum, by means of a steady pulling movement draws the patient's leg outward while the surgeon's thigh keeps the patient's femur in a fixed position; directly this movement is effected the patient must steadily extend the limb, and the displaced cartilage will probably go back with a slight click; if the first movement of extension is not successful the maneuver must be repeated without any hurry or unnecessary force, and after a few attempts the cartilage can usually be felt to slip in without pain or inconvenience."

In treating dislocation of a semilunar cartilage of the knee it is customary to give ether and reduce by forced flexion and external rotation. Extension becomes possible if the cartilage is freed. During these maneuvers an assistant endeavors to push any projection of cartilage into place. After reduction apply a splint for two weeks and combat inflammation by proper remedies (see Synovitis); then begin passive motion. At the end of two weeks apply a firm knee-cap made of leather and let the patient get about on crutches. After a couple of weeks the crutches can be laid aside. As recurrence of the displacement is usual, the patient should wear a knee-cap during the day for many months. A partial tear may entirely heal when thus treated by rest and support; an extensive tear will not, although even in such cases a useful but somewhat stiff joint may be obtained. If it is found impossible to unlock the blocked joint, or if the tear is extensive and redislocation is prone to occur, an operation is advisable. The joint is opened and the loose cartilage is pushed into place and held by stitches to the periosteum, or the loosened portion is excised. Annandale, in 1885, sutured through a transverse incision. Freeman and others used sutures to fix the cartilage. Excision is just as satisfactory as suturing, and after excision recurrence of the trouble is impossible. After excision the extremity is placed upon a posterior splint. Passive motion is begun in two weeks.

**Dislocation of the Fibula at the Superior Tibiofibular Articulation.**—This injury is rare. The head of the fibula may go forward or backward. The *causes* are direct force and violent adduction of the foot with abduction of the knee (Bryant).

*Symptoms.*—After dislocation of the fibula the position is one of semiflexion of the knee, voluntary extension and flexion being impaired or lost. A distinct movable projection is readily noticed in front or behind, which is found to be continuous with the fibula. There is a depression over the normal position of the head of the fibula.

*Treatment.*—In treating dislocation of the fibula bend the knee to relax the biceps, and proceed to push the bone into place. Put a compress over the head of the fibula, apply a bandage, and put the limb on a double inclined plane. The peroneal nerve passes over the fibula just below the head of the bone and care must be taken to avoid making injurious pressure upon the nerve. At the end of three weeks put a lacing knee-support upon the knee and let the

patient up. Displacement being liable to recur, a knee-cap must be worn for a year.

**Dislocation of the Ankle-joint.**—This injury is not unusual. Fracture is a frequent complication. There are five forms of ankle-joint dislocation—outward, inward, forward, backward, and upward.

**Lateral dislocation of the ankle-joint** is either outward or inward, and may be complete or incomplete. In these dislocations the astragalus rotates. In incomplete dislocation "there is no great separation of the trochlear surface of the astragalus from the under surface of the tibia, but the outer or inner margin of this surface is brought into contact with the articular surface of the tibia, and the whole foot presents a lateral twist" (Pick). The *causes* of these dislocations are twists of the joint.

*Symptoms.*—Incomplete outward dislocation of the ankle-joint occurs in *Pott's fracture* (see page 612). Complete outward dislocation, in which the articular surface of the astragalus is completely displaced outward from the articular surface of the tibia, and which condition is associated with a fracture of the fibula and separation of the inferior tibiofibular articulation, is known as *Dupuytren's fracture.* In incomplete dislocation the foot goes outward and upward, the fibula is fractured, and the tibiofibular ligaments are torn off. In Dupuytren's fracture the ankle is broad, the inner malleolus projects and looks lower than natural, the outer malleolus ascends with the foot, the foot rotates outward, and crepitus can be detected. In inward dislocation which is associated with fracture of the inner malleolus there is inversion, the outer malleolus projects, and crepitus can be detected. In incomplete separation the symptoms are similar, but are not so marked.

*Treatment.*—In treating a case of dislocation of the ankle-joint the deformity is reduced by flexing the leg on the thigh and the thigh on the pelvis; an assistant makes counterextension from the knee; the surgeon makes extension from the foot, and at the same time rocks the astragalus into place. Dupuytren's fracture is treated in the same manner as Pott's fracture (see page 613). Dislocation inward is treated in a fracture-box for the same period as Pott's fracture.

**Anteroposterior dislocation of the ankle-joint** is rare. The *cause* is the catching of the foot in jumping or falling—direct violence. In dislocation forward the foot is lengthened, the heel is not conspicuous, the tibia and fibula project against the tendo Achillis, and the relation of the malleoli to the tarsus is altered. In incomplete dislocation the symptoms are similar, but less pronounced. In dislocation backward the foot is shortened, the tibia and fibula project in front, the heel is prominent, and the relation between the malleoli and the tarsus is altered. In incomplete dislocation the symptoms are similar, but less marked.

*Treatment.*—In anteroposterior dislocation of the ankle-joint reduce as in lateral dislocations. Sometimes the tendo Achillis must be cut. Apply a plaster-of-Paris dressing and let it be worn for two weeks; then begin passive motion, and let the patient wear side-splints for a week longer.

**Dislocation upward of the ankle-joint,** or *Nélaton's dislocation,* is a very rare injury. The astragalus is wedged between the widely separated tibia and fibula. This dislocation is usually associated with fracture. The *cause* is a fall upon the feet from a great height.

*Symptoms.*—Upward dislocation of the ankle-joint is indicated by the widening of the ankle and by the flattening of the foot. The malleoli are nearly on a level with the plantar surface of the foot, and there is absolute rigidity.

*Treatment.*—In treating upward dislocation of the ankle-joint give ether, and try to reduce by powerful extension and counterextension. Treat the injury afterward in the same manner as an anteroposterior luxation.

**Dislocation of the Astragalus.**—The astragalus may be displaced from the bones of the leg and at the same time be separated from the rest of the tarsus. The displacement may be forward, backward, outward, inward, or rotary.

**Dislocation of the astragalus forward** or **backward** is caused by falls or twists.

*Symptoms.*—In forward dislocation the astragalus projects strongly; there is shortening of the foot, and the malleoli approach the plantar aspect of the foot; the foot is deviated to one side or to the other, and there is absolute rigidity of the ankle-joint. In incomplete luxations the symptoms are similar, but less marked. This dislocation may be obliquely forward. In backward dislocation of the astragalus the foot is not deviated to either side; the astragalus projects between the malleoli and above the os calcis, and the tendo Achillis is stretched over the projection. Rigidity is absolute. This dislocation may be obliquely backward.

**Lateral and Rotary Dislocations of the Astragalus.**—Lateral dislocations of the astragalus are rare, are always compound, and are always associated with fracture. In rotary dislocation the astragalus remains in its normal habitat after rotating on its own axis, either horizontal or vertical. The *causes* of rotary dislocation are twists of the foot when it is at a right angle to the leg (Barwell). The *symptoms* of rotary dislocations are obscure. There is rigidity, but sometimes the position of the astragalus may be made out.

*Treatment of Dislocation of the Astragalus.*—In treating astragalus dislocation reduce under ether by flexing the knee to relax the gastrocnemius, extending the foot, and pushing the bone into place. It may be necessary to cut the tendo Achillis. After reduction put up the foot and leg in a plaster-of-Paris dressing for two weeks, and then begin passive motion and apply side-splints, which are to be worn for one week more. If reduction fails, support the limb on splints, combat inflammation, and endeavor to bring about union between the dislocated bone and the tissues. Often, in unreduced dislocation, the skin sloughs over the projecting bone. Excision is demanded the moment sloughing is seen to be inevitable. Cases of compound dislocation of the astragalus require immediate excision.

**Subastragaloid Dislocation.**—This condition is a separation of the astragalus from the os calcis and scaphoid, without separation from the bones of the leg. Pick states that the usual classification for these dislocations is forward, backward, inward, and outward, but that the displacement is, as a rule, oblique, the foot passing backward and outward or backward and inward. The *cause* is twisting.

*Symptoms.*—In subastragaloid dislocation the astragalus projects on the dorsum; the foot is everted in outward dislocation and inverted in inward dislocation; the relation of the malleoli to the astragalus is unaltered; the ankle-joint is not absolutely rigid; the foot "is shortened in front and is elongated behind" (Pick).

*Treatment.*—To treat subastragaloid dislocation make extension in the direction opposite to that of the displacement. In dislocation of the tarsus backward fix a bandage around the foot, on a level with the heads of the metatarsal bones, which bandage the surgeon ties around his shoulders. The surgeon puts one knee in front of the ankle and thus fixes the leg, raises himself up to make extension upon the tarsus, and molds the bone into position. Tenotomy may be necessary. After reduction apply a plaster-of-Paris dressing and have it worn for three weeks. The ankle-joint, fortunately, is not involved, and stiffness of this articulation need not be apprehended. If reduction is impossible, take the same course as in luxations of the astragalus.

**Dislocations of the other tarsal bones** are very rare. Single bones may be dislocated, or the luxation may occur at the mediotarsal articulation.

*Symptoms and Treatment.*—Projection is an obvious *symptom* in dislocation of the other tarsal bones. The *treatment* is to reduce by extension and molding, the part being put up in plaster-of-Paris dressing for two weeks.

**Dislocations of the metatarsal bones** are rare.

*Symptoms and Treatment.*—Shortening of the toes and projection of the dislocated bone are *symptoms* of dislocation of the metatarsal bones. To *treat* these dislocations reduce by extension under ether and put up in a plaster-of-Paris dressing for two weeks. If reduction fails, the functions of the foot will not be much impaired.

**Dislocations of the phalanges** are very rare. The first phalanx of the big toe is the one most liable to dislocation.

*Symptoms and Treatment.*—Dislocations of the phalanges are obvious. The *treatment* is by reduction and fixation, as in dislocation of the thumb. Immobilize for two weeks.

## Operations Upon Bones and Joints

**Osteotomy.**—By the term *osteotomy* the modern surgeon means literally the sectioning of a bone for the purpose of straightening a limb ankylosed in a bad position, correcting a bony deformity, or amending a vicious union

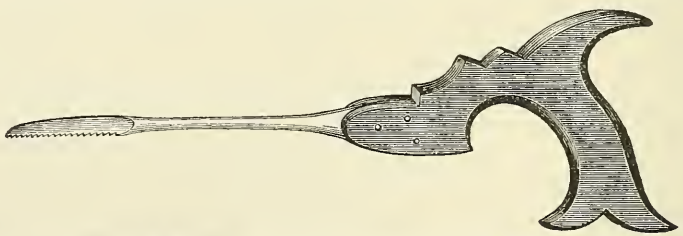


Fig. 422.—Adams's large saw. (In this day of surgery the saw has a metal handle.)

of a fracture. In a *linear osteotomy* the bone is transversely or obliquely divided at one spot; in a *cuneiform osteotomy* a wedge-shaped portion of bone is removed. The operation of osteotomy may be performed with a saw (Fig. 422) or with an osteotome. The saw creates dust, draws much air into the wound, and lacerates the tissues to a considerable degree. Most surgeons prefer the chisel or the osteotome. The osteotome slopes down to a point from each side (Fig. 423); the chisel is straight on one side and on the other is bevelled to a point.

Fig. 423.—Osteotome.

**Osteotomy for Genu Valgum, or Knock-knee** (*Macewen's Operation*, Fig. 424).—The patient lies upon his back, being rolled a little toward the diseased side. The leg of the diseased side is partly flexed upon the thigh and the thigh upon the pelvis, and the extremity is laid upon its outer surface, a sand-bag being pushed between the extremity and the bed, opposite to the site of section. The flexion of the knee relaxes the popliteal vessels and saves them from injury. The surgeon, if operating on the right leg, stands outside of that extremity; if operating on the left leg, he stands opposite the left hip (Barker). The knife is inserted into the tissues and carried to the bone at the inner side of the knee, just in front of the adductor tubercle of the inner condyle and on a level with the upper border of "the patellar articular surface of the femur" (Barker). An incision is made upward 1 inch in length, in the direction of the axis of the femur. The knife is left in as a guide until the osteotome is inserted at the lower

angle of this wound. After the insertion of the osteotome the knife is withdrawn and the blade of the osteotome is turned to a right angle with the shaft of the femur, ½ inch above the epiphysis (Fig. 424). The osteotome is struck several times with a mallet; the handle is moved several times toward and from the body, so as to widen the cut in the bone (Fig. 425); the osteotome is again struck with the mallet several times; it is again moved to and fro, and this process is continued until the bone is cut two-thirds through. If the osteotome becomes tightly fixed, it should be withdrawn and a smaller one introduced. In the soft bone of a young girl this to-and-fro movement of the chisel, if carefully executed, is not liable to break the instrument. In dense bone it may break the instrument; hence, when doing an osteotomy in dense bone, the osteotome is moved to and fro across the limb and slight downward pressure upon the handle will to a great extent prevent binding. When the bone is cut two-thirds through the osteotome is withdrawn, a piece of wet antiseptic gauze is held over the wound, and the surgeon fractures the femur by strong adduction. The wound is neither sutured nor drained, but is dressed aseptically, the entire extremity is wrapped in cotton, and a plaster-of-Paris dressing is applied and carried up to the groin. The dressing may be removed in two weeks, and the patient may subsequently be treated with sand-bags, as for an ordinary fracture of the thigh, but without extension. This operation is scarcely ever fatal.

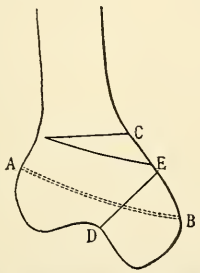



Fig. 424.—Osteotomy of the right femur in a case of knock-knee: A B, Epiphyseal line; C, section of Macewen; D E, section of Ogston.

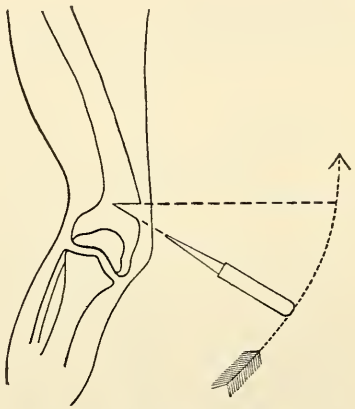


Fig. 425.—Macewen's operation for genu valgum. The chisel is held in the line for striking with a mallet; the arrow shows the direction in which the chisel is levered up and down so as to make a wide gap in the bone (after Barker).

*Ogston's Operation for Knock-knee* (Fig. 424).—In this operation the internal condyle is sawed off obliquely with an Adams saw—a proceeding which permits the straightening of the knee. The objection to the procedure is that it opens the knee-joint, and that this cavity fills up more or less with a mixture of blood and bone-dust. Macewen's operation is decidedly the safer.

**Osteotomy for a Bent Tibia.**—The tibia is divided transversely or obliquely (linear osteotomy), or a wedge-shaped piece is removed (cuneiform osteotomy). The oblique incision is the best. If the convexity of the tibial curve is inward, cut the bone from above downward and from in front backward; if the curve is forward, section the bone from above downward and from within outward. The fibula need rarely be interfered with. After the osteotomy the limb is treated just as it would be for a fracture.

**Osteotomy for Faulty Ankylosis of the Hip-joint.**—This operation is performed in order to allow straightening of a limb that has undergone bony ankylosis in a faulty or an inconvenient position. In some cases an attempt is made to obtain a movable joint, but in most cases the surgeon must be

satisfied with ankylosis in extension. Osteotomy may be performed through the neck of the femur or through the shaft of the femur below the trochanters.

**Osteotomy through the neck of the femur** is performed (1) with a saw (Adams's operation) or (2) with an osteotome.

*Adams's Operation* (Fig. 426, A).—The patient lies upon his sound hip; the surgeon stands upon the side to be operated upon, and back of the patient. The knife is entered a finger's breadth above the great trochanter, is pushed in until it strikes the neck of the bone, is then carried across the front of and at a right angle with the neck, and is withdrawn, enlarging the wound, in the soft parts as it emerges, to the extent of 1 inch. The saw is then introduced and the neck of the femur is entirely divided. After the osteotomy dress the wound antiseptically and place the extremity straight. To straighten the limb it may be found necessary to cut contracted tendons and fascial bands. After securing extension and applying dressings use the weight-extension apparatus and the sand-bags. Begin passive movements from the start if a movable joint is desired; few patients can tolerate the pain necessary to bring this about. If it is determined to aim for a stiff joint, treat the case as an intracapsular fracture would be treated.

*With an Osteotome.*—The position of the patient is the same as that for Adams's operation. An incision 1 inch long is made, starting just above the great trochanter, ascending in the axis of the femoral neck, and reaching to the bone. An osteotome is introduced, is turned to a right angle with the neck of the bone, and is struck with a mallet until the bone is *completely* divided. (It is not to be divided partially and then broken.) The after-treatment is the same as that for Adams's operation. The operation by the osteotome is to be preferred to that by the saw.

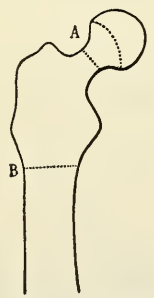


Fig. 426.—Osteotomy through the neck of the femur: A, Adams's operation; B, Gant's operation.

**Osteotomy of the Shaft of the Femur Below the Trochanters** (*Gant's Operation*).—In this operation (Fig. 426, B) the saw may be used, but the osteotome is to be preferred. The position in Gant's is like that in Adams's operation. A longitudinal incision 1 inch long is made upon the outer aspect of the femur and on a level with the lesser trochanter. The osteotome is inserted and the bone is completely divided below the lesser trochanter. The after-treatment is the same as that for Adams's operation. Gant's operation is the best method for correcting faulty position in bony ankylosis, and Adams's operation can only be employed in those cases in which the femur still has a neck which is practically unchanged.

**Osteotomy for Faulty Ankylosis of the Knee-joint.**—This operation is performed for bony ankylosis of a knee in a position of flexion. In these cases it is nearly always necessary to cut contracted tendons and fascia. These contractures tend to draw the tibia backward as in a posterior dislocation. The patient lies upon his back with his thighs flat upon the bed, the legs hanging over the end of the bed. The surgeon stands on the patient's right side. Just above the patellar articular surface upon the femur a transverse incision is made, 1 inch in length and reaching to the bone. The osteotome is introduced and the bone is cut *nearly* through. The leg is then forcibly extended. It must not be extended too violently or the popliteal vessels may be injured. In cases in which the structures of the popliteal space are tense and have not been divided the leg must not be brought at once into extension, but this position should be attained gradually by means of weights. The wound is dressed aseptically, and the extremity is placed upon a double inclined plane and is treated as for fracture near the knee-joint.

**Osteotomy for vicious union of a fracture** is performed in case of angular deformity, and is carried out in the same manner as are the above procedures. It is best, when possible, to enter the osteotome upon the concavity of the bent bone, so that the periosteum will not rupture when extension is made, and the patient will in consequence gain a longer limb.

**Osteotomy for Hallux Valgus.**—In this operation a linear osteotomy is made through the neck of the metatarsal bone of the great toe, the toe is forcibly adducted, and a splint is applied to the inside of the foot and the toe. A cuneiform osteotomy may be done instead of a linear osteotomy. The osteotomy is made through the neck of the metatarsal bone. A wedge-shaped piece of bone (the base of which is to the inner side of the foot) is removed. Charles H. Mayo makes a flap of the bursal sac and capsular ligament, divides the metatarsal bone with a Gigli wire saw, sutures the flap over the bone end, closes the skin incision, and fixes the toe in the adducted position.

**Osteotomy for Talipes Equinovarus** (*After Barker*).—The patient lies upon his back, the thigh is semiflexed, the knee is bent, and the sole of the foot rests upon the table. The surgeon stands to the right side if it is the right limb which is to be operated upon, and to the left side if it is the left limb. He feels for the outer surface of the cuboid bone, and cuts away from over the latter a piece of skin corresponding in size with the bone-wedge intended to be removed (this piece of skin must include the bursa which forms in these cases). The foot is then turned outward, the astragaloscaphoid articulation is located, and over this an incision is made "from the lower to the upper dorsal border of the scaphoid bone" (Barker), reaching through the skin only; the foot is placed again in the first position, all the soft parts are raised from off the superior surface of the tarsus, and a triangular surface corresponding with the base of the wedge to be removed is cleared; a "kite-shaped" director (Fig. 427) is passed into the external wound and projected from the internal wound; the saw is pushed through the groove of the director nearest the toes, and is made to cut through the tarsus, from the dorsum to the sole, at right angles to the metatarsal bones; the saw is pushed through the groove of the director nearest the ankle, and is made to cut from the dorsum to the sole, at right angles to the long axis of the calcaneum; the wedge-shaped piece of bone is grasped by sequestrum forceps and cut out by scissors, by bone-forceps, or by a blunt bistoury. The wound is well irrigated, the foot is straightened, the internal wound is sewed up, the external wound is sutured except at its lowest portion, where a drainage-tube is to be retained for twenty-four hours, and dressings are applied to the wound. The foot is put up in plaster or upon a Davy splint. Some surgeons insert the wedge of bone into the opening made by a linear osteotomy on the inner side of the foot.

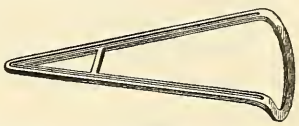


Fig. 427.—Davy's director (Pye).

**Osteotomy for Talipes Equinus.**—This operation is described by Mr. Davy, who devised it, as follows:[1] "Taking the line of the transverse tarsal joint as a guide, on the outer and inner sides of the foot, and immediately over the joint, two wedge-shaped pieces of skin are removed, equal in extent to the amount of bone demanded. The soft structures are freed on the dorsum of the foot in the way previously described; but, as the base of the osseous wedge for equinus cases is at the dorsum and its apex at the sole, the parallel wire director, instead of the kite-shaped varus one, is used. The saw is successively inserted in its grooves, and by keeping in mind the idea of a keystone a clean wedge of bone is cut out from the dorsum to the sole of the foot."

[1] Barker's "Manual of Surgical Operations."

The wedge is extracted, and the foot is straightened and is put up in plaster of Paris or is placed on a Davy splint.

**The Question of Operation for Recent Fractures.**—(See page 533.)

**Operation for Recent Simple Fracture.**—Very early after the injury is a fairly safe period for operation. Lane favors very early operation. During the first week, excluding the early hours after the injury, is a dangerous period to operate because the area of injury has a low resistance to infection. About the tenth day is a safer period for operation. Very careful sterilization is imperatively necessary. Make a free incision and expose the fracture. Remove all tissue from between the bone ends. König disagrees with Lane that all blood-clot should be removed. Arrest bleeding. Bring the fragments into apposition by extension and by using Lane's powerful forceps (see Fig. 283) as a lever. Apply nails, screws, or what apparatus we desire.

The Lane plate is an excellent device (see Fig. 281). Select a suitable plate, remove the lever, fix the plate and bones by Lowman's clamp (see Fig. 284), bore the bone by a drill (see Fig. 282) at each screw hole, insert and fix the screws, remove the clamp, and close the wound without drainage or with a cigarette drain. The part is placed upon a splint.

Figure 325 shows Lane's plate applied. If it does not loosen and does not give trouble a plate is not removed surgically.

**Recent Transverse Fracture of the Patella.**—(See page 604.)

**Recent Fracture of the Olecranon Process of the Ulna.**—(See page 568.)

**Bone-grafting, or Transplantation.**—(See pages 503 and 536.)

**Operative Treatment of Ununited Fracture.**—A method of operation long in vogue and still used is as follows: Incise longitudinally down to the

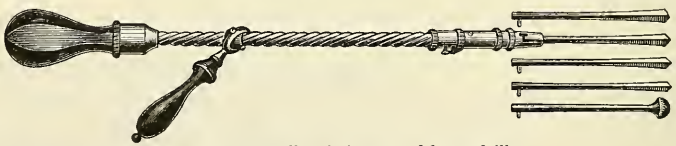


Fig. 428.—Hamilton's improved bone-drills.

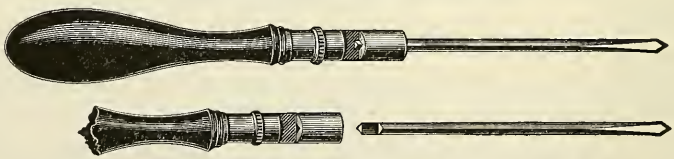


Fig. 429.—Brainard's drills with Wyeth's adjustable handles.

seat of fracture, retract the periosteum from the bone, drill the bones before cutting them, chisel away the material of imperfect union, saw through each bone end far enough from the seat of fracture to reach sound tissue, pass large silver wire through the holes (this wire should be $\frac{1}{10}$ inch in diameter for the femur, $\frac{1}{16}$ inch for the patella, etc.) (Fig. 430), twist the wires a fixed number of times (two complete turns) in the direction that the hands of a watch move (this is Keen's direction; in case removal of the wires should be demanded later we know how to untwist them; of course the surgeon must remember where he stood in relation to the limb and regard the hypothetical watch as being face up upon the part), sever the ends of the wires, and hammer their stems against the bones. The wires may never require removal. The soft parts are sutured, no drain or a cigarette drain is used, and the limb is encased in plaster of Paris. The objection to wire in fracture of a long bone is that the wire acts as a hinge and, as a consequence, alignment is apt to be disturbed. Various plans besides wiring have been employed in ununited

fracture. Gussenbauer's clamp is used by some. Clayton Parkhill's bone-clamp is a useful appliance, and holds the fragments firmly in contact. Some surgeons unite the fragments with kangaroo-tendon instead of wire (suturing of bone); others use nails of bone or ivory; others use screws. Senn asserted that the above methods will not hold fragments in contact if these fragments have a tendency to become displaced. Senn fastened the bones together by hollow cylinders of decalcified bone or ivory, the cylinders being perforated in many places (bone-ferrules). I regard the silver plate of Halsted and steel plate of Lane as the most satisfactory appliances in use.

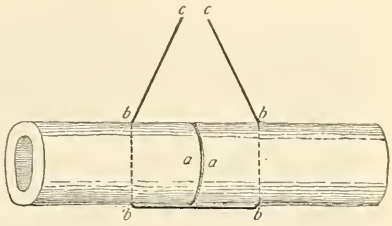



Fig. 430.—Wiring of bones for ununited fracture: *a, a,* Sawn surfaces approximated after removal of old material which was interposed between the fragments; *b–b, b–b,* perforations drilled completely across the bone; *c, c,* wires ready for twisting.

**Ununited Fractures of the Femoral Neck.**—Loreta did the first successful operation for this condition a number of years ago. The operation is not adapted to the aged, but should certainly be employed in youths and middle-aged individuals if the general condition of the patient or some particular diseased state does not forbid, and if pain is severe and disability is pronounced.

Leonard Freeman advises an anterior incision beginning below and external to the anterior superior iliac spine and extending downward, external to the sartorius, for 3 or 4 inches ("Annals of Surg.," Oct., 1904). When the fragments are exposed, the connective tissue between them is cut away by means of scissors, the surfaces of the fragments are freshened by a chisel or

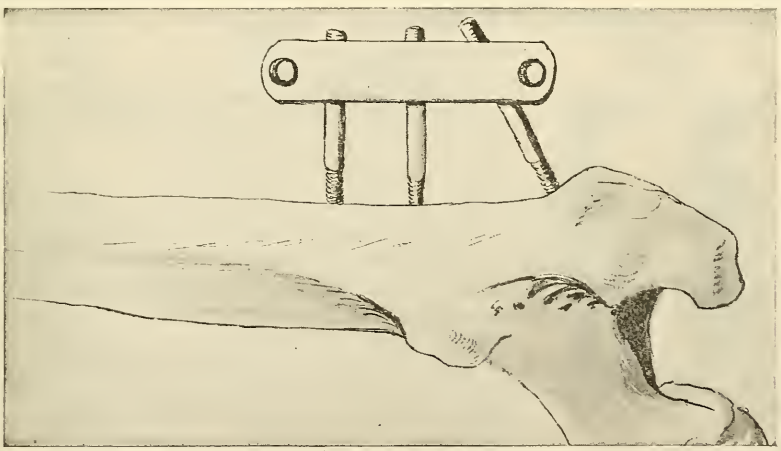


Fig. 431.—Method of securing screw of Freeman's apparatus in fracture of neck of femur; the wooden plates embracing screws (Freeman, in "Annals of Surgery," Oct., 1904).

a curet, oozing is arrested by pressure and hot water, and loose osseous splinters are removed (Freeman). Some surgeons have fixed the fragments together by nails, screws, or pegs of bone or ivory, access to the trochanter being best obtained for this purpose by making a second incision over the outer portion of that bony process. As Freeman points out, however, the head is often so very soft that none of these appliances will secure fixation.

Freeman has devised a clamp for this purpose (Figs. 431, 432). An additional incision is made over the trochanter and holes are bored for the clamp

screws, one hole being drilled "through the base of the trochanter, the external fragment of the neck, and into the head of the bone" ("Annals of Surgery," Oct., 1904). The wound is closed, dressings are applied, and extension is made on a long side splint, a pad being placed beneath the trochanter to prevent the disposition to pass backward, which movement, if it occurs, will

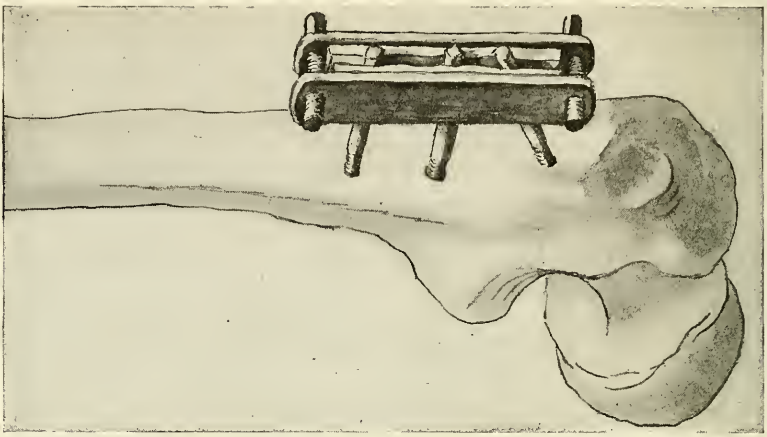


Fig. 432.—Completed screw and clamp of Freeman's apparatus for fixation of fracture of neck of femur (Freeman, in "Annals of Surgery," Oct., 1904).

cause external rotation of the limb and separation of the fragments. In about eight weeks the extension is removed and the patient is allowed about on crutches. Dr. H. Augustus Wilson has succeeded by simply nailing the fragments together without attempting to freshen their faces. He got his patient up on crutches in two weeks ("Amer. Jour. Orthopedic Surgery," Jan., 1908).

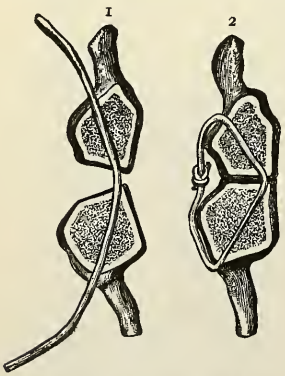



Fig. 433.—Wiring of the patella: 1, Fragments cut and cleaned and the wires passed; 2, wires twisted and hammered down upon the bone (after Barker).

In Freeman's case the screws were removed in two weeks because of infection of the cancellous tissue. A similar condition arose in Davis's case, in which two steel drills were used. Dr. H. Augustus Wilson has collected 36 cases of direct fixation of old intracapsular fractures (Ibid.).

**Ununited Fracture of Patella.**—A semilunar incision is made about the fragments, the convexity pointing up or down (this avoids the prepatellar bursa), or an incision is made in the long axis of the limb, over the middle of the space between the fragments, from well above the upper fragments to well below the lower piece. The soft parts are retracted, but the periosteum is undisturbed; each fragment is bored (Fig. 433, 1) in one or two places; the surfaces of the fragments are cut square through sound bone with a saw; all old reparative material is cut away; the wires are passed through the perforations, twisted, cut off, and hammered down (Fig. 433, 2). If the bone-fragments cannot be approximated, it may become necessary to incise the muscle around and above the patella or partially to separate the tuberosity of the tibia and bend this process upward. A small drain is inserted above the bone, the wound is sutured, aseptic dressings are applied, and the limb is put upon a Macewen splint.

**Treves's Operation for Caries of the Lumbar and Last Dorsal Vertebræ, with Abscess in the Psoas Magnus or Quadratus Lumborum Muscle.**—The patient lies upon his left side, with the knees drawn up and a sand-bag under the left loin. The surgeon stands behind the patient (Barker). An incision is made at the outer border of the erector spinæ mass, reaching from the last rib to the iliac crest and going down at once to the lumbar fascia. The lumbar aponeurosis is opened, the erector spinæ muscle is retracted inward, and the anterior portion of the erector spinæ sheath is incised. The quadratus lumborum muscle is next cut, and then the anterior leaflet of the lumbar aponeurosis is slit. The abscess is thus reached and opened and tuberculous pus flows out. The abscess-cavity is irrigated with quantities of warm corrosive sublimate solution (1 : 5000). The cavity is filled, the fluid is allowed to flow out, its exit being aided by pressure in front and changes of posture; the cavity is filled again, and so on, and, after all loose débris is removed, the bodies of the vertebræ are carefully examined with the finger and diverticula are opened. Loose pieces of bone are removed by spoons or forceps, and cavities are thoroughly but lightly curetted, as in some places the wall is very thin. By means of properly

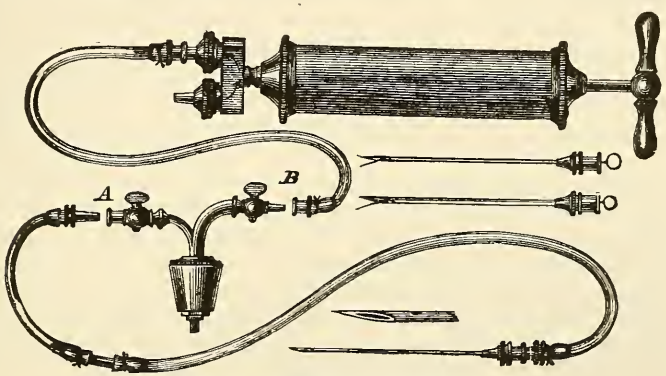



Fig. 434.—Aspirator and injector.

shaped spoons carious bone can be removed even from the anterior surface of the column (Treves). Thus the wall of the abscess is completely removed. Finally, all débris is washed out by irrigation with mercurial solution; any mercurial solution which might remain is washed out by warm water or salt solution, and the interior of the cavity is wiped dry. At this stage most operators introduce iodoform emulsion. Whether or not this is done, "the wound is closed by a series of silkworm-gut sutures, passed sufficiently deep to include the greater part of the muscular and tendinous structures with the skin" (Treves's "Operative Surgery"). Treves's operation gives a high mortality.

**Aspiration of Joints.**—In certain cases of joint-effusion from inflammation, tuberculous or otherwise, and sometimes in hemorrhage into a joint, it is desirable to remove the fluid by aspiration. The pneumatic aspirator (Fig. 434) is used. The trocar and cannula are thoroughly asepticized and the joint is prepared as for a set operation. The needle is entered at a surface free from vessels. The directions for using an aspirator are as follows: insert the stopper firmly into a strong bottle (preferably a clear glass one), then attach the short elastic hose to the stopcock *B* of the tube projecting from the stopper, and attach the other end of the same elastic hose to the exhausting or inward-flowing chamber of the pump. Next, attach one end of the longer elastic hose to the

stopcock *A* projecting from the stopper and the other end to the needle. Care should be taken that all the fittings or attachments are placed firmly into their respective places. Now close the stopcock *A* and open the stopcock *B*. By giving from thirty-five to fifty strokes of the pump a sufficient vacuum can be produced to fill with the fluid from the joint a bottle holding from a pint to a quart. After having formed the vacuum, close the stopcock *B*, and insert the needle in the joint. When the stopcock *A* is opened, suction through the needle draws the fluid from the joint. The trocar may also be used to inject antiseptic agents. After the completion of aspiration the part is dressed antiseptically and the extremity is put at rest upon a splint.

**Excisions of Bones and Joints.**—The ancients practised excision and resection for compound dislocations and fractures. For centuries surgeons removed pieces of diseased bone from joints. The operation was set forth as a definite procedure, and was first formally advised as a substitute for amputation in joint disease by Mr. Park, of Liverpool, in 1781. In 1782 the elder Moreau, of Bar-sur-Ornain, independently devised a like operation. The terms "excision" and "resection" are usually employed as synonymous, but such a use is not strictly accurate. According to Professor Ashhurst, the term *excision* means "the removal of an offending part without that total ablation of the affected portion of the body which is implied by the term 'amputation.' Hence we speak of excisions of tumors, of joints, of the eyeball, etc." *Resection* has a more restricted meaning; it signifies "an operation which takes away a middle portion and brings the ends together again, and is thus in strict surgical language limited to partial excisions of the long bones" ("International Encyclop. of Surgery," edited by John Ashhurst, Jr.). Excision of a joint is the removal of the articular portions of the bones of the joint, and also the cartilage and synovial membrane. In the hip-joint and shoulder-joint only the head of the long bone may be removed, and not the articular surfaces of both bones. In partial excision of a long bone, excision (resection) for bone disease, enough bone is known to have been removed only when the remaining bone bleeds. *Complete excision* of a bone is the removal of an entire bone. *Partial excision* or *resection* is the removal of a portion only of a bone. Excision is a conservative operation which often averts amputation.

Excision may be performed by the *open* method, in which the periosteum is not preserved, or it may be performed by the *subperiosteal* method, in which the periosteum is carefully separated by a rugine and the capsular ligament is preserved. This method was devised by Ollier, of Lyons. *Arthrectomy*, or *erasion*, is the excision of the diseased synovial membrane and ligament, and also small foci of disease of bone and cartilage.

Excision may be employed for compound dislocation, and in compound dislocations of the elbow and the shoulder it is usually performed. Excisions for compound dislocations in other large joints are very dangerous; they are rarely attempted in battlefield practice, and are to be avoided even in civil practice unless the patient is young and vigorous and every advantage can be given him during the operation and convalescence. Excision for deformity is rarely performed except upon the hip, the knee, and the shoulder, and these excisions must not be employed if the patient's condition leads one to fear the result of a protracted convalescence. Excision of the elbow, however, is usually a safe operation. In excising for deformity always consider the patient's trade and the demands of habitual position which it makes upon him.[1]

Excision is largely employed for joint-disease, especially for tuberculous joints. Bell states that attempts to preserve the limb without excision are

[1] Joseph Bell, in his "Manual of Surgical Operations."

more justifiable in the lower than in the upper limbs, because operation in the lower extremity is more dangerous than in the upper, and because a cure without operation in the lower limbs, if this cure can be brought about, gives as good a result as a cure by excision. In the upper extremity the danger from operation is less than is the danger from waiting. In a young subject an excision may remove the epiphysis, and thus lead to permanent shortening, which is productive of less inconvenience and deformity in the arm than in the leg. A danger of excision operations is that the section may be made through cancellous bony tissue; hence, if infection takes place, disastrous suppuration, phlebitis, myelitis, septicemia, or pyemia will follow; further, in excision the cut is often made through diseased tissue, and a protracted convalescence is then inevitable. Amputation is effected through healthy tissue, and the convalescence is short. Excision, however, when successful, gives the patient a very useful limb.

**Erasion, or Arthrectomy.**—This operation was suggested by Cross, of Bristol, and was perfected and established by Wright, of Manchester, between 1881 and 1888. *Erasion* is the complete removal of diseased synovial membrane, ligaments, and small foci of disease in bone and cartilage. This operation seeks to remove a depot of infection in an early stage of tuberculous synovitis, and it possesses the conspicuous merit of not interfering with the epiphysis. The term "erasion" is also used by some to designate the operation of removing healthy synovial membrane, ligaments, etc., for the purpose of producing fixation of a flail-joint due to infantile paralysis, but in such cases *arthrodesis* is the proper term for the operation. Erasion is oftenest practised upon the knee-joint.

*Erasion of the Knee-joint.*—The patient lies upon his back; the leg is flexed, with the sole of the foot planted upon the table, and an Esmarch bandage is applied at a point well up on the thigh. The surgeon stands to the right of the patient. The incision is begun in the midline of the thigh (on the side opposite to that occupied by the surgeon), about 3 inches above the patella; it is carried down across the ligament of the patella and up to a corresponding point on the opposite side of the thigh. This incision goes down to the bone; the flap is turned up and the joint exposed; the knee-joint is strongly flexed, and the synovial membrane and diseased ligaments are dissected away with scissors and forceps, great care being taken that the posterior ligaments (which, fortunately, are rarely implicated early in the case) are not divided and that the contents of the popliteal space remain intact. After removing the diseased ligaments and synovial membrane the cartilage is examined and any diseased portion is removed. The bone is then examined and any tuberculous foci are gouged away. Exposed vessels are ligated. The wound is irrigated by salt solution, the extremity is straightened, and the ends of the ligamentum patellæ are sutured, a drainage-tube is inserted at each angle of the wound, the skin is sutured, and antiseptic or sterile dressings are applied. The limb is placed upon a posterior splint for a few days, then the drainage-tubes are removed, the dressings are changed, and a plaster-of-Paris cast is applied, trap-doors being cut on each side, and the joint is kept immobile for two or three weeks. This operation is only suited to early cases in which the lesion involves chiefly or purely the synovial membrane and ligaments, and in these cases it frequently gives a good result, some capacity for motion being not unusually preserved.

**Excision of the Shoulder-joint.**—Bent, of New Castle, performed the operation in 1771 ("Manual of Operative Surgery," by Sir Frederick Treves). Syme really established the operation in surgical confidence. In the shoulder-joint *partial* excision is often performed, the head of the humerus being removed and the glenoid being undisturbed; but some patients require complete

excision, the entire glenoid depression, as well as the head of the humerus, being removed by the surgeon. Excision of the shoulder-joint is made, if possible, an intracapsular operation, the capsule being opened, but the capsular attachment to the anatomical neck of the humerus not being interfered with. In advanced cases, however, the capsular attachment must be destroyed. Excision of the shoulder-joint for trauma is a far less common operation in civil, than in military, practice. It is performed for gunshot-wounds, compound dislocations, tuberculous disease, and tumors of the head and upper portion of the humerus.

*Operation by Anterior Incision.*—The patient lies supine; a pillow is placed beneath the shoulders, and a sand-pillow is put beneath the shoulder to be operated upon. The arm is held to the side with the outer condyle forward and the bicipital groove inward (Barker's directions). The surgeon stands by the affected side. An incision 3 or 4 inches in length is made from just external to the coracoid process of the scapula, running straight down the humerus (Fig. 435, A). This incision divides the border of the deltoid muscle and brings into sight the long head of the biceps. The tendon of the biceps is retracted inward, unless it is diseased, in which case it is resected. The knife is carried up the groove and opens the capsule of the joint. The periosteum is lifted from the neck of the bone while an assistant rotates the elbow to make the muscles tense. In some places, if the periosteum tears, muscular insertions must be cut with a knife. The head of the bone is sawn off while the bone is in place, or the elbow is strongly pulled back, and the head of the bone is forced out of the wound, and is then sawn off at the point required. In ordinary cases only the articular head is removed; in other cases the section is made just above the surgical neck; in yet others a portion of the shaft must also be cut away. If the glenoid cavity is found slightly diseased, the dead bone must be removed by the chisel and mallet or by the cutting forceps. If the cavity is seriously diseased, the entire glenoid should be removed. Scrape away all damaged tissue; ligate bleeding points; irrigate the wound with corrosive sublimate solution; swab it out with a solution of chlorid of zinc (20 gr. to 1 oz.); dust with iodoform; close the upper portion of the wound and insert a drainage-tube in the lower angle; dress the wound antiseptically; place a small pad in the axilla; apply the second roller of Desault; and put the patient in bed with a pillow under the affected shoulder. In seven days the hand-sling is substituted for the bandage, and with the elbow hanging free the patient is permitted to get up and is advised to move his arm frequently. Drainage is maintained until the wound is well healed from the bottom. Great limitation of movement inevitably follows a shoulder-joint resection.

*Excision by the deltoid flap* is performed when the head of the bone is much enlarged (as by a tumor) or when the tissues are thick and indurated. The deltoid flap is in the shape of a U or is semilunar (Fig. 436, A). Raising this flap exposes the head of the bone most satisfactorily. Bell states that when the glenoid cavity is chiefly involved the incision should be posterior (Fig. 436, B).

*Senn's Method.*—Senn[1] described an incision which does not damage important vessels, muscles, tendons, or nerves, and which is followed by good functional results. A semilunar skin-flap is formed, the incision running from the coracoid process to the posterior border of the axillary space. The flap is turned up, exposing the upper half of the deltoid muscle. The acromion is sawn off and turned down with the attached deltoid. The capsule is now freely exposed; it is opened, and either arthrectomy or excision is performed, according to conditions. In closing the wound it is not necessary to bore the acromion and pass silver wires to join the fragments; it is enough to suture the periosteum with catgut.

[1] "Phila. Med. Jour.," Jan. 1, 1898.

**Excision of the Elbow-joint.**—This operation was suggested by Park in 1782, but the first employment of it was by Moreau in 1794. It is performed for wounds, faulty ankylosis, and chronic articular disease. Excision must be complete. Endeavor to make a subperiosteal resection; this maintains the shape of the articulation and gives the best chance for a movable joint. The patient is "supine, but inclining to the sound side, the affected arm being held almost vertical, with the forearm flexed and nearly horizontal" (Barker). The incision is made on the posterior surface of the joint. A single posterior incision is usually employed (Fig. 436, D). An incision is made a little internal to the long axis of the olecranon, beginning 2 inches above and terminating 2 inches below the tip of the olecranon. This incision goes down to the bone, and throughout the entire operation the surgeon must guard

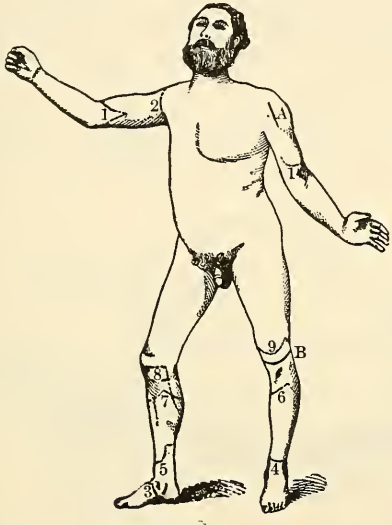



Fig. 435.

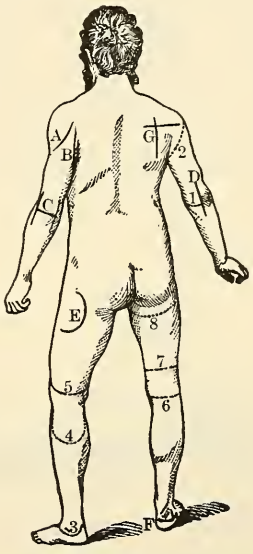



Fig. 436.

Fig. 435.—1–9, Amputations (Joseph Bell): 1, 1, of arm by double flaps; 2, at shoulder-joint; 3, at ankle-joint by internal flap (Mackenzie's); 4, 5, of leg just above the ankle-joint (Syme's); 6, 7, below the knee (modified circular); 8, through condyles of femur (Syme's); 9, at lower third of thigh (Syme's). A, excision of head of humerus; B, of knee-joint (semilunar incision).

Fig. 436.—1–8, Amputations (Joseph Bell): 1, at elbow-joint (posterior flap); 2, at shoulder-joint, posterior incision (first method); 3, at ankle-joint (Mackenzie's); 4, through condyles of femur (Syme's); 5, at lower third of thigh (Syme's); 6, at knee (posterior incision); 7, of thigh (Spence's); 8, at hip-joint. A–G, Excisions: A, excision of shoulder-joint (deltoid flap); B, of shoulder-joint (posterior incision); C, of elbow-joint (H-shaped incision); D, of elbow-joint (linear incision); E, of hip-joint (Gross's); F, of os calcis; G, of scapula.

and shield the ulnar nerve. The periosteum and soft parts are well separated; the olecranon is sawn off; forced flexion exposes the joint-cavity freely, and enables the surgeon to lift the periosteum and soft parts from the humerus; the humerus is sawn through at the beginning of its condyloid processes; the radius and ulna are cleared and are sawn at a level below that of the base of the coronoid process of the ulna. Diseased tissues are cut and scraped away; the wound is irrigated, sutured, drained, and dressed. In some cases an H-shaped incision is employed (Fig. 436, C), but the cicatrix of a transverse cut will limit flexion of the limb.

After excision of the elbow the patient is put to bed and the arm is laid upon a pillow, the elbow being placed midway between a right angle and complete extension, the forearm being placed midway between pronation

and supination. No splint is used, as a rule. Esmarch used the splint shown in Fig. 437. The aim in treatment is to obtain a freely movable joint. Passive motion is begun in one week, at which time the patient gets up. The hand is carried in a sling for a time after healing of the wound is complete.

**Excision of the Head of the Radius.**—This operation is practised for irreducible dislocation of the radius. An incision is made through the supinator longus muscle down to the head of the radius, and the neck of the bone is divided by means of a Gigli saw or bone-cutting forceps. The musculospiral nerve lies to the inner side. Some bone is always taken from the external condyle in order to make a sufficient gap to prevent subsequent ankylosis ("The Operations of Surgery," by Jacobson and Rowlands).

**Excision of the Wrist-joint.**—This operation was first performed by Moreau in 1794. Bell states that, whatever method of excision is chosen, three cardinal rules must be borne in mind: (1) remove all the diseased bone, including the portions of the radius, ulna, carpus, and metacarpus which are covered with cartilage; (2) interfere with the tendons to the least possible degree, and (3) begin passive motion of the fingers very early. Many surgeons prefer the simple gouging away of diseased foci and the scraping of sinuses instead of a formal resection of the wrist, amputation being employed in severe cases or when scraping fails after several trials. Formal excision is not frequently performed, and the results cannot be regarded as very favorable.

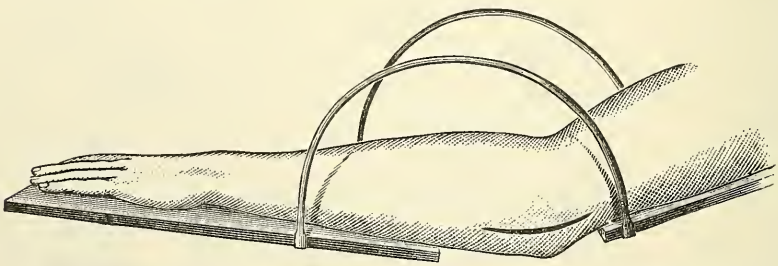


Fig. 437.—Esmarch's splint for the treatment of a limb after excision of the elbow-joint.

*Lister's Open Method of Excision.*—Break up adhesions as completely as possible by forcible movements. Apply a tourniquet or an Esmarch apparatus. The patient lies upon his back, the arm and the forearm being brought, from stage to stage, into the most desirable positions. Begin an incision over the middle of the dorsum of the radius, on a level with the styloid process; carry it downward in the direction of the inner edge of the articulation of the thumb with its metacarpal bone, and when the knife reaches the radial side of the second metacarpal bone alter the direction of the incision and carry it downward in the long axis of the metacarpal bone to about its middle (Fig. 438, A). This is known as the *radial incision*, and the only tendon divided is that of the extensor carpi radialis brevior muscle. The tissues upon the radial aspect of the incision are dissected up, the tendon of the extensor carpi radialis longior muscle is divided at its point of insertion (Bell), and all the soft structures are retracted outward, exposing the trapezium, which is cut off from the rest of the carpus, but which is left in place, as its removal at this stage endangers the radial artery (Barker). By extending the hand the tendons are loosened and the carpus is cleared in the direction of the ulnar border of the hand.

Another incision is made, starting upon the inner surface of the wrist, 2 inches above the articular surface of the ulna, and midway between the ulna and the flexor carpi ulnaris tendon. This incision, which is known

as the *ulnar incision*, is carried down until it is opposite the middle of the fifth metacarpal bone in the palm (Fig. 438, B). "The dorsal lip of this incision is raised" (Bell), and the extensor carpi ulnaris tendon is divided and dissected from its depression, but is not separated from the integument. The extensor tendons are lifted; the ligaments upon the dorsum and sides of the wrist-joint are cut; the flexor tendons are raised from the carpal bones; the pisiform bone is cut from the carpus, but is not yet removed; and the unciform process of the unciform bone is cut with forceps. The anterior radiocarpal ligament is divided, the carpometacarpal articulations are cut through, and the carpus is pulled out with bone-forceps. The ends of the radius and ulna are forced out of the ulnar incision. All that portion of the ulna which is crusted with cartilage is to be removed, the saw-cut is to be oblique, and the base of the styloid process is to be left behind. A thin section is to be sawn from the radius, and the tendon-grooves are not to be impinged upon. The articular surface of the ulna is cut away by pliers (Bell).

Fig. 438.

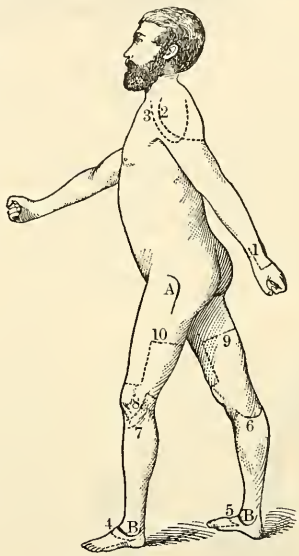



Fig. 439.

Fig. 438.—1–18, Amputations (Joseph Bell): 1, amputation at wrist-joint (dorsal incision); 2, at wrist-joint (palmar incision); 3, at forearm (dorsal incision); 4, at forearm (palmar incision); 5, at elbow-joint (anterior flap); 6, at arm (Teale's); 7, at shoulder-joint (first method); 8, 9, of metatarsus (Hey's); 10, 11, at ankle (Syme's); 12, 13, of leg, posterior flap (Lee's); 14, at knee-joint (Carden's); 15, of thigh (B. Bell's); 16, of thigh (Spence's); 17, of thigh in middle third; 18, 18, at hip-joint. A, excision of wrist (radial incision); B, of wrist (ulnar incision).

Fig. 439.—1–10, Amputations (Joseph Bell): 1, of lower third of forearm (Teale's); 2, at shoulder-joint by large postero-external flap (second method); 3, at shoulder-joint by triangular flap from deltoid (third method); 4, 5, through tarsus (Chopart's); 6, 7, at knee-joint; 8, by single flap (Carden's); 9, 10, of thigh (Teale's). A, excision of hip; B, B, of ankle-joint (Hancock's incision).

If foci of disease are discovered beyond these points, they are to be gouged out. The ends of the metacarpal bones are sawn off, and their articular facets are cut away by means of pliers. The trapezium is dissected out, the end of the first metacarpal bone is sawn off and its facet is cut away with pliers, and a portion of the pisiform bone is removed (the entire bone being removed if it be diseased). The wound is irrigated, vessels are tied, the radial incision is closed, the ulnar incision is partly closed, a drainage-tube is inserted by way of the ulnar incision, the wounds are dressed antiseptically, and the Esmarch apparatus is taken off. The forearm and hand are placed upon a splint which immobilizes the wrist and leaves the fingers semiflexed. Passive motion of the fingers is begun after thirty-six hours. The splint is worn for many months

until the wrist-joint is immobile and solid. Esmarch uses the splint shown in Fig. 440.

**Excision of Metacarpal Bones and of Phalanges.**—Excision of a metacarpal bone, except in cases of necrosis with the formation of large quantities of new bone, usually leaves a useless finger; hence amputation is usually preferred to excision. This rule does not apply to the metacarpal bone of

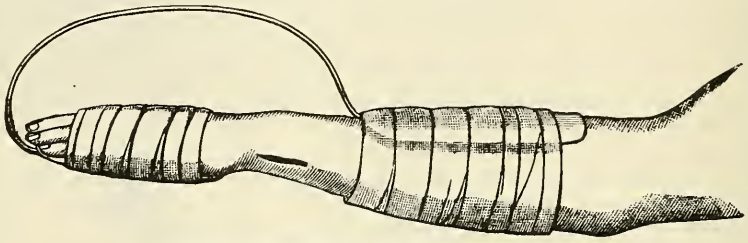


Fig. 440.—Esmarch's interrupted splint applied.

the thumb, which is occasionally excised. The incision for this operation is made upon the dorsum, and is straight. Excision of the proximal phalanx of the thumb is sometimes performed. Excision for disease is rarely performed upon the finger-joints, amputation being preferred, though the operation is sometimes undertaken for compound dislocation. In the metacarpophalangeal joint of the thumb excision, if it can be performed, is preferred to amputation. The incision for resection of this joint is placed upon the radial aspect.

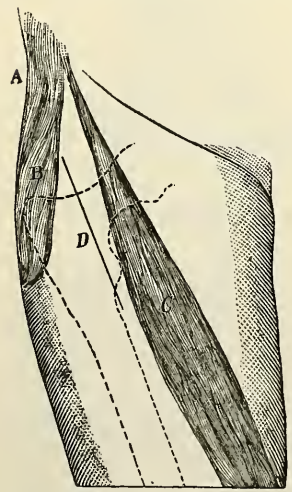



Fig. 441.—Excision of the hip-joint: A, Gluteus muscle; B, tensor vaginæ femoris muscle; C, sartorius muscle; D, anterior incision.

**Excision of the Hip-joint.**—Treves and Jonathan Hutchinson, Jr. ("Manual of Operative Surgery"), tell us that the operation was first performed by Anthony White, of the Westminster Hospital, in 1818. Sir William Fergusson established the operation in surgical confidence. Some surgeons advocate this operation; others, notably Marsh, are emphatically opposed to it. Excision should be performed in the early stage of tuberculous disease *if less radical treatment has failed.* In this stage the usual position of the limb is one of flexion, abduction, and eversion. In cases of long duration, especially where dislocation exists, excision is an easy and a comparatively safe operation; in recent cases it is difficult and carries with it decided dangers, but the peril of delay may be greater than the peril of an early excision. In cases of hip disease with involvement of the acetabulum the mortality is 50 per cent., whether operation is or is not attempted. Excision is performed especially for tuberculous disease and for gunshot-injuries.

*Operation by Anterior Incision* (Fig. 441) (*Barker's Operation*).—In this operation the patient is supine, with the thighs extended as thoroughly as circumstances permit. The surgeon stands to the right of the patient. An incision is begun $\frac{1}{2}$ inch below and $\frac{1}{2}$ inch external to the anterior superior iliac spine, and it is carried downward and a little inward for about 3 inches (Fig. 441, D). If dislocation exists, the incision need not be so long. This incision is carried at once deeply between the muscles, and the capsule of the joint is

opened. The neck of the bone is divided from its upper surface downward by a saw or an osteotome, and without dislocating the bone through the wound by forcible extension and eversion. The head of the bone is removed. All tuberculous foci must be scraped away, and the flushing gouge is used upon tuberculous areas of the acetabulum. All sinuses should be thoroughly scraped. Bleeding is arrested, the wound is irrigated with normal salt solution, mopped with chlorid of zinc solution, and dusted with iodoform. A drainage-tube is inserted at the lower angle of the incision, and the upper portion of the cut is closed. The wound is dressed antiseptically. Extension is made by the extension apparatus until healing has obtained good headway, when a double Thomas splint is applied, so that the patient may be taken out daily in the air and sunlight. As a rule, rigid ankylosis results from resection of the hip, but occasionally a joint results with a small range of movement.

*Operation by Lateral Incision (Langenbeck's Operation).*—In this operation a straight incision 2 inches in length is made in the direction of the axis of the femur, and passing downward from the apex of the great trochanter. From the beginning of this incision a curved incision is carried toward the head of the bone, the convexity of the curve being backward (see Fig. 439, A). Bell advises the use of the saw after bringing the head of the bone into the wound by abduction and eversion of the thigh. Barker applies the saw with the bone *in situ*, and strongly opposes wrenching the bone out of the incision because of the danger of peeling off the periosteum, which peeling, if it takes place, favors necrosis.

*Incision of Gross.*—In Gross's operation a semilunar flap is made with the convexity backward (see Fig. 436, E).

**Excision of the Knee-joint.**—The complete operation was first performed by Park, of Liverpool, in 1781. In this operation a complete excision should be performed, and the patella ought to be removed. The operation is performed for tuberculous disease, some compound fractures and compound dislocations, and some cases of angular ankylosis. It is rarely employed for gunshot-injuries.

*Operation by Anterior Semilunar Flap.*—The patient lies upon his back, and the joint, if not ankylosed in extension, should be semiflexed. The surgeon stands to the right side. An incision is made which at once opens the joint. The incision begins at one condyle and reaches the other condyle by a curve which passes through the ligamentum patellæ midway between the tuberosity of the tibia and the inferior margin of the patella (see Fig. 435, B). The flap is dissected up, the knee is thrown into forced flexion, the lateral ligaments and crucial ligaments are cut, and the end of the femur is well cleared. The blade of Butcher's saw is passed beneath the bone, which is sawn from below upward (Ashhurst). The end of the tibia is cleared and a portion is sawn off. If, after sawing, diseased foci are discovered, another section can be sawn off or the foci can be gouged away. Ashhurst, who had a vast experience with this operation, insisted that in sawing through the femur the natural obliquity of the bone must be borne in mind and the section must be made in "a line parallel to that of the free surface of the condyles." If the section is made transverse to the axis of the femur, "the limb, after adjustment, will be found to be markedly bowed outward." The same surgeon said that the epiphyseal line is somewhat higher on the front than it is on the back of the femur, and in consequence the following rule is formulated for section of the condyles: the section of the condyles should be "in a plane which, as regards the axis of the femur, is oblique from behind forward, from below upward, and from within outward." Ashhurst advocated section of the tibia "in a plane transverse to the long axis of the bone, with a slight anteroposterior obliquity, so as to correspond with that of the section of the condyles," and he further says that the

patella must be removed whether it is diseased or not, and quotes Péniére's observations to the effect that excision of the patella diminishes the risk of death one-third, and its retention doubles the probability of an amputation becoming necessary in the future.

After removing the patella the diseased synovial membrane is clipped away with scissors and all sinuses and diseased territories are well curetted. The posterior ligament of the joint is not removed unless it is diseased; its retention prevents displacement and guards the popliteal space. In some cases tenotomy is required to permit extension. In children the fragments should be wired together; in adults this need not be done. After hemostasis, irrigate by salt solution, insert a drainage-tube, suture, dress antiseptically, and adjust the limb upon Price's splint or Ashhurst's bracketed wire splint. Instead of the bracketed splint, a long fracture-box may be used. If the femur tends to project anteriorly, use an anterior splint. If there be a tendency to outward bowing, adopt Ashhurst's expedient of carrying a strip of adhesive plaster around the outside of the limb and fastening it to the inner side of the splint. The splint is kept on until bony union is complete, as in this operation a movable joint is never sought. Many surgeons use a fenestrated or interrupted plaster-of-Paris splint, which is employed until the parts have become apparently firm and solid (Fig. 442). Even for many months after the parts have apparently

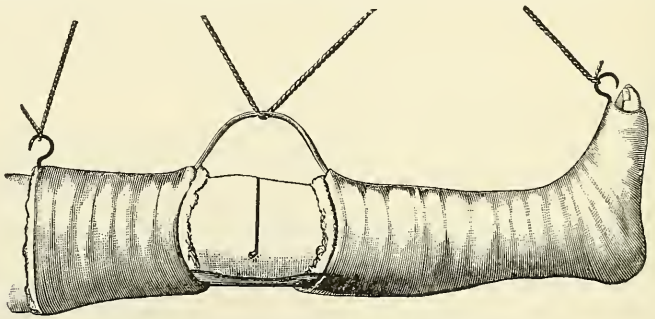


Fig. 442.—Watson's plaster-of-Paris swing-splint.

become solidly united bending may occur. How long fixation should be used has been much debated. In order to avoid the danger of flexion or other deformity fixation by some form of apparatus should be maintained for "at least a year and probably nearly two years after excision" (J. Torrance Rugh, "Am. Jour. Orthopedic Surgery," Feb., 1909).

**Excision of the Ankle-joint.**—Excision of the ankle was first performed by Moreau in 1792. This operation is performed chiefly for gunshot-wounds, compound dislocations, and in some cases of tuberculous joint disease. Excision of the ankle is an operation which is seldom performed.

*Operation by Hancock's Method.*—In this operation the patient lies upon his back, the foot rests upon its inner side, and the surgeon stands to the outer side of the damaged limb. Begin an incision just behind and 2 inches above the external malleolus, and carry it across the front of the joint to a corresponding point above and behind the internal malleolus (see Fig. 439, B); this incision goes only through the skin, and the flap thus marked out is reflected. "Cut down upon the external malleolus, carrying the knife close to the edge of the bone both behind and below the process, dislodge the peronei tendons, and divide the external lateral ligaments" (Joseph Bell). Cut the fibula 1 inch above the malleolus by means of pliers; divide the tibiofibular ligament; turn the foot upon its outer side; dissect from their

habitat back of the inner malleolus the tendons of the posterior tibial and the common flexor of the toes; carry the knife around the inner malleolus close to the bony edge; separate the internal lateral ligament, and dislocate the lower end of the tibia through the wound by turning the sole of the foot downward; saw off the lower end of the tibia and the articular process of the astragalus, sawing away from the tendo Achillis, and removing the fragments by bone-forceps. Cut away diseased synovial membrane, and curet all sinuses and tuberculous areas. Arrest bleeding, irrigate, and drain. Sew up the wound, insert a tube at the outer angle, and cause it to emerge at the inner angle. Apply antiseptic dressings, and put up the foot in fixed dressing or in splints at a right angle to the leg (Fig. 443). In Langenbeck's operation the excision is subperiosteal. If, in an excision of the ankle-joint, the astragalus is found extensively diseased, remove the entire bone.

**Excision of the Os Calcis.**—In caries limited to the os calcis most surgeons prefer to gouge away the dead bone, leaving the periosteum and, if possible, a shell of healthy bone, and draining thoroughly. Others advocate excision in some cases. Extensive disease limited purely to the os calcis is rare, and most surgeons advise gouging for limited caries, and Syme's amputation in the event of the disease extending beyond the periosteum or reaching adjacent bones.

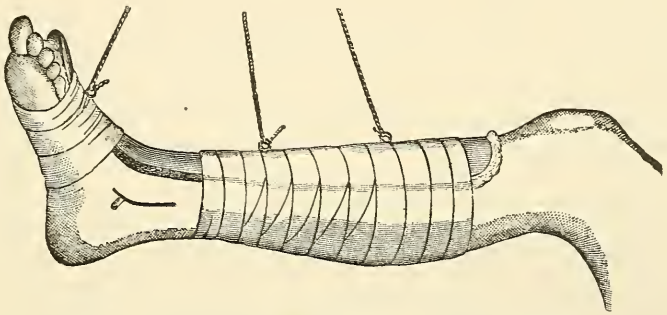


Fig. 443.—Volkmann's dorsal splint for excision of the ankle.

*Operation by Subperiosteal Method.*—In this operation the position assumed by the patient is supine, with the leg extended and the foot resting on its inner side. The incision, which cuts the tendo Achillis and reaches the bone at once, is begun at the upper border of the os calcis and the inner margin of the tendo Achillis, and is taken outward and horizontally forward to a point in front of the calcaneocuboid articulation (see Fig. 436, F). A vertical incision is begun near the forward termination of the initial incision, is carried across the outer edge and plantar surface of the foot, and terminates at the external margin of the inner surface of the os calcis. Some surgeons carry the vertical incision a little upward, toward the dorsum. The periosteum is entirely stripped by an elevator, the os calcis is removed, the cavity is packed with iodoform gauze, the wound is stitched, a drain is inserted posteriorly, the foot is dressed antiseptically, is placed at a right angle to the leg, and plaster of Paris is applied, trap-doors being cut for drainage.

**Astragalectomy,** or **excision of the astragalus,** is seldom performed. Astragalectomy is employed occasionally for relapsed and inveterate cases of club-foot. The indications are pointed out by Willard ("International Clinics," vol. iii, 12th series): "(1) Adults with great bony deformity; (2) neglected children of five to fifteen years, who have markedly distorted their tarsi by locomotion; (3) relapsed cases which have resisted the milder forms of operation, or which have been neglected by parents after previous operation; (4)

only occasionally, young children in whom from infancy the bones of the foot have been exceedingly rigid and unyielding, and where there is practically but little motion either at the ankle-joint or in the tarsus."

*Operation by the Subperiosteal Plan.*—Barker advises an incision going at once to the bone, from the "tip of the external malleolus forward and a little inward, curving toward the dorsum of the foot." The foot is extended and turned inward, the periosteum is lifted, the astragalus is removed, and the wound is treated and the foot is dressed as is done after excision of the os calcis.

In cases of *paralytic calcaneus* Whitman ("Orthopedic Surgery") recommends removal of the astragalus through a curved external incision; freeing the malleoli and making new sockets for them beside the cuboid and scaphoid bones. He puts the foot in marked plantar flexion and holds it by plaster.

**Excision of the Metatarsophalangeal Articulation of the Great Toe.**—In this operation make a lateral incision and cut off or saw off the proximal end of the first phalanx and the distal third of the first metatarsal bone. (See Mayo's Operation for Bunion, page 691.)

**Excision of the Metatarsal Bone of the Great Toe** (*Butcher's Method*).—In this operation a lateral straight incision is made, the periosteum is elevated, and the shaft is sawn from each extremity and removed.

**Excision of the clavicle** may be required for dislocation, caries, necrosis, gunshot-wound, tumor of this bone, as a preliminary to ligation of the artery and vein in certain cases of amputation at the shoulder-joint, or in cases of removal of the entire upper extremity. In excision of the clavicle the position of the patient is the same as that for ligation of the third part of the subclavian artery (see page 475). An incision is made down to the bone, from the sterno-clavicular joint to the acromioclavicular articulation. If the case is suitable, the periosteum is stripped and the bone is sawn and removed; if not, the bone is sawn and each half is separately disarticulated. The wound is sutured and dressed, and the limb is put up in a Velpeau bandage. McCreary, of Kentucky, in 1811 performed the first complete excision of the clavicle.

**Excision of the Scapula.**—Complete excision of the scapula is usually performed for tumors. Partial excision requires no detailed description. In excision of the scapula the patient lies upon his sound side. Treves suggests the following incisions: one outside the vertebral border of the scapula, from its superior to its inferior angle; another from over the acromioclavicular joint, along the acromion process and spine of the scapula, to meet the first incision. Syme used an incision carried transversely inward from the acromion process to the vertebral border of the scapula, and another cut directly downward from the center of the first incision (see Fig. 436, G). In the method of Treves[1] the upper flap is reflected and the trapezius muscle is divided; the lower flap is reflected and the deltoid muscle is divided. The patient's hand is placed on the sound shoulder; the muscles of the vertebral border are divided, the posterior scapular artery is tied, and while the vertebral border of the scapula is pulled toward the surgeon, the serratus magnus muscle is cut, the upper border of the shoulder-blade is cleared, and the suprascapular artery is tied. The hand is now brought down to the side; the acromioclavicular joint is disarticulated; the conoid and trapezoid ligaments are divided; the muscles of the coracoid process are cut; the capsule is incised, with the supraspinatus and infraspinatus, the subscapularis, and the scapular origins of the biceps and triceps muscles; and finally the teres major and minor muscles are divided, the subscapular artery is tied, and the bone is removed. The wound is stitched, a drain is introduced, and antiseptic dressings are applied. The patient lies upon his back until healing is well under way, when the arm is placed in a sling.

[1] "Manual of Operative Surgery."

The drainage-tube may be removed in twenty-four hours. Langenbeck, of Berlin, in 1855 performed the first complete excision of the scapula.

**Excision or Resection of a Rib** (Fig. 572).—In *caries* the gouge and rongeur may remove the disease. In other cases excision is performed. In this operation the patient lies upon his sound side unless the operation is performed for empyema, in which case he lies on his back or only partly on the sound side. (See Empyema, Operation for.) The surgeon faces the patient. Make an incision down to the bone, in the long axis of the rib. The periosteum, if not diseased, is lifted from the bone, and the intercostal artery is lifted out of the way with the periosteum and is thus saved from being cut. After dividing the bone beyond the limits of disease, remove it. During the sawing a metal retractor is held beneath the rib, between the rib and the periosteum. It is better to saw it than cut it with ordinary biting forceps, because the latter splinters the bone. The author usually uses a forceps known as a *costotome*, which cuts the rib without splintering. If the periosteum is diseased, remove it after tying the intercostal artery. It should be removed in a case of empyema, otherwise bone-formation may interfere with drainage. In *empyema*, after removing the periosteum, open into the pleural cavity, allow pus to flow out slowly, remove fibrinous masses, employ a finger to feel if there are adhesions and if the lung will probably expand, and insert a drainage-tube. In resection for rib disease curet sinuses and pack with iodoform gauze for some days. Sew up the wound except at one end. Dress antiseptically and apply a binder. (See Operations Upon the Chest and Estlander's Operation.)

In *removing a cervical rib* make an incision along the posterior edge of the sternocleidomastoid, avoid the pleura, subclavian vessels, and brachial plexus, and remove the periosteum with the rib in order that the bone will not be reproduced.

**Complete Excision of One-half of the Upper Jaw.**—The whole upper jaw has been removed, but in what follows only resection of one-half the jaw will be described. This operation is performed for malignant tumors of the superior maxillary bone or its antrum. Up to 1826, at which time Lizars, of Edinburgh, suggested the operation, tumors of the antrum were treated by scraping them away with a sharp spoon. Gensoul, of Lyons, in 1827 performed the first operation for resection of the upper jaw. Heyfelder, in 1844, removed both superior maxillary bones. Excision of a superior maxillary bone is not justifiable except as a palliative measure, if the orbit is invaded, if the skin and subcutaneous tissues are infiltrated, or if the disease extends widely beyond the superior maxillary and palate bones.

**Preliminary Closure of the External Carotid Artery.**—Some surgeons ligate the external carotid artery or compress it temporarily. In a number of excisions of the upper jaw I have always found the hemorrhage readily controllable as soon as the bone is removed, and have never felt it necessary to resort to preliminary ligation or compression.

*Operation by Median Incision.*—The patient, whose face has been shaved, is placed in the Trendelenburg position, thus avoiding the possible need of instant tracheotomy; or, what is even better, he lies horizontal or with the head a little raised and takes ether by intratracheal insufflation (see page 1199). The surgeon stands to the right side of, and faces, the patient. The incisor tooth on the diseased side is pulled out. The incision, which is really Weber's incision (called by some Nélaton's, by some Fergusson's, by some Liston's) (Fig. 444, line A–B), is begun $\frac{1}{2}$ inch below the inner canthus of the eye, and is carried along the side of the nose, around the ala of the nose, by the margin of the nostril, and through the middle of the lip. While the lip is being incised the assistant arrests hemorrhage by grasping the corners of the mouth, and after the lip has been divided the coronary

arteries are at once ligated. Some operators approach the mucous membrane cautiously and ligate the vessels before opening the cavity of the mouth. The upper portion of the wound having been compressed by another assistant during these manipulations, pressure is now removed and bleeding points are ligated. Another incision is now carried outward from the beginning of the first incision, along the orbital margin to well over the malar bone. The flap is lifted from the periosteum, and the bleeding from the infra-orbital artery and the small vessels is restrained by pressure. The nasal cartilage is separated from the bone, and the nasal process of the superior maxillary is sawn (line A–B, Fig. 445). The orbital periosteum is lifted up, and the orbital plate is cut by forceps from the saw-cut in the superior maxillary bone to the sphenomaxillary fissure (line B–C, Fig. 445). The malar bone is sawn or is bitten through about its center, the cut running into the sphenomaxillary fissure and taking a downward and outward direction (line C–D, Fig. 445). The soft parts covering the hard palate are incised in the median line, a corresponding incision is made along the floor of the nose near the septum, and the soft palate is separated from the hard palate by a transverse cut.

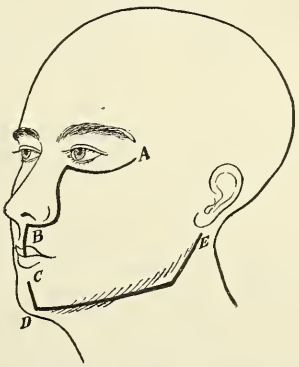



Fig. 444.—*A–B*, Incision of the soft parts preliminary to excision of the upper jaw; *C–D–E*, incision of soft parts preliminary to excision of the lower jaw.

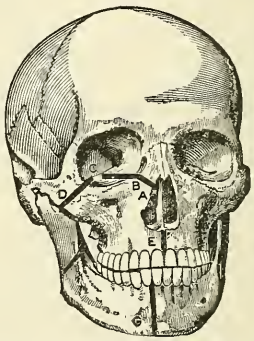



Fig. 445.—1, Excision of the upper jaw: A–B, Section of the nasal process; B–C, section of the orbital plate; D, section of the malar bone and orbital plate; E, section of the alveolus and hard palate. 2, Excision of the lower jaw: G, Section of the inferior maxillary; H, section of the ramus in partial resection.

The saw is introduced through the nose, and the palate is sawn (line E, Fig. 445). The upper jaw-bone is grasped by Fergusson's lion-jaw forceps and removed, the removal being aided by the use of the scissors and bone-cutters; the latter are used to separate the upper jaw from the pterygoid process (Treves). Every vessel that can be seen is tied, and severe bleeding from bone is arrested by antiseptic wax. Oozing is controlled by hot water and pressure or by Paquelin's cautery. Examine carefully to see if all the diseased area is removed; if it is not, use the gouge, scissors, chisel, and saw until healthy tissue is reached. The wound is packed with iodoform gauze, and the end of the strip is so placed as to be accessible through the mouth. The wound is sutured (the mucous membrane of the lip must be stitched, as well as the skin) and is dressed antiseptically (the eye being protected by aseptic gauze), and a crossed bandage of the angle of the jaw is applied. After this operation it is common to have the eye sink to a lower level (causing double vision), to have persistent swelling of the lower lid, and to have the tears flow over the lid instead of down the duct.

**Excision of One-half of the Lower Jaw.**—In some rare instances the entire inferior maxillary bone is removed. The lesions necessitating removal of the

lower jaw are of the same nature as cause us to remove the upper jaw. The names of many surgeons are connected with this operation, viz., Deadrick, White, Dupuytren, Sir Astley Cooper, and Valentine Mott.

In this operation the patient is placed in the same position as for excision of the upper jaw, the chin having been previously shaved. A vertical cut is made through the chin-tissue, starting below the margin of the lip and reaching to below the border of the jaw (C–D, Fig. 444). From the point D an incision is carried outward below the border of the jaw and then back of the ramus, as shown in the line D–E (Fig. 444). Treves's advice is to carry this incision down to the bone, except at the line of the facial artery, at which point it must go through the skin only. The facial artery is now to be sought for, tied in two places, and divided. Except in malignant cases the periosteum is lifted from the external surface of the bone, from the symphysis outward. Hemorrhage is arrested. The buccal mucous membrane is cut from the alveolus. A lateral incisor tooth is pulled, and the bone is sawn in the line G (Fig. 445). The bone is grasped in a lion-jaw forceps and is drawn outward. The mylohyoid insertion is cut; the internal pterygoid muscle is cut or the periosteum at this spot is lifted; the inferior dental artery is cut and tied; the jaw is pulled down; the insertion of the temporal muscle upon the coronoid process is cut away, and the external pterygoid muscle is divided. The capsule of the joint is opened, and the bone is separated from the ligaments which still hold it in place. Bleeding is arrested, the wound is sutured, a tube is introduced in the posterior portion of the wound and retained for twenty-four hours, and antiseptic dressings and a Gibson or a Barton bandage are applied. Partial excisions of the alveolus may be performed through the mouth by means of chisels and rongeur forceps, and Wyeth has thus removed half of the jaw; but if any considerable part of the body of the jaw is to be removed, it is usually best to make an incision below the inferior maxillary.

**Partial Excision.**—This operation is frequently made necessary by epithelioma involving the periosteum or bone. After this operation, unless means are taken to prevent deformity, there will be asymmetry of the two portions of jaw bone remaining and malrelation of the teeth. Various attempts have been made to fill up this gap at the time of operation by some material, or to hold the two portions of the mandible in symmetrical relation by the insertion of trusses of silver wire, or by fastening each piece of bone to a metal plate.

John B. Murphy suggested the use of a bridge of silver wire. All of my trials with such a bridge failed. It always ulcerated out and usually through the skin, but did some good by keeping the bone ends further apart than they otherwise would have been.

Stanley Stillman ("Annals of Surgery," July, 1912) advocates bone-grafting as the only proper method, in order to prevent contraction during healing after the removal of the jaw bone. "When the teeth on the sound side are numerous and firm enough to stand the strain, they may be prepared beforehand with the aid of a dentist, so that a few days after the operation they may be clamped firmly to those of the upper jaw" (Ibid.). When the wound heals, Stillman separates the flap, freshens the bone ends, and wedges a section of rib between them. The clamps are left in place until the bones unite, which requires six to eight weeks. In cases in which the teeth cannot be used to clamp, a silver bridge is inserted, which is left in place until the parts are ready for the insertion of the bone.

**Barker's Operation for Dislocation of the Semilunar Cartilages of the Knee-joint.**[1]—Begin the incision over the ligament of the patella, ½ inch above the articular surface of the tibia, and carry it in a curve downward and outward to the anterior edge of the internal ligament. The periosteum should be divided

[1] "Lancet," Jan. 4, 1902.

by the cut. This incision forms a flap the lower edge of which is ½ inch below the border of the articular surface of the tibia. The flap is lifted until the cartilage is seen "under the attachment of the meniscus, which if partially attached will rise with the flap until its under surface is seen." If partially torn anteriorly it is stitched to periosteum by a few silk sutures. The periosteum is then stitched in place, no drain is used, the joint is immobilized, and for one week ice is kept upon the part. If the meniscus is found completely separated and curled up, it may, if the injury was recent, be reduced. If the injury was old and if the cartilage is shrunken, it should be completely cut away (Barker).

**Operation for Congenital Dislocation of Hip.**—*Lorenz's Bloodless Method of Reduction.*—The method of reducing by manipulation a congenital dislocation of the hip was advised by Paci and modified and improved by Lorenz. It has long been known that reduction is easy at birth because an acetabulum, though probably a shallow one, exists, and the head of the bone is not firmly held in its new situation. In an older child the problem is far more difficult, because, even if reduction is effected, the acetabulum may be extremely shallow or absent, and redislocation may readily occur. Lorenz aims to effect thorough reduction and then fixes the limb in abduction for months, so that the acetabulum will deepen and the bone will become firm in its proper socket. This operation is rarely successful in children over six years of age. The child is anesthetized and an attempt is made to draw the femoral head on to a line with the acetabulum. If the child has never walked, this is readily accomplished. If it has walked, the procedure may be very difficult, and it may be necessary to make extension by a fillet fastened above the knee, and counterextension by a screw and a perineal band. The drawing down of the head is made easier by stretching and massaging the adductor muscles. The next step is to strongly flex the thigh, rotate it a trifle internally, and then abduct it while flexion is maintained. This causes the head of the femur to pass around the posterior margin of the acetabulum and frequently produces reduction. "Full abduction being kept up, the thigh is rotated out, thus forcing the head of the femur more firmly into the socket." (See the description of the Lorenz method in J. Jackson Clarke's "Orthopedic Surgery.") The strongly abducted limb is put up in plaster of Paris. In about three months the plaster is removed, the abduction is diminished, the plaster is reapplied, and is retained for another three months. During the continuance of immobilization of the hip the child walks about, with the knees bent. When the plaster is finally removed, manipulation, massage, and exercise strengthen the muscles and give freedom to the joint. In a double dislocation one joint may be cured before the other is operated upon, or both may be operated upon at the same séance. In double dislocation plaster must be worn more than six months. The Lorenz operation is safe when applied to very young children, but has elements of danger which increase with the years of the subject. A patient may suffer grave lacerations of muscles and ligaments, and even vessels and nerves. Death

Fig. 446.—Lorenz method. Unilateral congenital dislocation of hip (reduced). Cast applied with leg in "frog position."

may result from shock, and extensive deep-seated hemorrhage may occur. In fact, it is a mistake to call it a bloodless method. The blood flows, though we do not see it. An untrained man may do fearful mischief by this operation, and it should only be attempted by an experienced, skilful manipulator and upon properly selected cases, when it is a very successful procedure. I am satisfied that, except in the case of a very young child, in whom reduction is easy, one who performs the Lorenz operation should be something more than skilful and experienced. He should be physically strong, so that traction and abduction will be powerful and steady. A weak man will jerk, will throw his weight upon the part, and will be apt to tear structures instead of stretching them. Sudden forcible movements are apt to break the bone.

*Hoffa's Cutting Operation.*—Make the external incision of Langenbeck to open the joint (see page 703). The capsule is incised at its insertion into the neck, and the periosteum and muscles are lifted from the great trochanter. Hoffa claims that in children less than five years of age the head of the bone can be readily replaced into the acetabulum by flexing the thigh and making direct pressure upon the head of the bone. After replacing the femoral head it is held in place while an assistant extends the leg in order to stretch the muscles. In children over five years of age cut with a tenotome the muscles which spring from the ischial tuberosity and also the adductors; cut the fascia lata and muscles which arise from the anterior superior iliac spine by incision; open the joint and liberate the head of the bone; remove the ligamentum teres; scrape out the acetabulum, removing "cartilage, fat, and considerable spongy tissue" (Tubby); and replace the head of the bone in the acetabulum. The limb is maintained in inversion, abduction, and extension for several weeks, when it is straightened. Massage and passive motion are begun in the fifth week. The patient now gets about, wearing an apparatus for many weeks. This apparatus permits the head of the bone to move in the socket, but prevents redislocation.

*Lorenz's Cutting Operation.*—This is a modification of Hoffa's. The muscles inserted into the greater and the lesser trochanter are not cut; the sartorius, the hamstrings, and the external portion of the fascia lata are cut (Tubby).

The incision of Lorenz is longitudinally from the anterior superior spine. Another incision is carried inward from this at the level of the lesser trochanter. The capsule is opened by a crucial cut; the acetabulum is enlarged; the head of the bone, if it remains, is inserted into the acetabulum; if there is no true head, a new one is formed and inserted into the cavity. The limb is immobilized in a position of moderate abduction. Massage and passive motion are begun in the fifth week, and are continued for months.[1]

**Reduction by Means of Mechanical Appliances.**—As the chief factor in the reduction of a congenital dislocation is thorough stretching of the muscles and ligaments before attempting replacement, numerous forms of levers have been devised to accomplish this. The best-known one is that of Bartlett, of Boston. It consists of a pelvic rest with perineal support, rods to fix the pelvis and traction rods to make extension. Great care is necessary in the use of all such appliances because of the danger of injury to vessels and nerves or of fracturing bony parts. After the parts have been thoroughly stretched replacement is accomplished by manipulation, as in the usual methods of manual reposition.

[1] I have drawn upon the very lucid description of these operations in A. H. Tubby's treatise upon "Deformities."

## XXI. DISEASES AND INJURIES OF MUSCLES, TENDONS, AND BURSÆ

**Myalgia,** or **muscular rheumatism,** is a painful disorder of the voluntary muscles and of the fibrous and periosteal areas where they are attached. The term "muscular rheumatism" is not strictly correct. It is possible that in some cases the muscular structure is inflamed, but it is certain that in many cases the pain is distinctly neuralgic. Muscular rheumatism may be due to cold and wet, to overexertion and strain, to acute infectious disorders, to syphilis, to chronic intoxications (lead, mercury, and alcohol), and to disturbances of the circulation. Gouty and rheumatic persons are especially predisposed, men being more liable to the disease than women. The disease is usually acute, but it may be chronic.

**Symptoms.**—Muscular rheumatism is apt to come on suddenly. The pain, which may be very acute and lancinating or dull and aching, is in some cases constantly present; in other cases it is awakened only by muscular contraction, and it is frequently relieved by pressure, though there is often some soreness. The skin above the muscle is sometimes tender to light pressure. The disease usually lasts for a few days, but it tends to recur. There is little, if any, fever.

*Lumbago* is myalgia of the muscles of the loins. *Rheumatic torticollis* is myalgia of the muscles of the neck. Usually one side of the neck is attacked. The chin is turned from the affected side and the neck is stiff. *Pleurodynia* is myalgia of the intercostal muscles. The pain is very severe, is aggravated by deep respiration, by coughing, and by yawning, there may be tenderness, and the patient tries to limit chest-movement. In *intercostal neuralgia* the pain is limited, is not constant, but occurs in distinct paroxysms, and is linked with the presence of the tender spots of Valleix. Pleurodynia lacks the physical signs of pleurisy. *Cephalodynia* is myalgia of the muscles of the scalp. The muscles of the shoulder, upper dorsal region, abdomen, and extremities may also be attacked by myalgia. Myalgia must not be confused with the pains of locomotor ataxia.

**Treatment.**—Remove any obvious cause. Treat any existing diathesis, such as gout or rheumatism. Rest is of the first importance. For lumbago, put the person to bed. For pleurodynia, strap the side of the chest. A hypodermatic injection of morphin and atropin into the affected muscles at once allays the pain, and a deep injection of distilled water is sometimes curative. Relief may be afforded by painting the surface with 30 drops of a mixture of equal parts of guaiacol and glycerin and covering the painted area with cotton. The introduction of four or five aseptic needles into the muscles, and their retention for a few minutes, sometimes acts most favorably. Ironing the skin above the painful muscles with a very warm iron, a piece of flannel being interposed, is a useful domestic remedy. Vigorous rubbing of the area with a piece of ice allays the pain. Hot poultices do good. If the pain is widely diffused, alters its seat, or is very obstinate, order hot baths or Turkish baths and administer diuretics. In chronic cases employ blisters or counterirritation by the cautery, give iodid of potassium and nux vomica, and have the patient take a Turkish bath every week. The constant electric current finds advocates. In an ordinary severe case order a hot bath, put the patient to bed with a hot-water bag over the part, and administer 10 gr. of Dover's powder; the next morning order to be taken four times daily a capsule containing 5 gr. of salol and 3 gr. of phenacetin, until the pain disappears. Citrate of potassium, citrate of lithium, chlorid of ammonium, or the salicylate of colchicin may be ordered instead of salol and phenacetin.

**Infective myositis** is a widespread inflammation of the voluntary muscles, due to an unknown infective cause. It is a disorder accompanied by pain and stiffness, by cutaneous edema, and by various paresthesiæ. Myositis resembles trichinosis, and can be distinguished from it only by spearing out a bit of muscle and examining it microscopically. Occasionally diffuse suppuration occurs.

**Ordinary myositis** arises from injuries, from syphilis, or from rheumatism, and it presents the usual inflammatory symptoms. Contraction and adhesions may follow. I operated upon a case of myositis of the rectus abdominis in a boy of eight. There was a large mass like a full bladder. There had not been an attack of typhoid and there was not hereditary syphilis. Caseation existed. The condition was possibly tuberculous, although no bacilli were found.

**Treatment of Myositis.**—Infective myositis is treated by anodynes, stimulants, nutritious food, hot applications, and rest. If pus forms, it should be evacuated. Rheumatic myositis calls for the administration of the salicylates, the alkalis, or salol. Syphilitic myositis is treated with mercury and iodid of potassium. The remedies employed for myalgia are used in traumatic myositis.

**Hypertrophy of the muscles** may arise from their increased use. In *pseudohypertrophic paralysis* the muscle is greatly augmented in bulk, but it contains less muscle-structure and more fat or connective tissue.

**Atrophy of the muscles** arises from want of use, from injury, from continuous pressure, from interference with the blood-supply, from disease of the nerves or their centers, or from lead-poisoning.

**Degeneration of Muscles.**—The muscles may undergo granular degeneration, waxy degeneration, fatty degeneration and calcareous degeneration, and may become pigmented.

**Local Ossification and Myositis Ossificans.**—It is not unusual for a small portion of bone to form in the periosteal insertion of a muscle which is subjected to frequent strain. In persons who ride many hours a day there not infrequently develops the *riders' bone*, which is an area of ossification in the adductor muscles of the thigh. *Myositis ossificans*, a widespread ossification of the muscles, is a rare disorder the cause of which is unknown, and which, though not congenital, usually begins in early life. In some local cases a traumatic origin seems certain. It is seen more often among males than females. Columns of inflammatory swelling and induration slowly develop, each column running in the direction of the muscular fibers, and ossification of the indurated columns takes place. It is stated that the thumbs and great toes shorten (J. Jackson Clarke's "Orthopedic Surgery"). In traumatic cases the condition is localized. I operated upon a traumatic case by removing an ossified area in the thigh muscles. The laboratory report was myositis ossificans. Growth quickly recurred after operation, but the new growth showed very little ossification. The laboratory report was spindle-celled sarcoma. This case and also another one reacted to Coley's fluid. It seems possible that local myositis ossificans may, in some cases at least, be a sarcoma in which rapid ossification occurs.

**Tumors of the Muscles.**—Primary tumors of the muscles are rare. Among those which may occur are sarcoma, fibroma, lipoma, osteoma, angioma, myxoma, and enchondroma. Most cases of supposed primary sarcoma of muscle are, in reality, cases of syphiloma (Esmarch).

**Syphilis** may cause inflammation. Gummata may form, or gummatous infiltration may take place.

**Trichinosis** or **trichiniasis** is a disease due to the embryos of the Trichina or Trichinella spiralis. Sir James Paget recognized the nature of the pre-

viously known encysted or larval form. It was long believed that the Trichina spiralis was a harmless parasite, but in 1860 Zenker, of Dresden, proved that it might be responsible for dangerous epidemics. The trichina is normally a parasite of the rat. Man is infected by eating flesh which contains trichinæ and has been insufficiently cooked. The flesh of the pig is the medium of infecting man. The pig becomes infected by eating rats suffering from the disease or offal and slaughter-house refuse containing trichinallized flesh. Dr. Joseph Leidy discovered the trichina in pork. People are most commonly affected by eating raw sausage, smoked sausage, or underdone pork. Imperfectly boiled ham may be responsible. Albert reported 14 cases from eating boiled ham. W. Gilman Thompson found records of 52 sporadic cases occurring in New York City during six years ("Amer. Jour. Med. Sciences," August, 1910). These nematodes are carried into the intestine, there to develop and multiply. In from seven to nine days a horde of embryos develop in the bowel, and leave the alimentary canal by passing through the peritoneum or by means of the blood, and finally reach the connective tissue of the muscles. From the connective tissue the embryos migrate into the primitive muscle-fibers, where they dwell and enlarge. Myositis develops, and in the course of five or six weeks the parasites become encapsulated and devlop no further. The cyst-walls may calcify and the worms may become calcified, or may live for years. The eating of infected meat is not inevitably followed by the disease, and a few embryos lodged in muscle may cause no symptoms.

The **symptoms** of trichinosis often appear in a day or two after eating infected meat. The symptoms of acute gastro-intestinal catarrh or of cholera morbus are common, but in some cases no gastro-intestinal manifestations usher in the disease. In from seven to fourteen days after the infected meat is eaten the migration of the parasites develops obvious symptoms. A chill may be noted; there is usually fever; muscular pain, tenderness, swelling, and stiffness are complained of. This condition may be widespread. Involvement of the muscles of mastication interferes with chewing; of the larynx, with talking and respiration; of the intercostals and diaphragm, with respiration. Skin-edema and itching are marked. In some cases delirium exists. The writer saw in the Philadelphia Hospital one fatal case which was mistaken for erysipelas because of the high fever, the delirium, and the edematous redness of the face and neck. Dyspnea is frequent. Mild cases get well in a week or two; severe cases may last many weeks. The mortality varies in different epidemics from 1 to 30 per cent. (Osler). The diagnosis is made by spearing out a piece of muscle, which is then examined for trichinæ under a microscope; or the worms may perhaps be detected in the feces by means of a pocket-lens. In a case under the care of the author, in St. Joseph's Hospital, there was no record of any attack of gastro-intestinal disturbance and the first manifestation was enlargement of the calf of the left leg. In most cases of trichinosis there is eosinophilia, but in the author's case, previously referred to, eosinophilia was not present.

**Treatment.**—To treat trichinosis employ purgatives (senna and calomel) early in the case, and give glycerin, and also santonin or filix mas. When muscular invasion has taken place, sedatives, hypnotics, nourishing diet, and stimulants are indicated.

**Ischemic Myositis, or Volkmann's Contracture** (*Volkmann's Paralysis; Ischemic Paralysis; Ischemic Muscular Atrophy, with Contractures and Paralysis*, Fergusson calls it).—It is occasionally noticed, particularly in children, after prolonged fixation of the forearm, especially after prolonged fixation of the elbow-joint by some appliance that impedes the freedom of circulation in the part. Contracture of the fingers occurs and per-

haps rigidity and flexion of the wrist. In 1875 Volkmann described severe contractures of the hand observed in some cases as a result of the use of tight bandages to hold splints in place in treating fractures of the arm. He believed that the condition was due to deprivation of arterial blood, that the muscles perished for want of oxygen, and that rigor mortis occurred. He pointed out that paralysis and contracture occur simultaneously, whereas in primary nerve lesion paralysis precedes contracture. The condition may come on after the application of an Esmarch band, after a severe injury in the neighborhood of the elbow-joint, may follow ligation of the main artery of a limb, venous embolism, venous thrombosis from injury or infectious disease, Raynaud's disease, or exposure to cold. One of Jones's cases followed a rapidly developing traumatic myositis ossificans; two followed crushes; in one an elastic tourniquet had been kept on a child's arm to prevent bleeding after an operation for webbed-fingers; in one pad pressure had been maintained for twenty-four hours to check bleeding ("Amer. Jour. Orthop. Surg.," April, 1908). A case of mine resulted from embolism of the brachial artery. There are two forms, one due to almost complete arterial ischemia, lasting for several hours at least; another due to interference with venous return. Volkmann's contracture is due to muscular degeneration, infiltration, induration, and contraction, the result of marked and prolonged arterial ischemia or interrupted venous return, and it is frequently spoken of as ischemic myositis (Dudgeon, "Lancet," Jan. 11, 1902). In some cases distinct neuritis with paralysis also exists. One characteristic of ischemic contracture is the rapidity with which it comes on. Dudgeon points out that in half a day, or even in less time in some cases, the symptoms appear, these symptoms being paralysis of the part with contracture. Pain is unusual, unless the nerves are seriously involved. In some cases the fingers and hand swell and become discolored. The absence of pain frequently prevents the recognition of the condition; therefore, the causative splint or bandage pressure may be maintained for days after the trouble has become serious. When the splints and bandages are removed and the forearm is examined, there is almost always tenderness over the muscles and the nerve-trunks; and in the majority of cases in which a splint was the cause a portion of the skin will have sloughed. Dudgeon points out the characteristic position of the deformity as follows: When the wrist is extended, the metacarpophalangeal joints are also extended; but the interphalangeal joints of the fingers and the terminal joint of the thumb are so strongly bent that the tips of the fingers touch the palm, and this position cannot be corrected by any justifiable amount of force. As soon as the wrist-joint is bent to a right angle, the interphalangeal joints can readily be extended. In a very severe case the wrist itself will become markedly flexed, and it will be impossible to extend it. The forearm is usually semiflexed and the hand pronated. The ulceration or sloughing so frequently present causes a *splint-sore.* There is always marked induration about a splint-sore. The flexor muscles themselves are indurated and usually wasted. The condition of sensation depends upon the state of the nerves of the part. When neuritis is absent, sensation will be normal; but in accordance with the amount of neuritis and degeneration there will be hyperesthesia, partial anesthesia, or complete anesthesia. A curious feature of these cases that is dwelt upon by Dudgeon and commented upon by Turner is the fact that in young children there is a cessation of growth of the bone. Robert Jones (Loc. cit.) reports that 19 cases out of 40 were associated with fracture. In 13 of the 19 cases there was pronounced malunion.

**Treatment.**—The old view of this condition was that it is practically hopeless. Anderson and Dudgeon, however, maintain that restoration may usually be obtained, the treatment consisting in regular, active motion, passive move-

ment, massage, and electricity. Forcible extension under ether is of no benefit whatever.

Jones's plan of treatment is very beneficial ("Amer. Jour. Orthop. Surg.," April, 1908).

Operative procedures on arms that necessarily have deficient circulation are hazardous, and Jones has discontinued all operative correction and relies upon purely mechanical and manipulative routine, as follows:

Five splints are cut out of zinc, tin, or sheet wire, to fit each finger and thumb. The wrist is forcibly flexed and held while each finger is separately splinted. It will be observed that in the fully flexed position of the wrist the fingers are all relaxed.

Fig. 447.—Rugh's splint for Volkmann's contracture. Splint applied. Looking at the palmar surface.

When the finger splints have been applied the wrist is released, and the patient is directed to systematically extend the metacarpophalangeal joints. In a few days the second splinting may usually be employed by embedding the entire hand to the wrist and the already splinted fingers in plaster of Paris, while the wrist is again flexed. Several days are devoted to systematic voluntary efforts at extension of the hand in a similar manner employed for the metacarpophalangeal joints.

The third splinting embraces that already employed and in addition embraces the wrist in the fullest extension possible, which in a few days may be increased until full extension is obtained. The latter position of full extension of wrist, hand, and fingers is maintained for some weeks until all contractible elasticity has disappeared.

It is usually observed that when the hand can be held in hyperextension without tendency to relapse, the circulation will almost invariably improve and the fingers resume their normal function and appearance except in cases of nerve destruction. Jones has found that in many cases in which the nerves had lost their function during contracture, the extension of the hand was the starting-point of recovery.

In making any splint pressure to straighten the fingers the greatest care must be exercised. The skin of the dorsum of the fingers will not endure prolonged pressure. I have used Rugh's splint with much satisfaction (Figs. 447, 448). It is best in the beginning to apply the splint for five or ten minutes twice a day. The time of each séance is gradually increased as the tissues develop resistance. Frequent bathing with alcohol aids the skin to bear pressure.

R. H. Sayre ("Volkmann's Ischemic Paralysis and Contracture," "Amer. Jour. Orthop. Surg.," Nov., 1908, p. 221) advocates the Jones method in all cases, inasmuch as cutting operations may be employed later if the results of Jones's treatment are not entirely satisfactory. The improvement in the

circulation and function obtained by the Jones method will make the operative field more capable of rapid recovery.

In a persistent and long-continued case an operation may be necessary. The operation may consist in dividing in the forearm the flexor muscles of the fingers, as advised by Davies Colley, and then, at a later period, dividing the flexor tendons. The objection to his procedure is that it destroys the capacity to flex the fingers for all time. Another suggestion has been to excise a piece from both the radius and the ulna, and wire the fragments together. The best surgical treatment is probably exposing the nerves, separating them from adhesions, stretching them, and then doing tendon-lengthening, but this should not be done until all the improvement possible to secure by conservative treatment has been obtained by at least three months of effort.

**Wounds and Contusions of the Muscles.**—*Wounds* of muscles may be either *open* or *subcutaneous*. In a longitudinal wound the edges lie close together, and hence drainage must be provided for by the surgeon. In a transverse wound the edges separate widely, and catgut stitches must be inserted. *Contusions* of muscles, like contusions of other tissues, vary in extent and in severity. There are pain (which is increased by attempts to use the muscle), loss of function, swelling beneath the deep fascia, and discoloration, which may appear at once because of superficial damage from

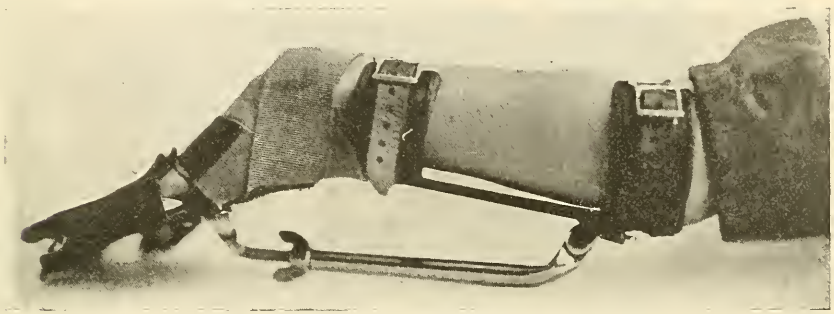


Fig. 448.—Lateral view of splint shown in Fig. 447.

the initial injury, or which may appear in dependent parts after many days because of gravitation of blood and blood-stained serum. As a result of contusion, suppuration, inflammation, or atrophy may arise.

**Treatment.**—In a longitudinal wound, drain; in a transverse wound, suture the muscle. The further indications in wounds and contusions of muscles are to obtain rest by means of splints and to secure relaxation. Limitation of swelling is secured by bandaging. Inflammation is combated first by cold and lead-water and laudanum; later by iodin, blue ointment, ichthyol, and intermittent heat. To prevent loss of function employ, as soon as the acute symptoms subside, massage, passive motion, and stimulating liniments, and, later in the case, electricity (galvanism if the reactions of degeneration exist; faradism, if they are absent).

**Strains.**—A muscular strain is a stretching of a muscle with a small amount of rupture. It is caused by traction in the long axis of the muscle. The muscle becomes swollen, tender, stiff, weak, and sore, and attempts at motion produce sharp pain. A strain of a tendon is a trivial or partial rupture. It leads to the development of acute thecitis, with fluid swelling and pseudocrepitation. Strains are common in the deltoid, the ham-string muscles, the back, the calf, the biceps, and the great pectoral. *Strain of the psoas muscle* causes pain on voluntary flexion of the thigh, and is associated

with tenderness in the iliac fossa. Strain of the right psoas may be mistaken for appendicitis, but it lacks the intense local tenderness, the abdominal rigidity, and the constitutional symptoms. *Lawn-tennis arm* is a strain of the pronator radii teres muscle. *Riders' leg* is a strain of the adductor muscles of the thigh. A strain of the long head of the biceps flexor cubiti produces the condition called by ball players a *glass arm.* A strain may be the only injury, or may be associated with some other condition (fracture of bone, dislocation, sprain, contusion, etc.). A strain may be followed by periostitis at the point of insertion of the muscle. Atrophy of the muscle occasionally follows a strain.

A strained muscle is usually rigid, is tender, and pains greatly when an attempt is made to use it. The skin over it, especially over its point of insertion, is usually tender.

A *strain of the back* is a very common accident, which is often associated with sprains of the vertebral articulations. There is great pain when the patient voluntarily straightens up. If the vertebral ligaments are not damaged, the patient can be straightened by passive motion without pain. The skin is tender in certain areas. The muscles are often rigid. There may be unilateral rigidity. In a back injury make a careful examination to be sure no damage has been inflicted upon the vertebræ or cord.

**Treatment.**—Relaxation by suitable position; rest by the use of splints or by putting the patient to bed; bandages for compression; hot fomentations or a hot-water bag, and ichthyol. As soon as acute symptoms subside, employ friction and massage. Strapping with adhesive plaster is of service in strain of the back and of the calf. If there is severe pain after a strain, administer Dover's powder, or even morphin.

**Rupture of Muscles and Tendons.—Rupture of a muscle** is announced by a sudden and violent pain and by loss of function, arising during powerful muscular contraction or strong traction in the long axis of a muscle. The rupture may be announced by a clearly audible snap (A. Pearce Gould). A distinct gap is felt between the ends; great pain develops on movement; there are tenderness, loss of power, and swelling. Rupture may be followed by atrophy, as is a contusion. Among the muscles which occasionally rupture we may mention the quadriceps, biceps, triceps, deltoid, plantaris, etc.

**Rupture of the biceps flexor cubiti or its tendon** is not very common; 72 cases have been collected (W. W. Keen, in "Annals of Surgery," May, 1905). It is much more common in men than in women. Loos's table of 66 cases contains records of only 2 women (Doane, in "Jour. Amer. Med. Assoc.," May 16, 1908). The rupture may be where the muscular belly passes into the lower tendon, through the muscular belly, in the muscular part passing either to the long or short head, or at the part where the muscular belly joins the long or short head. The tendon of the long head may be torn through or the long head may be torn from the glenoid cavity. The muscular portion is far more often injured than the tendinous. In rupture of the muscle belly a part of the muscle, in rupture of the long head the entire muscle, becomes soft and relaxed. In rupture of the belly there is a gap between the two portions and each portion causes a lump. In rupture of the tendon there are not two lumps with a gap between, but there will be a single muscular lump. In rupture of the long head the muscular belly is much nearer the elbow than in health (Figs. 449 and 450). If rupture takes place at the lower part of the belly, the muscle passes toward the shoulder. *Rupture of the long head of the biceps* allows the humerus to pass somewhat forward and upward.

Flexion with the forearm supinated is much less powerful than flexion with the forearm pronated (*Hüter's sign*).

In a case of my own in the Blockley Hospital the accident had occurred while carrying a heavy bucket. Forearm flexion was possible, but slow, feeble, partial, and incomplete. On flexion the short head contracted, but the muscular "bunch" of the belly was nearer the elbow than normally. *Rupture of the plantaris muscle* (*coup de fouet; lawn-tennis leg*) is an injury which is frequently not diagnosticated. It occurs during exercise (walking, bicycling, jumping, playing tennis) or is first complained of after exercise. It

Fig. 449.—Author's case of rupture of the long head of the biceps.

produces sudden pain in the middle of the calf, swelling, and often ecchymosis and inability to walk except with a rigid ankle and everted toes. *Rupture of the quadriceps extensor femoris tendon* results occasionally from force which in other cases fractures the patella. The rupture is just above the patella. The patient cannot extend the thigh and cannot walk or stand and there is severe pain. A gap can be felt just above the patella, unless it is hidden by synovial effusion, and the muscle is bunched above.

Fig. 450.—Author's case of rupture of the long head of the biceps.

*Treatment.*—In limited rupture treat as a severe strain. In treating extensive rupture of an important muscle, when the ends are widely separated, expose by incision, unite the divided ends by sutures of chromicized catgut (Fig. 109), and sew up the skin with silkworm-gut. Treat the part in any case by rest and relaxation and combat inflammation by appropriate means. Passive motion and massage are employed as soon as union is firm. In rupture of the quadriceps extensor femoris, operation should be undertaken, because mechanical treatment frequently gives a bad result and confines the patient to bed for weeks. *Rupture of the biceps* requires incision and suture.

In a case in the Blockley Hospital (Figs. 449, 450) I operated and found that the long head with a portion of periosteum had been torn off from the glenoid cavity. A portion of the upper end of the tendon was cut away and the tendon was fastened to the short head by splitting and suture. Nine months later the result was perfect (Keen, in "Annals of Surgery," May, 1905). *Rupture of the plantaris* is treated at first by rest on a posterior splint and compression and later by massage and the use of an elastic bandage. The patient is allowed to walk with the aid of a cane in one week, but he should not raise the heel for several weeks.

**Hernia of Muscles.**—When a tear takes place in a muscular sheath, a portion of the muscle protrudes.

The **treatment** is incision, restoration or extirpation of the protruding mass of muscle, and suturing of any muscle wound and of the sheath.

**Contractions of muscles** may result from injury, from joint disease, from malposition of parts (as in old dislocation or torticollis), or from diseases of the nervous system.

The **treatment** in some cases is sudden extension, in other cases gradual extension, tenotomy, or myotomy. Macewen recommends the making of a number of V-shaped incisions in the muscle. In some cases of spasmodic contraction nerve-stretching is of value.

**Dislocations of Muscles and Tendons.**—The long head of the biceps is oftenest displaced. The flexor carpi ulnaris, the peroneus brevis, the peroneus longus, the tibialis posticus, the sartorius, the plantaris, the quadriceps extensor femoris, and the extensors back of the wrist may be dislocated. What is known as dislocation of the latissimus dorsi, a condition in which that muscle no longer lies upon the angle of the scapula, is, in reality, paralysis of the serratus magnus (see page 751). Most of these accidents are associated with chronic joint disease or with fracture, but displacement may exist as a solitary injury. *Dislocation of the long head of the biceps* may occur tolerably early in the progress of rheumatoid arthritis of the shoulder-joint, and the displaced tendon may be absorbed.

**Symptoms.**—After dislocation of a tendon the muscle of the tendon can still contract, but it acts at a disadvantage; thus the corresponding joint exhibits partial loss of function. The displaced tendon can be felt, and a hollow exists where it normally resides.

When the muscle contracts, the tendon is felt to slip from its groove. When the tendon of the biceps is dislocated, the head of the bone passes forward (so-called *subluxation of the humerus*).

**Treatment.**—In tendon dislocation reduction is easy, but the displacement is apt to recur because of laceration of the sheath. The treatment usually advised is to effect reduction by relaxation of the limb and manipulation of the tendon, to place the part upon a splint so that the muscle belonging to the tendon will be relaxed, and to apply pressure over the point of injury. This treatment generally fails, and if the tendon does not become firmly anchored in its proper situation in four weeks we should operate. In some tendons it is enough to incise, freshen the edges of the torn sheath, and sew up with kangaroo-tendon or chromicized catgut. In a tendon lying in a long groove make a halter for the tendon by incising the periosteum and suturing it over the tendon.[1] Passive movements are begun at the end of the first week. Even if the tendon will not remain reduced, a useful joint will probably be obtained.

**Wounds of Tendons.**—Subcutaneous wounds of tendons are usually inflicted by the surgeon, and they heal well. Open wounds require rigid antisepsis and suturing of the tendon. In wounds of the wrist especially always

[1] Walsham's case of dislocation of the proneus longus, "Brit. Med. Jour.," Nov. 2, 1895.

suture the divided tendons (see Fig. 110), and be sure to bring the proper ends into apposition.

**Rupture of Tendons.**—A violent muscular effort may rupture a tendon, and as the accident occurs a snap may often be heard.

The **symptoms** are sudden pain and loss of power, fulness of the associated muscle from retraction, and absolute inability to bring the tendon into action. A gap may often be felt in the tendon (see page 718).

**Treatment.**—The best procedure in treating rupture of a tendon is exposure by incision and the introduction of sutures. Some surgeons relax the parts and apply splints (see page 718).

**Thecitis,** or **tenosynovitis,** is inflammation of the sheath of a tendon.

**Acute thecitis** may arise from a contusion, from a wound, from repeated overaction in working or while engaged in some sport, from rheumatism, from gonorrhea, from pyogenic infection, from influenza, from a continued fever, or from syphilis. In early syphilis certain tendon-sheaths may rapidly develop effusion because of hyperemia of the sheaths (Taylor).

**Symptoms.**—In *non-suppurative* cases of thecitis the symptoms are pain, swelling, tenderness, and moist crepitus along the tendon-sheath, due to inflammatory roughening. The crepitus disappears as the swelling increases, but it reappears as the swelling diminishes. In *suppurative* cases (*phlegmon of the tendon-sheaths*) the symptoms are great swelling, pulsatile pain, dusky discoloration, inflammation spreading up the tendon-sheaths, and often the constitutional symptoms of sepsis.

**Treatment.**—In treating non-suppurative thecitis employ splints, use the hot-air oven, and apply locally iodin, blue ointment, or ichthyol, and administer suitable remedies to combat any causative constitutional disease. In the suppurative form inject 1 c.c of formalin-glycerin (2 per cent.) after withdrawal of part or all of the exudate. If this fails, make free incisions, irrigate, drain, dress with hot antiseptic fomentations, and employ Bier's method (see page 112). (See Felon, page 724.)

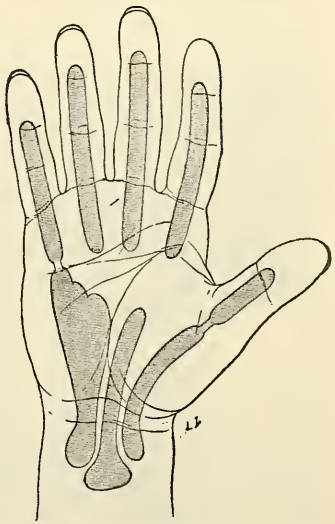


Fig. 451.—Palmar synovial sheaths (vaginæ tendinum), normal adult type (Poirier and Charpy).

**Palmar Abscess.**—We mean by this term an abscess beneath the palmar fascia and not a superfical collection of pus. Palmar abscess may arise after wounds, abrasions, burns, or inflammations of the skin of the palm. A thecal abscess in a flexor tendon of a finger travels rapidly upward and may produce a palmar abscess. A thecal abscess of either the index, ring, or middle finger is usually arrested at the lower end of the palm, but suppurative thecitis of the thumb or the little finger conducts pus along the tendon sheath and up the arm (Fig. 451). If the theca ruptures, pus is diffused over the palm. Abscess produces great swelling of the hand and fingers, the dorsum being swollen as well as the palm. The fingers become flexed and rigid. Violent pulsatile pain and decided constitutional disturbance exist. Discoloration is late in appearing. Related lymph-glands enlarge. Palmar abscess is a most serious affection. The pus may dissect up all the structures of the palm, may pass between the bones and reach the dorsum, or may pass beneath the anterior annular ligament into the connective-tissue planes of the forearm. In some cases it leaves a clawed, stiff, and useless hand.

**Treatment.**—A palmar abscess demands radical treatment at the earliest possible moment; delay will be responsible for stiff and contracted fingers and hyperesthetic skin, a damaged and perhaps a useless hand. The patient should be placed under the influence of ether. The incision is made in the line of the metacarpal bone and, if possible, below the palmar arches. A line transverse with the web of the thumb is below the palmar arches. In an incision above this line try not to cut either arch; but if one should be cut, at once take means to arrest the hemorrhage (see page 449). In a severe case it may be necessary to make several palmar incisions, to open the tendon-sheaths on the flexor surface of the forearm above the wrist, and to make counter-openings in the back of the hand, and it is sometimes necessary to introduce tubes, and drain through and through the hand. After operation apply hot antiseptic fomentations and put the part upon a splint. Bier's passive hyperemia is very useful. When granulations begin to form, dry dressings are substituted for hot moist dressing. It may be necessary to give morphin for pain, and stimulants may be needed. There is great danger of stiffness of the fingers occurring, the tendons becoming adherent to their sheaths. Hence passive movements are inaugurated as soon as granulations begin to form.

**Chronic thecitis** may follow acute thecitis, but may be due to injury, to rheumatism, to gummatous infiltration, to rheumatoid arthritis, or to

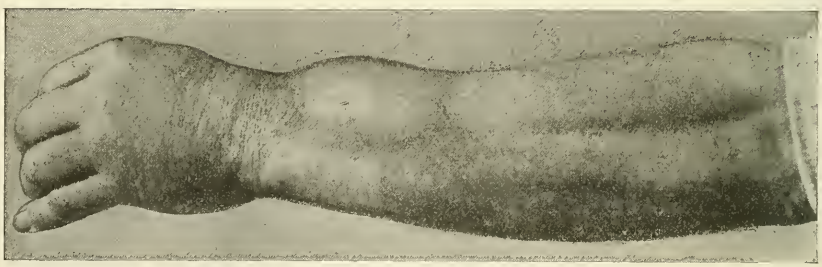


Fig. 452.—Tuberculous thecitis (compound ganglion).

tuberculous inflammation of a tendon-sheath. Chronic thecitis is commonest in the tendons at the wrists, the ankles, and the knees; it may spread to a joint or it may arise from a tuberculous joint. This condition causes very little pain. In ordinary non-tuberculous thecitis the part is weak, tender, painful and stiff, crepitates on motion, and is swollen. In *tuberculous thecitis* there is at first distention of the tendon-sheath with serum. The serum contains *rice*, *riziform*, or *melon-seed* bodies, and the wall of the tendon-sheath is here and there thickened and caseating. Later in the case the interior of the tendon-sheath becomes lined with tuberculous granulations and a tuberculous abscess may form. Rice bodies are sometimes fibrinous masses, are sometimes pieces of separated and dead recently formed fibrous tissue, and are sometimes masses of proliferating cells. In tuberculous cases the swelling is firm or doughy when due to granulation tissue, but is fluctuating when due to fluid. Grating is marked. Tubercle bacilli are present in the fluid or in the granulation tissue. Tuberculous thecitis is most common about the wrist, constituting the so-called *compound ganglion* (Fig. 452).

**Treatment.**—*Tuberculous cases* are treated as follows: If there is a fluid effusion and no rice bodies, make a small incision, wash out with salt solution, introduce iodoform emulsion or formalin-glycerin, and close the wound. In cases in which there are rice bodies, open the sheath, evacuate the con-

tents, scrape the walls thoroughly, inject iodoform emulsion or formalin-glycerin, and close the wound. (If the annular ligament requires division, stitch it before closing the wound—Fig. 453.) In cases with extensive thickening apply an Esmarch bandage, make a large incision, and remove all infected tissue from the sheath, around the sheath, and from the tendon. In tuberculous thecitis Bier's method (see page 112) may be of service and so may the *x*-rays. In *ordinary traumatic thecitis* use for the first few days rest associated with applications of ichthyol. Later employ hot and cold douches, massage and passive movements, strapping of the part, inunctions of ichthyol, and the hot-air bath. If effusion is persistent or rice bodies exist, make an incision and scrape the interior of the tendon-sheaths. In rheumatic cases give antirheumatic remedies and employ the hot-air bath. In syphilitic cases administer mercury and iodid of potassium.

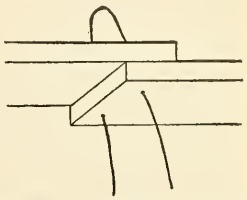


Fig. 453.—Method of suturing the annular ligament of the wrist.

**Simple Ganglia.**—In connection with tendon-sheaths and joints simple ganglia may develop. They are small, tense, round swellings, which are firm, grow progressively though slowly, are painless when uninflamed, and contain a fluid of the appearance and consistence of glycerin-jelly (Bowlby). Ganglia are commonest upon the dorsum of the wrist and they occur especially in those who constantly use the wrist muscles. Ganglia are occasionally seen on the dorsum of the foot. Paget states that a *simple* ganglion is due to cystic degeneration of a synovial fringe inside a tendon-sheath, and that the fluid of the ganglion does not communicate with the fluid of the tendon-sheath. Other pathologists have maintained that a simple ganglion is a hernia of synovial membrane through a rent in a tendon-sheath, all communication between the

Fig. 454.—Ganglion of extensor tendon-sheaths of the wrist.

herniated part and the tendon-sheath being soon obliterated. The belief is now general that a ganglion is due to cystic degeneration of an area of connective tissue adjacent to a joint or a tendon, this area of tissue having been rendered extremely cellular by traumatism. A number of minute cysts form and they coalesce into one cyst. The cyst may form a secondary communication with the interior of a tendon-sheath or joint. Ganglia occasionally diminish in size or even disappear spontaneously.

**Treatment.**—A ganglion is treated by aseptic puncture by a tenotome, evacuation, scarification of the walls, antiseptic dressing, and pressure. An old-time method of treatment was subcutaneous rupture brought about by

striking with a heavy book. Duplay treats a ganglion by injecting a few drops of iodin through a hypodermatic needle. The cyst is not evacuated before injection. The parts are dressed antiseptically, and cure is obtained in one week. Recurrent ganglia, very large ganglia, and ganglia with very thick contents should be dissected out.

**Felon, or whitlow,** is a violent, rapidly spreading pyogenic inflammation of a finger or a toe which resembles cellulitis, and which is sometimes followed by gangrene of the soft parts or by necrosis of bone (Fig. 455). An injury precedes the whitlow—an abrasion of the surface which admits pus-organisms or a contusion which creates a point of least resistance. The commonest seat of a felon is the last digit of a finger or the thumb. An abrasion of the surface at this point absorbs pus-organisms and the superficial lymphatics carry the bacteria directly inward, the micro-organisms lodging, it may be, in the skin, in the subcutaneous tissues, in the tendon-sheath, or beneath the periosteum. The perpendicular direction of the fibers of the subcutaneous tissue favors this passage inward.

Fig. 455.—Deep felon, with sloughing of soft parts and necrosis of bone.

Felons are very rare in infants, but may occur in children. Women are more liable to them than men. The fingers are much more prone to infection than the toes, because they are more exposed to injury. Several fingers may be attacked at once or successively in persons of dilapidated constitution. Whitlow is most apt to occur and is most severe in persons broken down by disease, alcoholism, overwork, or worry. In certain cases of neuritis painless suppuration may arise. In syringomyelia *painless felons* are common, and they are apt to be associated with necrosis of bone. Painless and destructive whitlows constitute a characteristic part of *Morvan's disease.*

There are two forms of felons, the *superficial* and the *deep.*

**Superficial Felons.**—One form of superficial felon is between the cuticle and the true skin and is rarely followed by involvement of deeper parts. The infection is in the skin. The point of infection becomes dark red, swollen, painful, and tender. The epidermis is lifted up into a pustule by the seropus which forms, and a considerable area may be attacked before the spread of the process is arrested. The commonest form of superficial felon is subcutaneous suppuration, the pus collecting in the fibrofatty pad at the palmar surface of the last digit (G. B. Mower White, in "Brit. Med. Jour.," Feb. 24, 1906). This form often spreads deeply. If the subcutaneous tissues only are involved the symptoms are those of an ordinary cellulitis. There is severe pain, increased by motion, pressure, and a dependent position. Swelling and discoloration are early and marked. Pus forms within forty-eight hours. *Paronychia,* or *ring around,* is cellulitis starting at the end or side of the digit, and involving the parts around and below the nail. The pus-organisms obtain entrance by means of an abrasion, a puncture, or an ulcerated "step-mother."

In paronychia pain is throbbing and violent; is increased by motion, pressure, or a dependent position; the skin is dusky red, but the swelling is slight. In about forty-eight hours pus forms in the superficial parts, the epidermis being lifted into pustules or blebs, and pus may also form under the nail. A portion of the nail or the entire nail may be lost.

If the tendon-sheath becomes involved as well as the subcutaneous tissue, the symptoms are those of suppurative thecitis, with more marked discoloration of the skin.

**Deep Felons** (Fig. 455).—There are two forms of deep felon. One is a thecal suppuration involving the flexor tendon-sheath, arising secondarily to subcutaneous suppuration and spreading widely. In suppurative thecitis of the three middle fingers the process seldom reaches the palm; in suppurative thecitis of the theca of the thumb or little finger the pus may pass above the wrist and a true palmar abscess may form (see Fig. 451). Another form is suppuration beneath the periosteum. This form is the so-called *bone felon.* It is occasionally primary, but more often arises secondarily to suppurative thecitis or to subcutaneous suppuration. In some cases a deep felon involves most of the structures of the finger (periosteum, bone, tendon, tendon-sheath, and cellular tissue), and may destroy the digit or the finger. The bacteria causative of a deep felon are lodged in the deeper parts. The pain is agonizing, entirely preventing sleep, pulsatile in character, associated with excruciating tenderness, greatly aggravated by motion or a dependent position, and often extending up the hand and forearm. The skin is dusky red and edematous, and the part is enormously swollen. Pus forms quickly; diffuse cellulitis may arise; sloughing of the tendon and subcutaneous tissue may take place; necrosis of one or more bones may ensue, and in some cases gangrene of the finger follows.

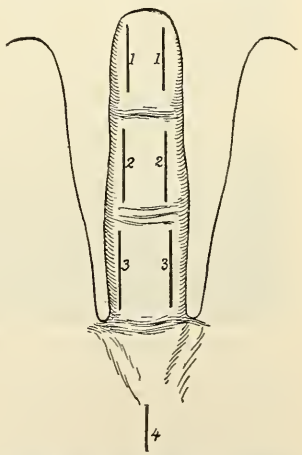


Fig. 456.—1, 2, and 3, Incisions for felon of finger and for ordinary suppuration; 4, palmar incision.

In deep whitlow lymphangitis of the forearm and arm is not unusual, adenitis of the axillary glands is common, and almost always there is fever. In superficial felon constitutional symptoms are slight or absent, and lymphangitis and adenitis arise in a minority of cases.

*Treatment.*—In a subcuticular felon, after cleansing, soften the parts well in an antiseptic fluid and then pare off the cuticle with a very sharp knife. This plan of White's is an excellent one; it gives vent to pus and prevents the inoculation of the deeper tissues which may follow incision. In subcutaneous suppuration incise the abscess, but be careful not to open the tendon-sheath or periosteum, as this would diffuse infection (White, in "Brit. Med. Jour.," Feb. 24, 1906). In neither of the above instances is it necessary to give an anesthetic. After operating, the parts must be irrigated, dressed with hot antiseptic fomentations, the hand must be placed upon a splint, and Bier's passive hyperemia is to be induced daily. In a deep felon I am convinced that we should operate immediately. Allay tension and prevent pus formation by early incision. Do not waste time with poultices; to wait means agonizing pain, sleepless nights, constitutional involvement, and, perhaps, sloughing of tendons or death of bone. Incision and drainage constitute *the* treatment, but incision conducted in a particular manner. I have only lately learned how to treat a deep felon. I formerly treated all cases by incisions down to

the bone alongside of the tendon (Fig. 456), and was frequently disappointed by a spread of the suppuration in spite of incisions, by necrosis of bone, or by extensive sloughing of tendons. I obtained new light upon this subject from an article on "Whitlow," by G. B. Mower White ("Brit. Med. Jour.," Feb. 24, 1906). I immediately put in practice the common-sense suggestions in this valuable article and have seen a surprising improvement in results. The chief points in White's plan of treatment are as follows: To plunge a knife through an area of infection into a tendon-sheath, if that sheath is not infected, will lead to infection, and the way to be sure whether it is or is not infected is to look through a carefully made incision and see. After careful sterilization, anesthetize, drain the extremity of blood by elevation, and apply an Esmarch band to the arm. This enables us to see what we are doing. Slowly and carefully make an incision by the side of the tendon-sheath (Fig. 456), and on reaching it see if it is distended. If in doubt, insert a hypodermatic needle and withdraw fluid. If we get turbid serum, the theca is infected. If the theca is not infected, do not open it, but incise the subperiosteal area of suppuration if it exists. If the theca is infected, remember that this infection has surely ascended more or less, and we must not only open at the lower point, but must also incise at the upper point. Do not incise the theca over the length of the tendon, as sloughing will follow. If one of the three middle fingers is involved, incise the distal end of the theca and also the proximal end over the head of a metacarpal bone in the midline, wash from opening to opening, and drain. If the theca of the thumb or little finger is involved, open distally and then proximally above the wrist. To reach the proximal end of the theca of the thumb cut at the radial side of the tendon of the flexor carpi radialis. Also open the palmar sac of the flexor longus pollicis, making the cut along the inner border of the outer head of the flexor brevis pollicis.

To reach the proximal end of the theca of the little finger begin an incision at the upper margin of the annular ligament and carry it up along the inner border of the flexor sublimis. Retract the tendons and pus will usually be found between the tendons of the superficial and deep flexor. Look beneath the profundus tendons for the bursa and open it. Then open the palm by an incision in the line of the axis of the ring-finger. Thus three openings are made in either case, and the theca can be thoroughly washed and drained. If either the thumb or little finger bursa is found infected, the other must be exposed and examined, as they usually communicate at their proximal ends or a communication may form as a result of suppuration. Rupture of either bursa may diffuse pus widely. White, in order to prevent secondary hemorrhage, ligates the radial artery in two places and removes $1\frac{1}{2}$ inches of it (if operating on the thumb bursa); and ligates the superficial arch and removes 1 inch of it (if operating on the palmar expansion of the little finger theca). These arterial ligations seem a serious and perhaps unnecessary addition to the operation and I have not *practised* them. After thorough irrigation apply antiseptic fomentations, splint the extremity, and induce Bier's passive hyperemia daily. If the patient cannot sleep, give morphin. See that the bowels are moved once a day. Give quinin, iron, and milk-punch. As soon as granulations begin to form, use dry dressings and make passive motion daily. If bone undergoes necrosis, let it loosen and then remove it. Amputation is sometimes necessary.

**Bursitis** is inflammation of a bursa. *Acute bursitis* arises from strain, from traumatism, or from infection. The symptoms of acute bursitis are pain, limited swelling, moist crepitus, fluctuation, and discoloration in the anatomical position of a bursa. In *chronic bursitis* there is intermittent pain, tenderness, and progressive, fluctuating swelling. *Bursitis of the retrocalcaneal bursa* (*Albert's disease*) is a painful affection which is often overlooked. It is rather

common in storekeepers who rise often on the toes to reach shelves, in motormen who use a foot gong, in street-car conductors, and in clerks who stand at desks. It may follow gonorrhea and may be tuberculous. Walking causes great pain in the heel. Raising up on the toes is exceedingly painful. It is usually associated with flat-foot. In these cases osteophytes often form within the bursa. There are numerous bursæ about the hip. Some anatomists count twenty-one.[1] The two most important bursæ and the ones usually affected are the iliac and the deep bursa over the great trochanter.[2] Inflammation of the *iliac* or *iliopsoas bursa* produces swelling below Poupart's ligament, which swelling is tense, but exhibits fluctuation on careful examination. Often the swelling attains large size. In some cases the sac can be emptied by pressure, the fluid passing into an adjacent bursa or into the joint. The swelling is beneath the femoral artery and consequently lifts that vessel (F. B. Lund, in "Boston Med. and Surg. Jour.," Sept. 25, 1902). The enlargement often presses on the anterior crural nerve and causes spasmodic pain throughout the nerve's trajectory. The limb, according to Zuelzer, is usually slightly flexed, abducted and rotated outward, and movement in an opposite direction causes pain. Inflammation of the bursæ about the hip may produce symptoms resembling those of incipient coxalgia, but in bursitis the symptoms do not remit, as in hip-disease. Iliopsoas bursitis occasionally results from gonorrhea. The bursa is sometimes involved in joint-disease. In inflammation of the iliac bursa flexion is not so marked as in coxalgia, and the trochanter is never above Nélaton's line. In inflammation of the deep trochanteric bursa the position is the same as in iliac bursitis, and resembles that of coxalgia. In coxalgia, however, there is pain on pressure upon the front of the joint or directly on the trochanter or on tapping the sole of the foot. These manipulations do not cause pain in bursitis (Zuelzer). In inflammation of the *gluteal bursæ* there is moderate pain back of the thigh and knee, which disappears when the patient is at rest; there are a marked limp, limitation of motion, and an area of deep fluctuation in the buttock (Brackett).

Fig. 457.—Olecranon bursitis.

It is difficult to differentiate between inflammation of a deep bursa and synovitis; indeed, in bursitis the joint is apt to be secondarily affected. This difficulty is especially vexatious in distinguishing between joint-injury and injury of the bursa beneath the deltoid. In subdeltoid bursitis there is a tender spot over the bursa when the arm is by the side. When the surgeon abducts the patient's arm the bursa slips up under the acromion and no tender spot can be found. Suppuration may take place in a bursa. Direct force may rupture a bursa. The bursa beneath the deltoid is frequently ruptured. When this accident happens, there are pain, marked swelling, a large area of moist crepitus, and later extensive discoloration from blood. Chronic bursitis may follow acute bursitis, or the disease may be chronic from the start. It may be due to tuberculosis. Bursæ particularly apt to become tuberculous are those about the hip, the subdeltoid, the olecranon, the prepatellar, and the retrocalcaneal. In tuberculous bursitis during the

[1] Synnestvedt, of Sweden.

[2] Zuelzer, in "Zeit. f. Chir.," vol. l.

first stage the bursa is distended by fluid, due to oversecretion, the walls are thickened here and there, and perhaps contain caseous foci and rice bodies are found in the bursal fluid. In a more advanced stage the bursal wall is lined with caseating granulation tissue and the bursa may become a tuberculous abscess, the walls may give way with diffusion of the process, or mixed infection with pyogenic organisms may occur. In some cases of tuberculous bursitis tending to cure the bursal walls become enormously thickened by fibrous tissue.

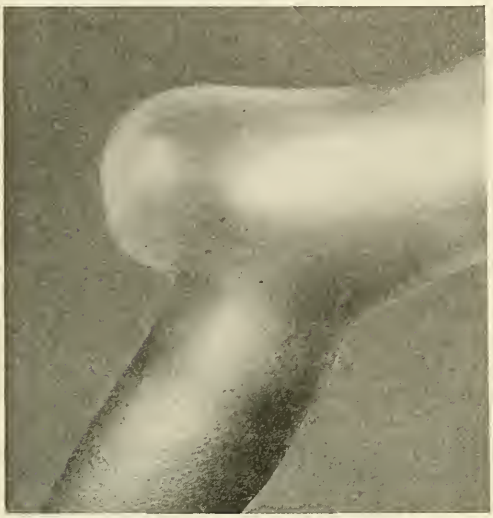


Fig. 458.—Housemaids' knee.

The **symptom** of chronic bursitis is swelling but little or no pain unless acute inflammation arises. Chronic bursitis of the prepatellar bursa is known as *housemaids' knee* (Fig. 458). Chronic bursitis of the subhyoid bursa is known as *Boyer's cyst.* There are six bursæ about the ham, the largest of which is the bursa of the semimembranosus muscle.

**Treatment.**—Acute bursitis is treated by rest, pressure, and the application of iodin, blue ointment, or ichthyol. If the swelling persists, aspirate and apply pressure, or incise the sac and remove it partly or completely. If pus forms, incise, paint the interior of the sac with pure carbolic acid, and pack with iodoform gauze. Chronic bursitis may be cured by the use of pressure and the application of blue ointment, and with treatment of any causative diathesis, but most cases require incision and packing. A ruptured bursa is treated as an acute bursitis. In bursal tuberculosis the best treatment is excision. If we are dealing with a very deep bursa the proper treatment is incision, scraping with a sharp spoon, mopping with carbolic acid, and packing with iodoform gauze.

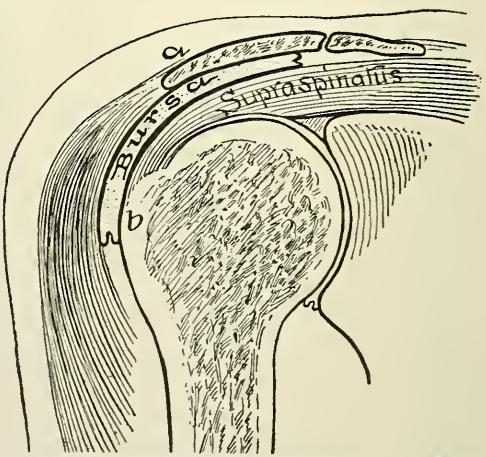



Fig. 459.—Diagram from a frozen section. Notice the deltoid and its origin, from the edge of the acromion. Notice the subdeltoid or subacromial bursa with its roof made by the under surface of the acromion and by the fascia beneath the upper portion of the deltoid. Its base is on the greater tuberosity and the tendon of the supraspinatus, which separates it like an interarticular fibrocartilage from the true joint (Codman).

**Bursitis of the subacromial bursa** has been considered by A. E. Codman ("Boston Med. and Surg. Jour.," Oct. 22 and 29, Nov. 5, 12, 19, and 26, and Dec. 3, 1908), who points out that the deltoid and the subacromial bursa are one and the same thing. When the arm is abducted, the entire bursa is subacromial; when it is adducted, a large portion of the bursa is

subdeltoid. Codman describes three types of conditions associated with inflammation of this bursa, first: The acute, or spasmodic type, in which there is local tenderness on the point of the shoulder, just below the acromion process and outside the bicipital groove. In some cases Dawbarn has shown that the tender point, which is the base of the bursa, disappears under the acromion when the arm is abducted. Codman goes on to show that in attempting abduction about ten degrees of motion can be obtained without moving the scapula. Then the scapula is locked by spasm and moves with the humerus. This spasm may be temporary in mild cases. Sometimes pain prevents the patient from voluntarily raising the arm, though it may be raised by passive motion. The pain may run down the outer side of the arm, even into the hand; the patient frequently locates the pain about the insertion of the deltoid, and may be able to note swelling of the bursa.

Codman describes type two, the subacute or adherent type, in which there are adhesions between the roof and floor of the bursa and a definite mechanical hindrance to abduction and external rotation. There may or may not be local tenderness, but Dawbarn's sign is absent, owing to the presence of the adhesions. Abduction is limited to such a great degree that, as a rule, the tuberosity will not pass beneath the acromion. Any movement in abduction beyond ten degrees causes the scapula to move. The pain is located as in type one, and frequently also passes into the neck. In some cases it is very severe.

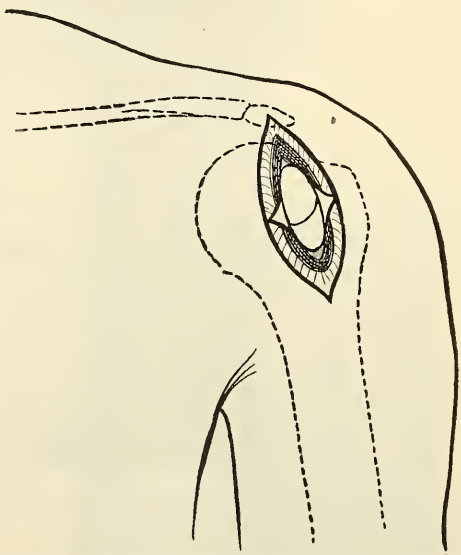


Fig. 460.—Showing incision used for demonstration of the bursa (Codman).

Codman's third form is the chronic and non-adherent. In this the full arc of motion is retained, but motion is painful. The bursa is thickened and irregular. There may or may not be local tenderness; and if this is present, one will find Dawbarn's sign. Abduction and external rotation are limited little, if at all, but at some point during abduction there is severe tenderness, which disappears as soon as the tuberosity passes beneath the acromion. The scapula does not accompany the motions of the humerus. There is often considerable pain after motion.

Codman points out that the prognosis in type one is very favorable if treatment is correct. In type two the disability, even without treatment, seldom lasts more than two years. He says that even severe or adherent cases, if there are no secondary contractures in the forearm muscles, will recover in from one to two years. In infective cases the prognosis is far worse than in traumatic cases. In chronic cases, in which the arc of mobility is not affected, the prognosis is fairly good.

**Treatment.**—Acute cases of subacromial bursitis should be treated by keeping the arm abducted in a splint (Fig. 461). Monks suggests that the patient may sit by a table, the arm being abducted and placed upon a pillow that is on the table. This relaxes the short rotators and the deltoid, and keeps the base of the bursa from being in contact with the acromion. At night

Codman places the arm on a pillow, with its long axis at right angles to the patient's body as he lies recumbent. If the patient has to get about, he may use a sling most cautiously. He should take the arm out from time to time and rest it on a table. Massage should be used about the bursa, but not directly over it. In the more severe cases with adhesions one may employ massage, passive and active movements, baking, forcible movement and

Fig. 461.—Showing the abduction splint in position, the patient standing. It is held by a figure-of-8 bandage, which crosses behind the shoulders and by a belt about the hips. The arm is at rest (Codman).

*Description of Splint.*—The frame of the splint which I use is made of iron wire (diameter, $\frac{1}{4}$ inch), stiff enough to maintain its form and to carry the weight of the arm securely. Sufficient cotton wadding to thoroughly pad it is bandaged over it and the whole covered with cotton or linen cloth. The general shape is shown in the photograph. It should be just long enough to extend from the axilla to the seat of the chair on which the patient sits. It is best held in position by a belt around the pelvis and a figure-of-8 flannel bandage about the shoulders crossing back of the neck. A pad should be placed in the opposite axilla to prevent excoriation of the skin by the bandage.

During the first twenty-four hours and afterward, if worn at night, the arm should also be lightly bandaged to the projecting part of the splint. Additional security is given by the application of a swathe, which may be pinned to the bandage of the axilla.

When properly adjusted it is perfectly comfortable. Unless it is comfortable it is useless. The use of the splint is not essential and is even harmful if not skilfully cared for.

manipulation under an anesthetic, followed by fixation in the position of abduction (Fig. 461), or perhaps incision of the bursa with division of the adhesions or excision of the subdeltoid portion of the bursa (Codman). In the cases in which there is irregularity of the surface of the bursa, one should excise and remove the thickened folds or other irregularities.

**Housemaids' knee** (see Fig. 458) is thickening and enlargement of the prepatellar bursa, the result of intermittent pressure. In effusion into the knee-

joint the fluid is behind the patella and the bone floats up; in housemaids' knee the fluid is above the bone and the osseous surface can be felt beneath it.

Fig. 462.—Bursitis of left olecranon bursa of three years' duration.

In **bursitis of the deep infrapatellar bursa** the swelling is under the ligament of the patella.

**Miners' elbow** (Figs. 457 and 462), which is a condition similar to housemaids' knee, affects the olecranon bursa.

**Weavers' bottom** is enlargement of the bursa over the tuberosity of the ischium. A bursa which is simply thickened and enlarged rarely gives rise to annoyance; but when it inflames, as it is apt to do, it causes the ordinary symptoms of bursitis.

The **bursa of the semimembranosus muscle** is the largest one about the ham. It lies between the inner femoral condyle and inner head of the gastrocnemius and the semimembranosus muscle. It communicates with the knee-joint. When the joint is flexed, it partly empties into the joint and becomes small and lax. When the joint is extended, it becomes large and tense.

Fig. 463.—Enlargement of the deep infrapatellar bursa; chronic and the result of traumatism.

**Treatment of Special Forms.**—Some few cases of housemaids' knee may be cured by rest and blistering, but in most cases it is necessary to incise and pack with iodoform gauze. In enlargement of the bursa beneath the ligamentum patellæ, if rest and blistering fail to cure, aspirate or incise. In enlargement of the bursa beneath the tendon of the semimembranosus and also in "weavers' bottom" and in "miners' elbow," incise and pack. In operating for iliopsoas bursitis I follow Lund's advice and make a vertical

incision below Poupart's ligament, and between the anterior crural nerve and the femoral artery. The fibers of the iliopsoas muscle are separated and the bursa is opened and drained. Some few cases of retrocalcaneal bursitis recover after rest, but most of them require incision and drainage. If osteophytic formations exist, the bony stalactites must be removed by means of the rongeur. Flat-foot, if it exists, is treated by a support (see page 742). The treatment of subacromial bursitis is considered on page 729.

A **bunion** is a bursa due to pressure, and it is most commonly situated above the metatarsophalangeal articulation of the great toe, but is occasionally seen over the joint of another toe. When the big toe is pushed toward the other toes by ill-fitting boots a bunion forms. When a bunion is not inflamed it may cause but little trouble, but when it inflames the bursa enlarges and the parts become hot, tender, and exceedingly painful. Suppuration may occur and pus may invade the joint, and the bone not unusually becomes diseased and very greatly enlarged.

**Treatment.**—In treating a bunion the patient must wear shoes that are not pointed, that have the inner border straight, and that have rounded toes (Jacobson). For a mild case a bunion-plaster gives comfort. Sayre advises the use of a linen glove over the toe, the toe being drawn inward by a piece of elastic webbing, one end of which is fastened to the glove and the other end to a piece of strapping from the heel. A special apparatus may be worn (Fig. 464). In many cases osteotomy of the first phalanx or of the first metatarsal bone is required; in some cases excision of the joint is necessary; in others amputation must be performed. Charles H. Mayo has operated on 65 cases successfully. He removes the head of the metatarsal bone and with it two-thirds of the hypertrophy on the inner side, and turns the bursa into the joint area in front of the bone. He sutures this bursa in place and now has a synovial membrane for a joint which becomes satisfactorily movable ("Annals of Surgery," August, 1908). When the bursa is not inflamed, but only thickened, blisters should be employed over it, or there should be applied tincture of iodin, ichthyol, or mercurial ointment. When the bursa inflames, ichthyol ointment is applied, and intermittent heat by foot-baths gives relief. Suppuration demands immediate incision and antiseptic dressing. If an ulcerated bunion does not heal by antiseptic dressing, stimulate it with nitrate of silver and dress it with unguent. hydrarg. nitrat. (1 part to 7 of cosmolin). Jacobson recommends skin-grafting for some cases.

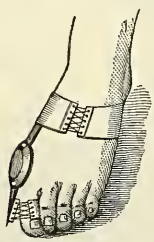


Fig. 464.—Bigg's apparatus for bunions.

## Operations Upon Muscles and Tendons

**Tenotomy** is the cutting of a tendon. It may be *open* or *subcutaneous*, the open operation being preferred in dangerous regions.

**Open Division of the Sternocleidomastoid Muscle for Wry-neck.**—Subcutaneous tenotomy for wry-neck has been largely abandoned. It is not only more unsafe than the open operation, but it never completely divides all the contracted band.

The patient is placed recumbent, the chin being drawn more than is habitual toward the opposite side. A transverse incision is made over the muscle about $\frac{1}{4}$ inch above the clavicle. The superficial parts are divided, the muscle is exposed and sectioned, bleeding is arrested, and the skin is sutured. Avoid the anterior jugular vein, which is underneath the muscle, and also the external jugular, which is close to the outer edge of the muscle. Mikulicz advocates the removal of almost the entire muscle, leaving, however,

the upper and posterior portion where the spinal accessory nerve passes. After operation for wry-neck plaster of Paris is used to secure fixation for from four to eight weeks. Then inaugurate motions, active and passive.

**Subcutaneous Tenotomy of the Tendo Achillis.**—This operation is performed for club-foot, in which the heel is raised. The tendon is cut about 1 inch above its point of insertion. The instrument used for the first puncture is a sharp tenotome. The patient lies upon his back, "with his body rolled a little toward the affected side" (Treves), the foot being placed upon its outer side on a sand-pillow. The surgeon stands to the outer side. The tendon is rendered moderately rigid, and a sharp tenotome, with its blade turned upward, is inserted along the anterior border of the tendon until the surgeon's finger feels the knife approaching the outer side. The sharp-pointed instrument is withdrawn and a blunt-pointed tenotome is inserted in its place. The tendon is drawn into rigidity, and the surgeon turns the blade of his knife toward the tendon, places his finger over the skin, and saws toward his finger. The tendon gives way with a snap. Treves states that a beginner is apt not to push the knife far enough toward the outside, or he may in the first puncture push the knife through the tendon; in either case the tendon is not completely cut. Another method is to insert the tenotome between the skin and the tendon and cut the tendon by a sawing motion. In this method the danger of cutting through the skin is obviated. The little wound, which is covered by a bit of gauze, will be entirely closed in forty-eight hours. In club-foot cases after tenotomy some surgeons at once correct the deformity and immobilize the limb in plaster; some partially correct the deformity and apply plaster for one week, at which time they remove the plaster, correct the deformity further, reapply the plaster, and so on; other surgeons do not attempt correction of the deformity until the cut tendon has begun to unite, when they gradually stretch the new material.

**Subcutaneous Tenotomy of the Tendon of the Tibialis Anticus Muscle.**—The tendon is divided about $1\frac{1}{2}$ inches above its point of insertion. It can be made tense by extending and abducting the foot. The sharp-pointed tenotome is entered upon the outside of the tendon, and is passed well around it. The blunt-pointed tenotome is used to cut the tense tendon.

**Subcutaneous Tenotomy of the Tendons of the Peroneus Longus and Brevis Muscles.**—These two tendons are cut together back of the external malleolus, and $1\frac{1}{2}$ inches above the tip of the malleolus, so as to avoid the synovial sheath (Treves). The patient lies upon the sound side, the outer aspect of the deformed foot being upward and the inner aspect of the ankle resting upon a sand-pillow. A sharp tenotome is introduced close to the fibula, and is carried around the loose tendons. A blunt-pointed tenotome is now introduced, its edge is turned toward the tendons, and these structures are cut as they are made tense.

**Subcutaneous Tenotomy of the Tendon of the Tibialis Posticus Muscle.**—This tendon is sectioned above the point where its synovial sheath begins; that is, above the internal annular ligament (Treves). The tendon is made tense and the pointed knife is entered above the base of the inner malleolus. The knife is entered just back of the inner edge of the tibia, and is carried around the muscle and is kept close to the bone. The tendon is sectioned with a blunt knife.

**Subcutaneous Fasciotomy of the Plantar Fascia.**—The contracted bands are discovered by motions which render them tense, and they are divided just in front of the attachments to the os calcis. The sharp knife passes between the skin and fascia at the inner side of the sole of the foot. The fascia is cut from without inward by the blunt-pointed tenotome. It is usually necessary to section the fascia at more than one point.

**Tendon-suture and Tendon-lengthening.**—Chromicized gut, kangaroo-tendon, or silk is used for an ordinary case, silver wire for a suppurating wound. In performing tendon-suture make the part aseptic and bloodless. It is wise to apply a rubber bandage on the proximal side, the bandage being applied centrifugally, forcing the proximal end of the tendon into view (Haegler). If searching for the próximal end of a flexor of the finger, flex the injured finger and hyperextend the adjoining fingers (Filiget). If this expedient fails, enlarge the incision or, what is better, make a large flap in the skin. After finding the ends, approximate them, being sure the proper ends are brought into contact; stitch them together by a continuous suture or by one of the sutures shown in Fig. 465, 1, 2, and 3.

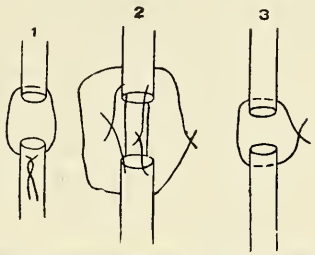



Fig. 465.—Tendon-sutures: 1, Of Le Fort; 2, of Le Dentu; 3, of Lejars.

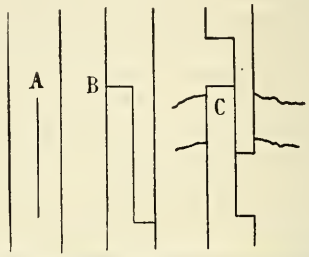



Fig. 466.—Anderson's method of tendon-lengthening.

In a suppurating wound suture by silver wire should be tried, though it usually fails. After suturing, remove the Esmarch apparatus, arrest bleeding, close the wound and dress it antiseptically, relax the parts, and place the limb on a splint. If, after suturing, there is much tension, stitch the cut tendon above the sutures to an adjacent tendon, and apply a splint, the finger which was injured being flexed, the others being extended. If only the distal end of the tendon can be found, graft it upon the nearest tendon with a like anatomical course and function. After a tendon has been sutured, begin gentle massage in two weeks. Positive passive motion is begun in three or four weeks. In old injuries, when the ends cannot be brought into apposition, lengthen one end or both ends, either by the method of Anderson (Fig. 466) or by the method of Czerny (Fig. 467). Dr. J. Neely Rhoads ("Med. News," Nov. 28, 1891) suggested that slight lengthening could be accomplished by "cutting half through the tendon at different levels and from opposite sides, leaving some longitudinal fibers to slip on each other, thus gaining slight elongation" (H. Augustus Wilson, in "International Clinics," vol. i, 4th series). Poncet makes several zigzag incisions on each side of the tendon, and when the tendon is pulled upon it elongates decidedly. Hibbs's method is shown in Fig. 468. One of these methods of lengthening may be used if there is deformity from tendon contraction. If the tendon cannot be lengthened sufficiently, make a bridge of catgut from one cut end of it to the other, or graft in another tendon from one of the lower animals, or graft the distal end to a tendon of like function (*tendon-grafting*).

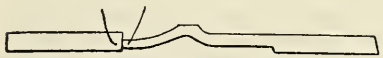


Fig. 467.—Czerny's method of tendon-lengthening.

The annular ligament is sutured as shown in Fig. 453.

**Tendon-transplantation and Silk Inserts.**—Tendon-transplantation is the transplantation of the tendon of a healthy muscle to take the place of the tendon of a paralyzed muscle. Silk inserts are used to take the place of paralyzed muscles, "to lengthen normal tendons for the purpose of using them

in transplanting, and to reinforce joints as artificial ligaments, in place of arthrodesis" (James W. Sever, "Jour. Am. Med. Assoc.," May 11, 1912). Tendon-transplantation is usually said to have been devised by Nicoladoni in 1882; as a matter of fact, Duplay did the operation in 1876, endeavoring to secure function in an arm rendered nearly powerless by an injury (Elting, in "Albany Med. Annals," April, 1902).

The first American surgeon to do the operation was Parrish, of New York, who in 1892 transplanted tendons in a case of club-foot. In some cases in which a muscle has been paralyzed surgeons have divided the tendon of the paralyzed muscle and have united its distal end with the tendon of a normal muscle, the normal tendon being split to receive it. It has also been stated that when a muscle or the tendon of a muscle is sutured to a paralyzed antagonistic muscle, the transplanted structure will actually execute the functions of the paralyzed muscle. For instance, a flexor, when so transplanted, may become an extensor and act under the mental impulse of extension; a pronator may become a supinator (H. A. Wilson, in "American Med.," April 8, 1905). These principles have been utilized when some or many of the muscles of a limb have been paralyzed, the tendon of an unparalyzed muscle or the tendons of an unparalyzed group of muscles being fastened to the tendons of the paralyzed muscle. It has been shown that the success of the procedure depends upon the accuracy of diagnosis, the division of secondary contractures, the correction of existing deformities, and careful after-treatment. (See the article by Dr. J. Hilton Waterman, in "Med. News," July 12, 1902.) In a paralysis

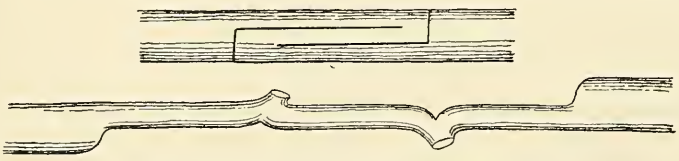


Fig. 468.—Hibbs's method of tendon-lengthening.

of the lower extremity, as Goldthwait points out, the sartorius usually retains power, and it may be advisable in such a case to divide the sartorius and suture its upper end to the quadriceps above the patella. A strip of the tendo Achillis may be grafted upon the peronei in certain cases. An artificial tendon may be made of silk, the silk being passed from the sound to the paralyzed tendon. This method was devised by Auger in 1875. The silk must have been boiled in paraffin, otherwise it will slough out. The silk eventually becomes surrounded by fibrous tissue. Some maintain that silk is eventually absorbed. Lange showed that tendon regeneration may take place along the strands of silk. Strands of silkworm-gut may be used for the same purpose (Kummell). It is useless to make a silk insert on a tendon in a patient under nine or ten years of age. Coöperation between surgeon and patient is essential if we would develop function, and coöperation is sure to be lacking before that period of life. The operation of tendon-transplantation is occasionally of distinct benefit, but I agree with Ridlon, and am not usually sanguine of results. Ridlon wisely reminds us that in such cases much good may perhaps result from the proper use of braces, tenotomy, and hand stretching, followed by prolonged retention in plaster, the patient using his limb actively.

Ridlon points out that most brace treatment is not curative because it only aims to prevent deformity developing, and tenotomy and stretching fail because they only seek to remove existing deformity. The object should be some restoration of function. This is often obtained by following Thomas's direction and "posturing" the limb so as to permit structural shortening of the paralyzed muscles and then fixing them for months.

## XXII. ORTHOPEDIC SURGERY

This branch of surgery formerly dealt only with the treatment of deformities by means of mechanical appliances, but of recent years its domain has been enlarged to include the treatment, surgical and mechanical, of deformities, contractures, and many joint diseases.

**Torticollis** (**wry=neck**) is a condition in which contraction of certain of the neck muscles causes an alteration in the position of the head. The disease is one sided; the sternocleidomastoid is the muscle chiefly involved, though the trapezius, the splenius, and other muscles sometimes suffer. Acute torticollis, which is rare, is a temporary condition, and results from cold or from injury (see Myalgia). Chronic torticollis may be congenital (due to injury before birth or during birth), may be due to nerve irritation, to an assumed attitude because of eye defect, to polio-encephalitis (Golding Bird), to inflammation of the glands or to disease of the vertebræ, and it may be intermittent, but is usually persistent. The muscle stands out in bold outline, the head is turned to the opposite side, the ear of the disordered side is turned toward the shoulder, the chin is thrown forward, and spinal curvature may arise. The corresponding side of the face atrophies. There is no pain. In many cases the head may be restored to its normal position by passive movement or by voluntary effort, but it at once returns to its habitual position. Mikulicz asserts that torticollis is a chronic fibrous myositis, due often to compression during labor. He further says that the lesion known as hematoma of the sternomastoid, which occasionally follows labor, is not hematoma, but thickening due to myositis. D'Arcy Power reported the autopsy on a child one month of age. The sternomastoid muscle contained a fibrous mass, the result, Power believes, of a hemorrhage into the muscle prior to birth ("Med. Chir. Trans.," vol. lxxvi). Power, Clutton, and Owens have all traced cases of hematoma of the sternomastoid from early infancy to the time when tenotomy was required for torticollis. W. W. Richardson ("Surg., Gynec., and Obstet.," 1906) believes that interstitial myositis is always present, but doubts the causal influence of the lateral position of the head *in utero*. In some cases hereditary influence is evident. One woman gave birth successively to 7 wry-necked children (Nove-Jusserand and Vianny, in "Revue D'-Orthop.," 1906). Many writers advocate the ischemic theory as explanatory of the causation of torticollis. This theory is, that during labor lateral flexion of the head with elongation of the neck or lateral flexion with torsion produces occlusion of the sternomastoid branch of the superior thyroid artery, which vessel supplies the sternocleidomastoid muscle (see Tubby's "Orthop. Surgery"). In congenital torticollis the muscle and the cervical fascia are shortened, and the muscle does not relax under the influence of an anesthetic. In torticollis due to rheumatism and reflex causes the tonically contracted muscle relaxes when the patient is anesthetized. In spasmodic wry-neck the muscle is thrown repeatedly into clonic contractions.

**Symptoms.**—*Congenital* wry-neck is due to central nervous disease, to spinal deformity, or to injury during birth, and in this form the sternomastoid is shortened, hardened, and atrophied. It may not be noticed for some years because of the short neck of infancy. It is associated with asymmetrical development of the face, and is almost invariably upon the right side. *Spasmodic* wry-neck may present tonic spasm only, intermittent spasm alone, or both may appear alternately. It sometimes arises in those whose occupation demands frequent rotation of the head, but more often no such cause can be discovered. It is probably a disease of the cortical area which presides over rotation of the head. (See article by C. A. Hamann, in "Buffalo

Med. Jour.," Dec., 1901.) It is a disease especially of adults; in women it is often linked with hysteria. Pahl ("California State Med. Soc.," 1906) analyzed 68 reported cases. Men and women appeared equally liable, it was most frequent between the ages of twenty and thirty, and the right side was affected in twice as many cases as the left side. The exciting cause may be a cold, a blow, or a mental storm; the predisposing cause is the neurotic temperament. It may be due to enlarged glands, to carious teeth, or to eye-strain. In some rare cases bilateral spasm occurs, the head being pulled backward and the face being turned upward. Clonic spasms may come on unannounced, or they may be preceded by pain and stiffness; the head can be held still for a moment only; there is sometimes pain, always fatigue, but during sleep the contractions cease. The attack will probably pass away, but will almost certainly occur.

**Treatment.**—Congenital wry-neck is treated by myotenotomy through a superficial incision which is vertical or transverse (see page 732). The muscle is divided in a line parallel to and just above the clavicle or just below the mastoid attachment. The flaps are raised, access is free to the origin of the muscle, the muscle is readily divided, the wounds in the skin and fascia are closed. This incision leaves a trivial scar. After operation plaster of Paris is used to secure fixation and fixation is maintained from four to eight weeks. Early operation favors the establishment of muscular co-ordination before the development of permanent bony deformity of the vertebræ. Some surgeons cut the scalene muscles as well as the sternocleidomastoid. Gerdes Rowland lengthens the sternomastoid and carefully sutures it ("Practitioner," Sept., 1908). The old subcutaneous myotenotomy should be abandoned, as aseptic incision enables the surgeon to see and to feel all the contracted bands of fascia, muscle, and tendon, and to avoid vital structures (see page 732). In spasmodic wry-neck there is a fair chance of recovery. Pahl's table (Loc. cit.) shows that out of 68 cases, 28 recovered, 17 were improved, and 11 were not improved by treatment. Results in 12 were not stated. Treat the neurotic temperament and remove any obvious irritation (eye-strain, carious teeth, enlarged glands). Drugs usually are practically useless, although Chas. S. Potts reported a cure after the hypodermatic use of atropin. The rest cure is sometimes beneficial. Tenotomy is not to be employed. In 1890 Mayo Collier suggested ligation of the spinal accessory nerve with silver wire. In persistent cases stretch or divide and exsect a part of the spinal accessory nerve (Keen). To reach this nerve make an incision along the posterior edge of the sternocleidomastoid muscle, find the nerve as it emerges from under the middle of the muscle, about $1\frac{1}{2}$ inches below the tip of the mastoid process, retract the muscle at this point, and remove at least 1 inch of nerve. Neurectomy of the spinal accessory nerve paralyzes the sternocleidomastoid muscle, in spite of the fact that that muscle has also a nerve-supply from the cervical nerves. The paralysis is followed by atrophy, and if the spasm affected the sternomastoid muscle only the operation will cure the case. Unfortunately, other muscles are usually involved, and cure will only be obtained by performing neurectomy on the nerves which innervate the affected muscles. (For the treatment of rheumatic wry-neck, see Myalgia, page 712.)

**Dupuytren's contraction** is a contraction of the palmar fascia, of its digital prolongations, and of the fibers joining the fascia and skin. Fixed contraction of one or more fingers occurs (Fig. 469). The ring-finger and the little finger most often suffer, but any finger or the thumb may be involved. The condition may be symmetrical. It is far more common in men than in women. The disease arises oftenest in men beyond middle age, but is sometimes met with in youths. The **cause** of this disease is unknown; some refer it to gout, rheumatism, or osteo-arthritis; others, to traumatism, syphilis, organic

nervous diseases, arteriosclerosis, reflex irritation, or neuritis. In one-fourth of the cases heredity seems to be influential. If due to traumatism, the right hand should suffer most frequently; but it occurs in the left hand nearly as often as in the right (P. Jansen, in "Arch. f. klin. Chir.," Bd. lxvii, H. 4). Jansen examined specimens from 7 cases and found connective-tissue hypertrophy and circulatory disturbance, the contraction being a result of the above-named processes.

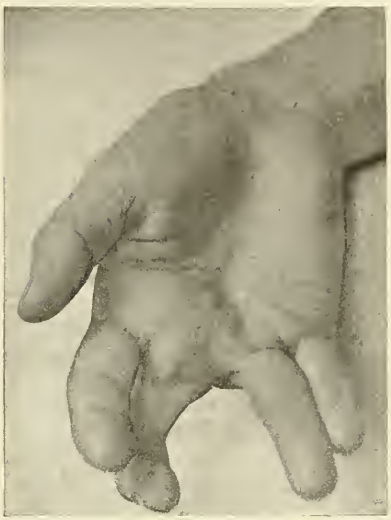


Fig. 469.—Dupuytren's contraction of the middle finger.

**Symptoms.**—Dupuytren's contraction is indicated by a small hard lump or crease which appears over the palmar surface of the metacarpophalangeal joint. This nodule grows and the corresponding finger is gradually pulled down. In some cases the tip of the finger is forced against the palm. The skin becomes dimpled or puckered.

**Treatment.**—Fibrolysin, which is a soluble combination of thiosinamin and salicylate of sodium, has been used hypodermatically in Dupuytren's contraction and, it is claimed, with success (Schwalbach). In treating Dupuytren's contraction subcutaneous multiple incisions may be made, the tense fascia and the fasciocutaneous fibers being cut. The finger is straightened and is placed upon a straight splint, which is worn continuously for a week or ten days and is worn at night for at least a month. A more satisfactory operation is that of Keen. He divides the skin by a V-shaped cut, the base of the V being downward, lifts up the flap, and dissects out the contracted tissue. A valuable method is that of McCurdy. He makes a long incision which crosses the contracture obliquely, stretches thoroughly, closes the wound, and keeps up mechanical fixation for a time. A cure is most certain to be obtained by Lexer's radical operation. This surgeon excises the entire aponeurosis and considerable portions of the palmar skin adherent to the aponeurosis.

Fig. 470.—Agnew's operation for webbed fingers (Pye).

Fig. 471.—Diday's operation for webbed fingers (Pye).

In order to cover this wound it may be necessary to slide a pedunculated flap into the raw surface.

**Syndactylism (webbed fingers)** is always congenital, and may persist through several generations. Simple incision of the web is useless; the operation to be performed is that of Agnew or of Diday (Figs. 470, 471).

In Agnew's operation a flap of skin from the dorsum is inserted between the fingers and sutured in place.

In Diday's operation a flap is taken from the dorsal surface and another flap is raised from the palmar surface, and each flap is sutured to the finger to which it is attached.

**Polydactylism (supernumerary digits)** is always congenital, is often hereditary, and is usually symmetrical. There may be an incomplete digit, or there may be an entire and well-developed finger or toe with a metacarpal or metatarsal bone. The connection to the metacarpus or metatarsus may be by a fibrous pedicle only. If the digit is complete, with a metacarpal bone, no operation is required; if it is incomplete or is ill-developed, it should be removed.

**Trigger-finger or Jerk-finger (Lock-finger, Snapping-finger).**—The patient can usually close the fingers, but on trying to open them one finger remains closed. It can be opened by grasping it with the other hand, but flies open with a snap, like opening a knife (Abbe). In some cases two fingers are involved. In a reported case (Frederick Griffith, "Annals of Surgery," 1904) the ring and middle fingers of the left hand locked at the knuckle-joints on attempting flexion. The locking occurred when about one-third the amount of flexion necessary to grasp an object was achieved. By bending the fingers with the other hand unlocking was accomplished and flexion was finished voluntarily. In attempting extension blocking occurred at the same point and unlocking was accomplished in the same manner. In most cases, but not in all, there is pain when locking occurs. The condition is gradual in onset. Trigger-finger is often associated with rheumatism (in 52 cases out of 121, according to Necker). It is said by Tubby to be due to enlargement of the flexor tendon, or to contraction of the groove in the transverse ligament in the palm. It may be due to a ganglion, enchondroma, or tenosynovitis. Traumatism or irritation may produce it. The tendon-sheath may be thickened or, according to Marcano, there may be a nodule on the tendon which rubs against the sesamoid bone. It may result from occupation.

**Treatment.**—If a ganglion, a loose cartilage, or a tendon nodule exists, treat by excision. A sesamoid bone may be excised. If there is inflammation, use massage and counterirritation. If there is no obvious cause, put a compress over the tunnel in the ligament and apply a splint.

**Mallet-finger.**—This is called also *drop-finger* and *rupture of the extensor tendon.* It is due to a blow in the direction of flexion when the finger is extended. It is supposed to be due partly to stretching and partly to rupture of the extensor tendon at the point at which it is the posterior ligament of the distal interphalangeal joint. Abbe has shown that baseball players are liable to a condition which is the reverse of this, in which the last phalanx is dislocated backward. Drop-finger is treated by incision and suture of the tendon to the periosteum.

**Genu valgum (knock-knee)** results from an unnatural growth of the internal condyle, causing the shaft of the femur to curve inward and the internal lateral ligament of the knee-joint to stretch, the knees coming close together and the feet being widely separated. The condition may also be caused by curving of the tibial shaft just below the epiphysis. This deformity is usually noted when the child begins to walk, but it may not appear until puberty or even long after. Knock-knee may arise from rickets, from an occupation demanding prolonged standing, or from flat-foot. It may occur in one knee or in both knees.

**Treatment.**—Mild rachitic cases of knock-knee may remain in slight deformity, or may get well from improvement of the general health, though they seldom do. In an early case properly applied braces will correct deformity. In a later case operation will be necessary. In ordinary cases simply treat the

rickety condition. The patient is forbidden to stand or to walk, and the limb, after being put as straight as can be, is fixed on an external splint and a pad is put over the inner condyle. Later in the case plaster of Paris is used. Some surgeons prefer to immobilize while the leg is flexed to a right angle with the thigh. In a severe case the surgeon can immobilize after forcibly straightening (causing an epiphyseal separation) or after the performance of osteotomy (see page 688). Osteotomy is preferable to fracture by a mechanical appliance (osteoclasis).

**Genu varum** (**bow-legs**) is the opposite of knock-knee. It tends much more to self-correction than knock-knee because of the arrangement of the thigh muscles, the powerful adductors acting strongly on the knee and middle of the leg (J. Torrance Rugh, in "Am. Jour. Orthop. Surg.," April, 1908). Usually both legs are bowed *out*, the knees being widely separated, the tibiæ and femora, as a rule, being curved, and the feet being turned in. This disease in early life is due to rickets, the weight of the body producing the deformity. In older people incurable bow-legs may arise from arthritis deformans.

Fig. 472.—Club-hand.

**Treatment.**—Some mild cases of genu varum recover as a result of improvement in the health. Ordinary cases are treated by braces, by plaster-of-Paris bandages, and by attention to the general health. Braces usually suffice prior to three and a half years of age. Later, when the bones have hardened in severe deformity, osteotomy is necessary.

**Club-hand** (Fig. 472).—A congenital deformity in which the hand deviates from the normal relation to the forearm. It is usually associated with other deformities. In some cases the radius and possibly some of the carpal bones are absent.

**Treatment.**—By massage and passive motion, by immobilization, by tenotomy or osteotomy, or by bone-grafting.

**Talipes** (**club-foot**) is a permanent deviation of the foot into deformity. There are several forms: *talipes equinus* (Fig. 473) is a confirmed extension; *talipes calcaneus* (Fig. 474) is a confirmed flexion; *talipes varus* is a confirmed adduction and inversion, and *talipes valgus* is a confirmed abduction and eversion. Two of these forms may be combined, as in talipes equinovarus (Fig. 475), talipes equinovalgus, talipes calcaneovarus, and talipes calcaneovalgus. The **causes** of talipes are congenital or acquired. The congenital form is due to persistence of the fetal form of the foot. There are three theories of the cause of the deformity: viz., the nervous theory, the mechanical theory (intra-uterine pressure), and arrest of development. Acquired cases may arise from infantile paralysis, from spastic contractions, from cicatrices, from traumatisms, from arrest of bony growth following upon the inflammation of bone, or from hysterical contractures.

*Talipes equinus* is rarely congenital. In this condition the patient walks upon the toes and cannot bring the heel to the ground.

*Talipes Calcaneus.*—The patient walks upon the heel and cannot bring the toes to the ground. The true form is seen in congenital cases, the flexors of the foot being shortened and the tendo Achillis being lengthened.

*Talipes varus* is rarely met with without equinus. In this condition the patient walks on the outer edge of the foot.

*Talipes valgus* is met with in flat-foot. The patient walks on the inner edge of the foot.

*Talipes Equinovarus.*—The heel is raised and the patient walks upon the outer edge of the foot. This is the usual congenital form.

*Talipes equinovalgus* is very rarely congenital. The heel is raised and the patient walks upon the inner side of the foot.

*Talipes calcaneovarus* is a combination of calcaneus and varus.

*Talipes calcaneovalgus* is a combination of calcaneus and valgus.

**Treatment.**—In congenital cases the condition is usually manifest on both sides, and is nearly always talipes equinovarus. It is better that both sides should be affected, as the feet will then be symmetrical through life. Congenital club-foot should be treated in infancy, and when a restoration to position can be effected by the hands of the surgeon, is treated by plaster-of-Paris

Fig. 473.—Talipes equinis (Albert).

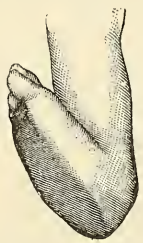


Fig. 474.—Talipes calcaneus (Albert).

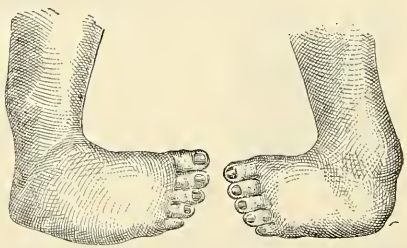


Fig. 475.—Double equinovarus ("American Text-Book of Surgery").

bandages. If a child has begun to walk, it may still be possible to correct the deformity eventually by manipulations, by plaster-of-Paris bandages, or by club-foot shoes, but most cases require tenotomy of the tendo Achillis before the application of the shoe or the plaster. The club-foot shoe may do good service, but in many instances it is painful and is not so efficient as plaster of Paris. In severe cases, before applying the plaster, the patient is given ether; the surgeon cuts the tendons of the anterior and posterior tibial muscles, the plantar fascia, the tendo Achillis, and the long flexor of the toes, in the order named, and forcibly corrects the deformity. In old cases, with alteration in the shape of the bones, cuneiform osteotomy, or the removal of the cuboid or other tarsal bones, may be indicated. In these cases Phelps advises an open transverse division of all rigid plantar soft parts. Buchanan employs subcutaneous division of all resistant structures. Occasionally, in relapsed and inveterate cases, *astragalectomy* is performed. It is seldom practised upon young children (see page 705). In some cases of talipes calcaneus shortening of the tendo Achillis is advised; but such an operation is only of temporary value, as stretching occurs after two years or more. In talipes due to infantile paralysis the operative treatment is the same, but we should not immobilize in plaster, but rather in some apparatus which can easily be removed to permit the use of massage and electricity. In paralytic cases tendon-transplantation is occasionally employed. This consists in transferring the tendon of an active

muscle so that it will take the place of the tendon of a paralyzed muscle. The transferred tendon should be always attached to the periosteum (Tubby and Jones on the "Surgery of Paralysis").

**Pes planus (flat-foot)** is a condition in which there is loss of the arch of the foot due to muscular paralysis or ligamentous weakness, to prolonged standing, or to trauma. Flat-foot is especially apt to occur in rickets. *Spurious flat-foot*, or *inflammatory flat-foot*, occurs in Pott's fracture and in inflammation of the ankle-joint or of the tendon of the peroneus longus muscle. *Paralytic flat-foot* is seen after infantile paralysis. *Static flat-foot* is due to disproportion between the body weight and the support of that weight. All children are born with pronated feet; the arch usually begins to form soon after birth, but in some individuals it never forms. Flat-foot, according to de Vlaccos, is thus produced: If we suppose a straight line prolonged downward from the center of the leg, most of the astragalus and os calcis will be external to it; hence the body weight presses on the inner side of the foot, and tends to flatten the arch and cause outward rotation, tendencies which are antagonized by the flexors of the toes and by the tibialis posticus muscle. The os calcis is pronated and is pushed to the side, the astragalus moves after the os calcis, and the ligaments are stretched ("Rev. de Chir.," Aug., 1901). A very common cause is contraction of the tendo Achillis. In childhood the condition is seldom recognized, but in an adult with contracted Achilles tendon long hours of standing will quickly precipitate the acute symptoms of flat-foot. Pes planus is productive of much pain upon standing or walking; in fact, the individual may be completely crippled. Pain is quickly relieved upon sitting down. Walking upon the toes is not painful. A marked flat-foot can at once be recognized by wetting the sole of the patient's foot with a colored fluid and causing him to step firmly upon a piece of paper (Fig. 476, B). Beginning flat-foot cannot be thus recognized and is frequently overlooked, the patient being treated for gout or rheumatism. Even a slight case can be detected by carefully observing the inner surface of the foot. When weight is placed upon it, it is seen to descend as the arch falls. A more accurate method is measurement, to find the middle of the foot. In flat-foot the extremity is lengthened. Golding-Bird points out that the middle of the normal foot is the point of articulation of the inner cuneiform and the metatarsal bone of the great toe. In flat-foot the greatest change is in the posterior half of this line. The extent to which the posterior measurement exceeds the anterior is the degree of flat-foot. The excess may reach $\frac{3}{4}$ inch.

A B

Fig. 476.—Print of a normal foot sole (A) and of a flat-foot sole (B) (Albert).

Treatment.—In *paralytic flat-foot*, which arises from infantile paralysis, employ exercise, electricity, and massage. To maintain a correct position of the ankle and to facilitate normal muscular action, apply suitable braces. In some cases of paralytic flat-foot it is advisable to permanently stiffen the ankle-joint by operation. Operation is not indicated before the twelfth year, because during the earlier years of life union will probably fail to occur. Goldthwait removes the cartilage from the articular surfaces of the astragalus, calcaneus, tibia, and malleoli, and seeks to obtain permanent bony ankylosis. In *static flat-foot* it has long been customary to advise rest in bed for two weeks, and then exercise for several hours a day to increase the arch. The usually recommended exercise has been to rise upon the toes and lower again and again, with the ankles turned outward. The patient rests for a time after each séance of exercise by sitting tailor-fashion with the legs crossed under him or by standing on the outer edges of the feet. Massage is ordered and a special shoe

is made to raise the arch of the foot. The shoe must fit the heel snugly and have a firm, broad heel. In some cases it is necessary to use a Thomas heel; in others, a steel shank. The patient's general health is, of course, attended to.

Many orthopedic surgeons have come to regard this usual treatment as unphilosophical and improper for many cases.

In static flat-foot it is essential to understand that a flat-foot may be a fully functionating foot, free from pain and disability, and, therefore, not a

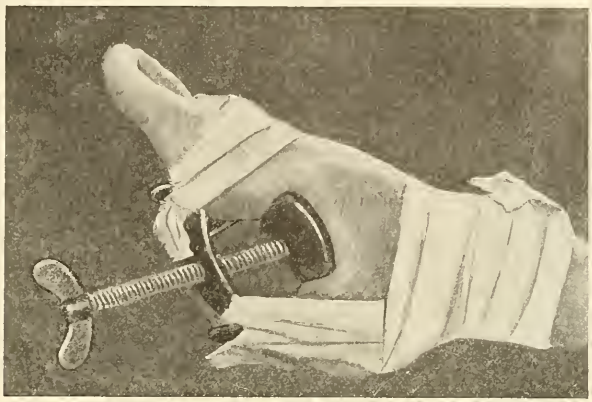


Fig. 477.—H. Augustus Wilson's flat-foot correction screw.

subject for treatment. For convenience, flat-foot is divided into rigid and flexible. Either form may be from pain. The pain of flat-foot is usually the result of excessive use. It must be differentiated from Albert's disease (achillodynia), metatarsalgia, osteophytes on the under surface of the os calcis, and Raynaud's disease. Rigid flat-foot can be made flexible by manipulative measures (according to Whitman's method) or by the employment of H. Augustus Wilson's flat-foot correction screw (Fig. 477). This appa-

Fig. 478.—Whitman's plate to support the arch of the foot in flat-foot (Fowler's "Surgery").

ratus pulls down on the posterior part of the os calcis and the distal extremities of the metatarsal bones and pushes up beneath the tarsus. The force employed is very great and much care should be exercised when it is used upon a patient under anesthesia. It is preferable to use it without ether, relying upon the patient to state when the pressure becomes unendurable. A flexible flat-foot is capable of correction by exercises.

It was formerly customary to always prescribe various forms of steel plates

to correct the broken-down arch, but some orthopedic surgeons are discouraging their use, believing that they destroy the muscular control of the foot, and by weakening the foot render it susceptible to sprains and other injuries. It is my belief that steel plates should often be used, but never abused (Fig. 478). When plates are necessary because the patient is heavy or because he must continue a trying occupation, they must be fitted to the individual case and must be worn until such time as the use of exercise has enabled the patient to properly maintain the body weight with the strengthened arches. If the tendo Achillis is shortened it must be lengthened by operation or else the heel of the shoe must be raised to permit the fullest range of dorsal flexion the tendon allows. When muscle tone is low and there is small chance of restoring it, H. A. Wilson ("Amer. Medicine," May 6, 1905, page 725) advocates the employment of the method devised by Professor Müller ("Central. f. Chir.," January 10, 1903, page 40) for paralytic valgus. It consists of an arthrodesis of the astragaloscaphoid joint, and transplantation of the tendon of the extensor proprius hallucis into a hole drilled free from above downward through the scaphoid. Fixation in plaster of Paris in an overcorrected position is maintained for four weeks and then corrective exercises are employed. The anterior tibial tendon is supplemented in its action by the transplanted tendon.

Gleich shortens the foot and raises the arch by sawing through the os calcis and fastening the posterior part of this bone at a lower level. Trendelenburg advises supramalleolar osteotomy. This operation permits of adduction, and the adducted foot should be put up in an immovable dressing of plaster of Paris. Ogston resects the astragaloscaphoid joint. Golding-Bird and Davy remove the scaphoid bone. Stokes removes a wedge-shaped piece from the head and neck of the astragalus. Rugh has taken a wedge-shaped piece of bone from the inner side and inserted it in the outer side.

**Pes cavus (hollow foot)** is an increase in the arch of the foot, due, possibly, according to Golding-Bird, to paralysis of the peronei muscles. When the peronei muscles are paralyzed the adductors act unopposed, and secondary contraction of the plantar fascia occurs. Certain it is that a contracted plantar fascia is the chief obstacle to correction. In many cases the cause is the wearing of shoes which are too short for the feet. The pressure made upon the toes causes spasm of the plantar flexors and this spasm permits the fascia to contract.

**Treatment.**—A shoe is worn containing a plate of steel in the sole, and pressure is applied over the instep. Tenotomy, division of the plantar fascia, or excision of bone may be required. In paralytic cases apply electricity and massage to the paralyzed muscles. Transplantation of the tendons of the dorsal flexors into the ends of the metatarsal bones has proved very satisfactory.

**Hallus valgus** or **varus,** a displacement of the great toe outward or inward, may occur in the young, but it is most frequent in old persons, especially old women. It arises often from wearing pointed shoes, shoes that are too short, or high heels, but may be due to gout or to rheumatic gout. In many cases an exostosis forms in the inner portion of the distal end of the metatarsal bone. In hallux valgus a *bunion* (bursa) is apt to form over the metatarsophalangeal joint and it may inflame or ulcerate.

**Treatment.**—An arrangement may be worn to straighten the toe and to protect the bunion (see Fig. 464). The prominent and hypertrophied inner portion of the head of the metatarsal bone may be removed by means of a chisel, osteotomy may be performed upon the metatarsal bone, the joint may be excised, or amputation may be required. H. A. Wilson advocates lateral excision. By means of bone-forceps he cuts away that part of the distal extremity beyond the phalanx, and by a chisel removes the remaining

sharp line edge. He places the phalanx in normal position and holds it so for two weeks ("Am. Jour. Orthopedic Surgery," Jan., 1906). Mayo's operation is described on page 732.

**Hammer-toe** (Figs. 479 and 480) is a condition in which there is flexion of one or more toes at the first interphalangeal joint. Shattuck shows that this condition is due to contraction of "the plantar fibers of the lateral ligaments of the joint."[1] This disease usually begins in youth and may be congenital. A bunion is apt to form, and the joint may become dislocated.

**Treatment.**—Subcutaneous division of the lateral ligaments and flexor tendon usually allow of straightening of the toe. After the operation the toe must be held in extension for several months by means of a short splint and adhesive plaster. Terrier's plan of treatment consists in making a dorsal flap, removing a bursa if one is found, dividing the extensor tendon, opening the articulation, removing each articular surface by cutting forceps, suturing the soft parts, and applying a plantar splint for two weeks.[2] Some surgeons simply excise the joint. In some cases amputation of the toe is the best treatment.

Fig. 479.—Hammer-toe.

Fig. 480.—*X*-ray of hammer-toe.

**Metatarsalgia (Morton's Disease).**—This disease was first described by Dr. Thomas G. Morton, of Philadelphia, in 1876. It is a painful condition of the foot, due to jamming of a nerve between the heads of the fourth and fifth metatarsal bones. The head of the fifth metatarsal bone is, by lateral pressure, forced against and below the neck of the fourth metatarsal, and as a result the superficial branch of the external plantar nerve and its two digital branches are squeezed. It is usually associated with flat-foot. Loss of the metatarsal arch will jam other nerves than that between the fourth and fifth metatarsals, and will produce a condition similar to Morton's disease, the pain being differently situated. Pain, in Morton's disease, is produced by walking, and the suffering may be so severe that the patient is obliged to sit down at once. When the shoe is removed and the foot is rested the pain soon abates. The pain is felt on the outer and inner sides of the little toe, the outer side of the fourth toe, and about the head of the fifth and the neck of the fourth metatarsal bones. Pain can be developed by grasping the foot in the hand and squeezing it. If flat-foot exists, there is also pain due to this trouble.

**Treatment.**—Mild cases may be cured by wearing well-fitting shoes and employing massage. The shoe should have a small elevation in the sole to restore the metatarsal arch and so relieve lateral pressure on the nerve. Some cases require a brace. Severe cases demand resection of the fourth metatarsophalangeal joint, or amputation of the fourth toe, and with it the head of the fourth metatarsal bone. Graham, of Washington, has cured cases by excising a portion of the superficial branch of the external plantar nerve.

**Coxa Vara and Coxa Valga.—Coxa vara** (*incurvation* or *infraction of the neck of the femur*) (Fig. 481) is a disease characterized by bending of the neck of the femur, the femoral neck being depressed below its normal obtuse angle with

[1] "American Text-Book of Surgery."
[2] "Rev. de Chir.," July, 1895.

the shaft, the hip-joint being perfectly healthy, and the condition, as a rule, being unilateral, but sometimes bilateral. This condition was described by Müller in 1889. Coxa vara is first noticed, as a rule, between the thirteenth and twentieth years, and the commonly accepted view has been that the deformity is rachitic, but Kredel has reported 2 congenital cases.[1] Traumatic coxa vara may follow impacted fracture of the neck of the femur in a child. An individual with coxa vara develops a limp, and grows tired after slight exertion, but there is no swelling, no tenderness, and little or no pain. Shortening after a time becomes apparent, the great trochanter can be detected above Nélaton's line, and the head of the bone is in the acetabulum. If the head is in the acetabulum it can be recognized as being there by locating the femoral artery ½ inch below Poupart's ligament and making pressure directly backward at that point. The head, if in place, will be felt. The extremity is adducted and usually rotated outward. Abduction is limited. In some cases in which there is joint irritation all joint motions may be distinctly limited.

Fig. 481.—Congenital dislocation of the hip on one side and coxa vara on the other.

In a bilateral case there is lordosis, but shortening may not be detected because both legs are the same length. Each great trochanter, however, is above Nélaton's line and the head of each bone is in the acetabulum.

**Coxa valga** is a condition in which the angle of the neck to the shaft of the femur is more obtuse than normal. The neck may assume a position in line with the long axis of the shaft. It occurs particularly in children who have had infantile palsy, but it may be congenital, may occur in rickets and osteomalacia, and after a prolonged period of disuse of a limb. The patient has pain and a limp, the extremity is lengthened, abducted, and in external rotation there is limitation of adduction and the trochanter is flattened. Coxa valga is usually unilateral, but may be bilateral.

Without the *x*-rays the differential diagnosis in coxa vara and coxa valga is often very difficult, and the accompanying table will materially aid in contrasting the conspicuous features of the various conditions with which these two deformities of the neck of the femur may be confounded:

[1] "Centralbl. f. Chir.," Oct. 17, 1896.

| | Hip Disease. | Infantile Paralysis. | Congenital Dislocation. | Psoas Abscess. | Coxa Vara or Valga. | Knee Disease. |
|---|---|---|---|---|---|---|
| Age. | Four to six. | Four to six. | Any age. | Four to six. | Any age. | Four to six. |
| Onset. | Insidious. | Sudden. | From birth. | Insidious. | Childhood. | Insidious. |
| Pain. | Referred to knee. | None. | None. | Referred to abdomen. | None. | Referred from knee. |
| History. | Tuberculous. | Inflammatory disease. | Limp from birth. | Tuberculous. | Limp. | Tuberculous. |
| Posture. | Flexion, abduction, external rotation. | Uncontrolled. | Shortening and adduction. | Flexion, adduction, external rotation. | Great trochanter higher in vara and lower in valga. | Knee flexed. |
| Muscular rigidity. | Present in all directions. | Absent. | Absent. | In one direction. | None. | About knee. |
| Temperature. | 1 degree high. | Normal. | Normal. | 1 degree. | Normal. | 1 degree. |
| Local tenderness. | In hip. | None. | None. | In spine. | None. | In knee. |
| Night cries. | Present. | Absent. | Absent. | Present. | Absent. | Present. |
| Tendency to abscess. | Yes. | No. | No. | Yes. | No. | Yes. |
| *X*-ray. | Diseased focus in hip. | Atrophy. | Alteration in joint. | Normal hip. | Alteration in neck angle. | Normal hip, focus in knee. |

The *x*-rays clearly show the deformed bone in either coxa vara or coxa valga.

**Treatment.**—In coxa vara, as long as bending is progressing, employ rest. When the bone hardens, it may be necessary to perform osteotomy below the trochanters. In coxa valga Galeazzi performs osteotomy through the neck of the femur and allows the trochanter to ascend.

**Flail-joints.**—After an attack of infantile paralysis involving the entire lower extremity of each side the limbs become limp and swing flail-like when the extremity is made to move, and the joints are much relaxed. In such cases the psoas and iliacus muscles are never completely paralyzed, and the aim of the surgeon is to utilize these muscles in enabling the patient to walk. In many cases the application of apparatus is sufficient. In others ankylosis may be established in the ankles and knees by operation. If ankylosis is established in these joints, the psoas and iliacus muscles become able to move the legs. (For more elaborate discussion, see a work on Orthopedic Surgery, and Tubby and Jones on the "Surgery of Paralysis.")

## XXIII. DISEASES AND INJURIES OF NERVES

### Diseases of Nerves

**Neuritis,** or **inflammation of a nerve,** may be limited or be widely distributed (*multiple neuritis*). The first-mentioned form will here be considered. The causes of neuritis are traumatism, wounds, overaction of muscles, gout, rheumatism, syphilis, fevers, and alcoholism.

The **symptoms** of neuritis are as follows: excessive pain, usually intermittent, in the area of nerve-distribution. The pain is worse at night, is aggravated by motion and pressure, and occasionally diffuses to adjacent nerve-areas or awakens sympathetic pains in the opposite side of the body. The nerve is very tender. The area of nerve-distribution feels numb and is often swollen. Early in the case the skin is hyperesthetic; later it may become anesthetic. The muscles atrophy and present the reactions of degeneration; that is, the muscles first cease to respond to a *rapidly* interrupted faradic current, and next to one with slower interruptions; faradic excitability diminishes, but galvanic excitability increases. When, in neuritis, faradism produces no contraction, a slowly interrupted galvanic current which is so weak that it would produce no movement in the healthy muscle causes marked response in the degenerated muscle. In health the most

vigorous contraction is obtained by closing with the − pole; in degenerated muscles the most vigorous contraction is obtained by closing with the + pole. When voluntary power returns, galvanic excitability declines; but power is often nearly restored before faradic excitability becomes manifest (Buzzard).

The **treatment** of neuritis consists of rest upon splints and the use of an ice-bag early in the case and a hot-water bag later. Blisters over the course of the nerve are of value, especially in traumatic neuritis. Massage and electricity must be used to antagonize degeneration. A descending galvanic current allays pain to some extent. Deep injections of chloroform or cocain may allay pain. Treat the patient's general health, especially any constitutional disease or causative diathesis. The salicylate of ammonium or phenacetin may be given internally. In some cases nerve-stretching is advisable.

**Neuralgia** is manifested by violent paroxysmal pain in the trajectory of a nerve. This disease, unless it is exceedingly severe and persistent, is treated, as a rule, by the physician. Injections of alcohol or osmic acid into the nerve may secure relief or cure. If neuralgia is due to adhesions about the nerve or to the pressure of scar or callus, these conditions should be amended surgically. There is some evidence that neuralgia arises, at least occasionally, from dilatation of the vessels of the sheath and subsequent edema and exudation. If this be the case cure may follow opening the sheath and separating adhesions between the sheath and nerve (Robert M. Simon, in "Brit. Med. Jour.," April 10, 1909). Neuralgia of stumps and scars is a surgical condition, and is due to neuromata, or entanglement of nerve-filaments in a cicatrix. *Tic douloureux* and other intractable neuralgias require careful removal of any cause of reflex irritation. Causal reflex irritation may arise from disease of the stomach, eyes, teeth, uterus, nose, throat, etc. Tic douloureux has been treated by removal of the Gasserian ganglion; intracranial neurectomy of the second and third divisions; division of the sensory root; removal of Meckel's ganglion; ligation of the common carotid artery; neurectomy of terminal branches of the fifth nerve; division of motor nerves; injections of osmic acid (see page 762); injections of alcohol (see page 763); advancing doses of strychnin (Dana) and purgatives (Esmarch). The distribution of the fifth nerve, the seat of pain in tic douloureux, is shown in Fig. 482.

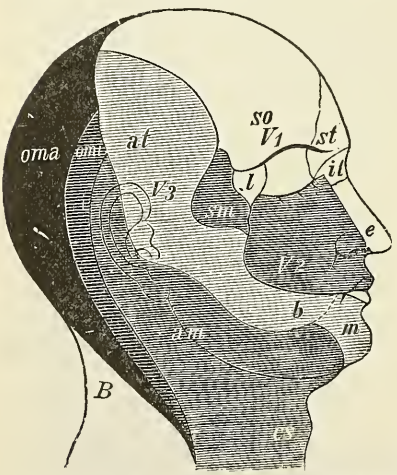



Fig. 482.—Distribution of the cutaneous sensitive nerves upon the head: *oma, omi,* The occipit. maj. and minor (from the N. cervical. II and III); *am,* N. auricular magn. (from N. cervic. III); *cs,* N. cervical. superfic. (from N. cervic. III); $V_1$, first branch of the fifth (*so,* N. supraorbit.; *st.* N. supratrochl.; N. infratrochl.; *e,* N. ethmoid.; *l,* N. lachrymal.); $V_2$, second branch of the fifth (*sm,* N. subcutan. malæ seu zygomaticus); $V_3$, third branch of the fifth (*at,* N. auriculotempor.; *b,* N. buccinator; *m,* N. mental.); *B,* posterior branches of the cervical nerves (Seeligmüller).

**Treatment of Neuralgia of Stumps.**—Excise the scar; find the bulbous end of the nerve and cut it off. Senn tells us to section the nerve by V-shaped cuts, the apex of the V being toward the body, and to suture the flaps together. Senn's method will prevent recurrence. In some cases reamputation is performed. In entanglement of a nerve in a scar remove a portion of the nerve above the scar and also the neuroma in the scar.

## Wounds and Injuries of Nerves

**Section of Nerves** (as from an incised wound).—After nerve-section sensation and motor power are lost at once. The entire peripheral portion of the nerve degenerates and ceases structurally to be a nerve in a few weeks, but after many months, or even years, the nerve may regenerate. The proximal end degenerates only in the portion immediately adjacent to the section; it rapidly regenerates, and if it does not adhere to the peripheral segment a bulb or enlargement composed of fibrous tissue and small nerve-fibers forms just above the line of section; this bulb adheres to the perineural tissues. The entire distal end degenerates, but new axis-cylinders form in this segment by proliferation of the nuclei on the sheath of Schwann. Union of a divided nerve is brought about by the projection of axis-cylinders from the proximal end or from each end and the fusion of these cylinders. The nearer the two ends are to each other, the better the chance of union. When a nerve has been divided and has not been sutured, abolition of function may be permanent or restoration may occur. Sensation may return in from six weeks to several months. Motor power may never return. If it does return it will do so long after sensory restoration. Restoration of motor power requires from twelve weeks to three and one-half years. It is seldom noted before six months. The return is always slow (John B. Murphy, in "Surg., Gynec., and Obstet.," April, 1907). Failure of return means that the ends are separated by a wide interval or that fascia is interfused between them. In some recorded cases motion and sensation have returned with great rapidity, due, some have said, to anastomoses with adjacent nerves. Murphy shows that when restoration begins, trophic energy returns even before sensation, and when trophic energy returns the blueness and coldness of the limb lessen.

The nerve-fibers which convey impressions of cutaneous pain and of extreme heat and cold regenerate far more rapidly than those which subserve sensations of light touch and slight degrees of heat and cold.

The investigations of Head and others show that restoration of sensation does not begin at the normal area and spread from there over the anesthetic area, but that the reverse is the case. It begins from the confines of the anesthetic area and spreads toward the normal part.

**General Symptoms.**—Immediately after nerve-section vasomotor paralysis comes on, and for a few days the paralyzed part presents a temperature higher than normal. It then becomes blue and cold. Pronounced changes occur in the trajectory of a divided nerve. The muscles degenerate, atrophy and shorten, and develop the reactions of degeneration. When union of the nerve occurs the muscles are restored to a normal condition. If the nerve contains sensory fibers, complete anesthesia (to touch, pain, and temperature) usually follows its division; but if a part is supplied by another nerve as well as by the divided one, anesthesia will not be complete. Trophic changes arise in the paralyzed parts. Among these changes are muscular atrophy; glossy skin; cutaneous eruptions; ulcers; dry gangrene; painless felons; falling of the hair; brittleness, furrowing, or casting off of the nails; joint inflammations, and anklylosis. The diagnosis as to which nerve is cut depends upon a study of the distribution of motor and sensory paralysis.

A curious fact that was pointed out by Letiévant is that after division of a nerve blunt pressure may be appreciated over the entire analgesic area. This phenomenon was long thought to be due to nerve anastomoses, the sensory areas from different nerves overlapping. The explanation now given is founded on Sherrington's demonstration that the motor branches of a mixed nerve carry sensory fibers through muscles and tendons, and Head's proof that certain afferent fibers convey impressions of deep sensibility as produced by pressure.

**The Symptoms of Division of Nerves.**—*Brachial Plexus.*—If one or more cords of the brachial plexus are divided, motor paralysis and anesthesia appear in the limb, the extent of the paralysis and the area of the anesthesia depending upon the cord or cords involved. It should be remembered that the inner cord of the brachial plexus gives origin to the ulnar nerve; the inner and outer cords give branches which fuse to form the median nerve. The posterior cord gives origin to the subscapular, the circumflex, and the musculospiral nerves. The outer cord gives origin to the external anterior thoracic and the musculocutaneous, as well as to the outer trunk of origin of the median.

*Avulsion* or *rupture of the brachial plexus* is sometimes effected by an injury, when the arm is not lost. Most cases are due to indirect violence and are associated with skeletal injury, for instance, dislocation of the shoulder, fracture of the humerus, or fracture of the clavicle (Frazier and Skillern, paper before Section on Surgery, "Amer. Med. Assoc.," 1911). Bristow ("Annals of Surgery," Sept., 1902) reported 3 cases of avulsion of the plexus and collected 24 more. Frazier and Skillern (Loc. cit.) collected 23 cases of avulsion without skeletal injuries and confirmed by operation. In each of these cases the rupture was between the clavicle and the transverse process of the vertebra. In the case of Frazier and Skillern the rupture was within the dura. The patient was seen by them three months after the accident. He suffered from horrible pain in the arm and hand. Sensations to touch and pain were entirely lost in the hand and forearm and in the arm to 2 or 3 inches above the elbow. From this point to about 4 inches below the shoulder they were much impaired. Sensation was impaired in the entire distribution of the intercostohumeral nerve, and of the fifth and sixth cervical roots, as well as in the distribution of the brachial plexus. There was flaccid palsy of the entire extremity, including the deltoid. On the injured side the pupil was contracted and the palpebral fissure narrowed. One of Bristow's cases was operated upon the third day after the accident (Loc. cit.). In this case there was complete paralysis of the upper extremity, with the exception of the sensory area of the intercostohumeral and the circumflex nerves. The accident had been inflicted by the patient's forearm becoming entangled in a rope, which was pulled upon by a steam winch. On reaching the hospital he felt severe pain, referred to the arm. There was much swelling in the inner portion of the subclavian triangle, the left pupil was contracted, and it seemed likely that the nerves had been avulsed close to the intervertebral foramina. From the fact that sensation was preserved in the skin of the convexity of the shoulder down to the insertion of the deltoid, Bristow concluded that some fibers of the posterior cord of the plexus had escaped division; but when the operation was performed this conclusion was found to be erroneous. An incision was made, and it was found that the plexus had given way at the point where the four cervical nerves and the last dorsal unite to form the three trunks. In order to reach the lower ends it was necessary to saw the clavicle and divide the two pectoral muscles; and the torn ends of the nerve-trunks were found underneath the clavicle. Suturing was performed. The ends of the sawn clavicle were sutured together, the wound was closed and dressed, and the arm was put up in Sayre's dressing.

After the performance of this operation sensation over the entire upper arm returned. The author once operated upon a patient that had developed paralysis, motor and sensory, after violent stretching of the arm. In the light of Bristow's case I assumed that avulsion of the plexus had probably taken place. Incision disclosed the fact that the plexus was intact, but was surrounded with dense scar-tissue. The tissue was removed, so as to loosen the nerves, which felt like hard cords; but I have lost track of the patient, and do not know the result. My patient was operated upon many months after the injury.

I operated upon another case and found intravertebral rupture. I anastomosed two divided nerves to a sound one. The operation was done months after the injury and the patient is apparently slightly improved three months after operation. The location of the seat of injury is of great importance. Lesion within the vertebræ is positively indicated by contraction of the pupil, narrowing of the palpebral fissure, and enophthalmos on the affected side (Frazier and Skillern, "Amer. Med. Assoc.," 1911), but there can be intravertebral injury without these phenomena. The ciliospinal fibers on which these phenomena depend usually accompany the eighth cervical and first thoracic nerves.

Injury above the clavicle will involve the circumflex and musculospiral. As the great pectoral is supplied from both the external and internal cords of the plexus, complete paralysis of the great pectoral proves that both cords are involved, and unimpaired movements of the diaphragm on the side of the lesion (observed with the fluoroscope) show that the cords of the plexus are not divided within the foramina, but well outside of them (John B. Murphy, in "Surg., Gynec., and Obstet.," April, 1907). In all cases of rupture prompt operation is indicated. Long delay means a hopeless prognosis.

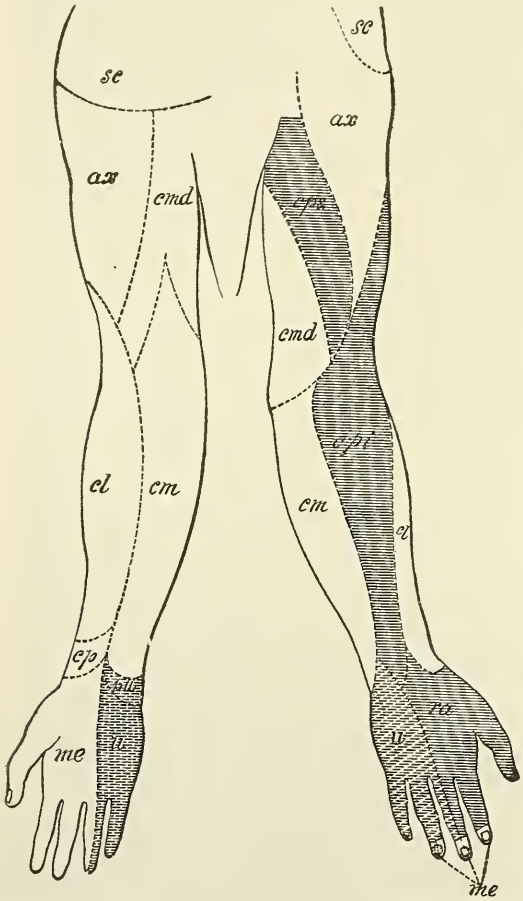
In injury without complete rupture the nerves become fibrous and embedded in scar-tissue. If operation is done before scar-tissue forms follow the advice of Frazier and incise the sheaths of the trunks or cords to liberate inflammatory exudate. If scar-tissue has formed, excise it. It may be necessary to excise portions of nerves or trunks with it. If this is done, suture the divided ends together. It may be necessary to do nerve-lengthening, suture à distance, or grafting in of a nerve from a recently amputated limb (if one can be obtained), or the sciatic of a rabbit. In accessible rupture suture the divided nerves. In some cases a divided cord or trunk may be anastomosed into a sound one. Ruptures near to, in, or within the intervertebral foramina do not admit of suturing. If a sound nerve is left the divided nerve or nerves should be anastomosed to it. If the entire plexus is ruptured we should consider the plan of Alexinsky (quoted by Frazier and Skillern, Loc. cit.): effect anastomosis between nerves of the injured side and nerves of the sound side. When intractable neuralgia follows rupture the posterior roots may be divided.

*Brachial Birth Palsy.*—It has been pointed out by Clark, Taylor, and Prout ("Am. Jour. Med. Sciences," Oct., 1905) that brachial birth palsy results from tension on the nerve-trunks by overstretching during delivery, the nerve-sheath first rupturing and then the nerve-fibers. When the sheath ruptures hemorrhage occurs, fibrous tissue forms, and the scar presses on the intact, slightly stretched, or actually lacerated nerve, and prevents repair. The authors tell us that the fifth cervical root first gives way, then the sixth, and so on down the plexus if there is sufficient force. In the milder cases the fifth root alone suffers. They call it brachial birth palsy, or *laceration palsy*, and sum up the symptoms in a severe case as follows: The arm hangs powerless; abduction at the shoulder is impossible because of deltoid and supraspinatus palsy; the forearm is extended and flexion is impossible because of biceps, brachialis anticus, and supinator longus palsy; palsy of supinator brevis and biceps causes pronation of hand; there is inward rotation of the humerus because of palsy of the supraspinatus, infraspinatus, and teres minor.

Brachial birth palsy is manifest soon after its infliction by evidences of pain on handling the extremity, the pain being due to neuritis (authors above quoted). Medical treatment is relied on for one year, and then, if improvement is not manifest, operation is indicated (see page 769).

*Posterior (Long) Thoracic Nerve.*—Division of this nerve causes paralysis of the serratus magnus muscle, which is made evident by eversion from the thorax and rotation of the scapula when the arm is taken forward (*wing-like scapula*). In paralysis of this muscle the arm cannot be raised above the horizontal.

*Suprascapular Nerve.*—Division of this nerve produces some anesthesia over the scapula and paralysis of the supraspinatus and the infraspinatus muscles. The supraspinatus is but an adjuvant to the deltoid and palsy of it is not manifestly disabling. Palsy of the infraspinatus renders external rotation of the humerus impossible, and writing becomes most difficult because the pen cannot be moved along the paper. Sewing, too, is greatly interfered with.




Anterior surface. Posterior surface.

Fig. 483.—Distribution of the cutaneous nerves to the shoulder, arm, and hand. The region of the N. radialis is represented by the unbroken hatched line, that of the N. ulnaris by the broken hatched lines. *a*, Anterior, *b*, posterior surface; *sc*, Nn. suprascapular (plexus cervicalis); *ax*, chief branch of N. axillar.; *cps*, *cpi*, Nn. cutanei post. sup. and inf. (from N. radialis); *ra*, terminal branches of N. radialis; *cm*, *cl*, Nn. cutanei medius (also to the plexus) and lateralis (chiefly to the N. medianus); *cp*, N. cutan. palmar., N. rad.; *cmd*, N. cutan. medialis; *me*, N. medianus; *u*, N. ulnaris; *epu*, N. cutan. palm. ulnaris (Henle).

*Circumflex Nerve.*—Division of the circumflex nerve produces paralysis of the deltoid muscle, so that it becomes impossible to lift the arm to a right angle with the body. There is some slight retention of power in the anterior fibers, which are supplied by the anterior thoracic nerve. The skin over the lower part of the muscle is usually anesthetic.

*Musculocutaneous Nerve.*—Division of this nerve produces paralysis of the biceps and of the brachialis anticus muscles (paralysis of the forearm flexors). This palsy becomes especially evident when the forearm is supinated, because in this position the supinator longus can no longer act as a flexor of the elbow. There is anesthesia of the radial side of the forearm anteriorly and posteriorly.

*The Musculospiral or Radial Nerve.*—Division of this nerve high up near the plexus causes paralysis of the extensor muscles of the elbow and the wrist, of the supinators, and of the long extensors of the thumb and fingers. When divided near the middle of the humerus, the triceps usually, but not invariably, escapes. If the injury is below the branch going to the supinator longus, that muscle will escape; otherwise it will become paralyzed. The extensor palsy causes wrist-drop and loss of the power of extending the first phalanges of the fingers and thumb; and, as Gowers has pointed out, flexion is reduced to one-third of the normal, the flexors having lost power "from the loss of antergic support." As a rule, in musculospiral palsy there is loss

of supination. Sensibility is sometimes greatly affected, and sometimes very slightly. If the injury is above the level of the musculospiral groove there will be anesthesia in the area supplied by the sensory fibers of the nerve (Fig. 483). If the nerve is injured in the musculospiral groove there are seldom sensory disturbances. Anesthesia rarely occurs in the upper arm in any case, and even after an injury in the groove sensation in the hand may be normal or nearly so. Fig. 484 shows the position of the parts in musculospiral palsy and Figs. 483 and 485 the sensory distribution of the nerve. Fracture of the humerus may cause division of the musculospiral nerve.

*The Median Nerve.*—After division of this nerve there is paralysis of the pronators; the flexor carpi radialis; the finger flexors, except the ulnar portion of the deep flexor; the abductors, and the flexors of the thumb; and the two radial lumbricales. The forearm can be placed in a position midway between pronation and supination; but further pronation cannot be voluntarily effected. In executing flexion of the wrist a strong deviation toward the ulnar side takes place. The thumb is in a position of extension and abduction, and cannot be brought into apposition with the finger-tips. The second phalanges of the fingers cannot be flexed on the first, and the distal phalanges

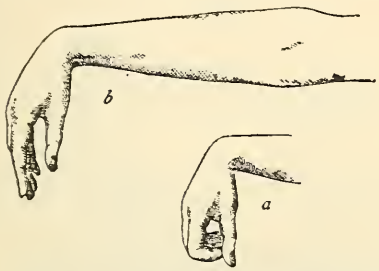



Fig. 484.—Paralysis of musculospiral nerve after fracture of the humerus ("wrist-drop"); but when fingers have been flexed into palm, *a*, they can be extended, *b*, at first interphalangeal joints by lumbricals and interossei, which are supplied by the ulnar and median nerves (Erichsen).

Fig. 485.—Distribution of sensory nerves on the backs of the fingers: *r*, Musculospiral or radial nerve; *u*, ulnar nerve; *m*, median nerve (Krause).

of the first and second fingers cannot be voluntarily flexed. The corresponding phalanges of the third and fourth fingers can be flexed, this being accomplished by the unparalyzed ulnar half of the deep flexor. Flexion of the first phalanges is still possible, as it is accomplished by means of the interossei. The extensor action of the interossei muscles upon the middle and distal phalanges, being unopposed, may eventually cause subluxation. The sensory distribution of the median nerve is shown in Figs. 483 and 485–487. It is the sensory nerve of the radial side of the palm, the front of the thumb, the first and second fingers and half of the third finger, and the back of the last phalanx of the index and the middle finger (Gowers). The sensory changes after median paralysis are quite variable—sometimes widespread and complete, at other times trivial, and occasionally absent. Gowers says that if there is anesthesia it is usually of the palmar surface, but it may also occur on the dorsal aspect of the ends of the first two fingers.

*The Ulnar Nerve.*—When this nerve is divided there is paralysis of the flexor carpi ulnaris, of the ulnar portion of the deep flexor, of the muscles of the little finger, of the abductor pollicis, and of the inner end of the flexor brevis pollicis (Gowers). It becomes impossible to adduct the thumb, and the majority of the movements of the little finger are abolished. Flexion of

the fingers is impossible at the first joints, and extension is impossible at the other joints; but, as Gowers points out, the loss is slighter in the first two

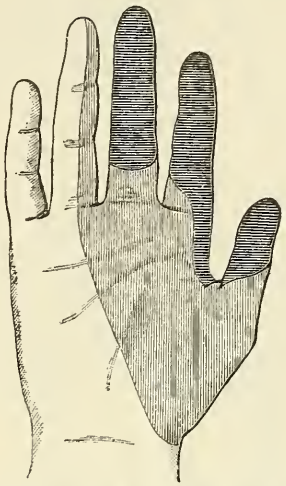


Fig. 486.—Section of median nerve; areas of anesthesia (heavy shading) and of dysesthesia (light shading) on palmar surface of hand (Bowlby).

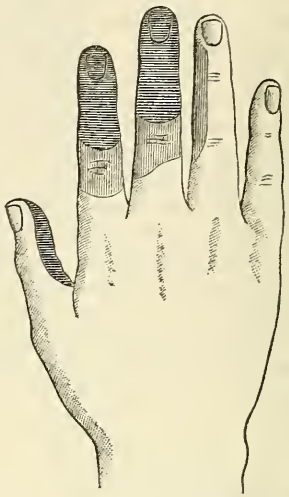


Fig. 487.—Section of median nerve; regions of anesthesia and dysesthesia on dorsal surface of hand (Bowlby).

fingers than in the others because the lumbricales of the first two fingers are supplied by the median nerve. Interosseal flexion is impossible, and the op-

Fig. 488.—Division of ulnar nerve.

ponents of the interossei, acting without normal antagonism, contract and produce what is known as *claw-hand* (Figs. 488, 489), a condition in which the

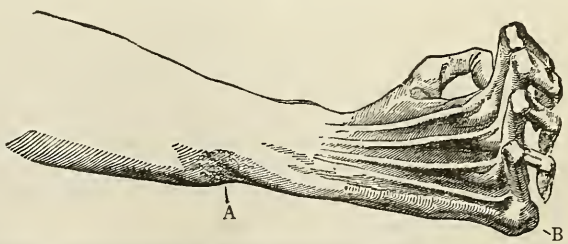



Fig. 489.—Paralysis of ulnar nerve from wound at *A*; contracture of common extensor with posterior luxation of first phalanges; *B*, head of metacarpal bone (Duchenne).

first phalanges are overextended and the others are flexed. The sensory loss in ulnar paralysis is extremely variable. The sensory distribution is

to the ulnar side of the hand, both back and front, involving the little finger, the ring-finger, and the ulnar half of the middle finger (Figs. 483, 485, 490, and 491).

The *lumbar plexus* supplies the cutaneous surface of the lower portion of the abdomen, of the front and the sides of the thigh, and of the inner portion of the leg and foot (Fig. 492). It innervates the flexors and adductors of the hip-joint, the extensors of the knee, and the cremaster muscle. The branches sent to the leg are the obturator and the anterior crural nerves.

The *sacral plexus* supplies the extensors and rotators of the hip, the knee-flexors, and all the muscles of the foot; also the skin of the gluteal region, the back of the thigh, the outer portion and the posterior part of the lower leg, and most of the foot (Gowers) (Fig. 492). Its chief branches are those to the external rotators of the hip—the gluteal nerve, the small sciatic, and the great sciatic.

*The Anterior Crural Nerve.*—When this nerve is divided the extensor muscles of the knee are paralyzed. The psoas muscle is not affected, even if

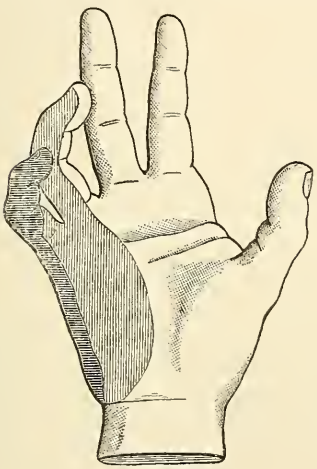


Fig. 490.

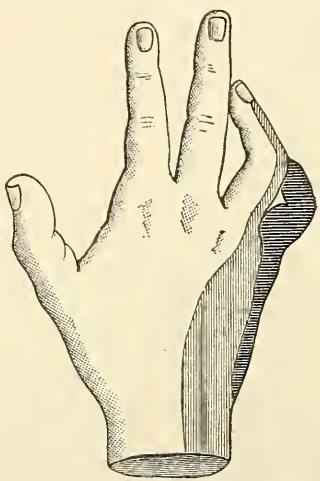


Fig. 491.

Figs. 490, 491.—Showing sensory loss and ordinary position in injuries of the ulnar nerve (Bowlby).

the nerve is divided within the abdomen; but high division may produce paralysis of the iliacus muscle. In anterior crural palsy the skin is anesthetic over almost the entire thigh, the inner surface of the leg and foot, and the inner sides of the first and second toes (Fig. 492).

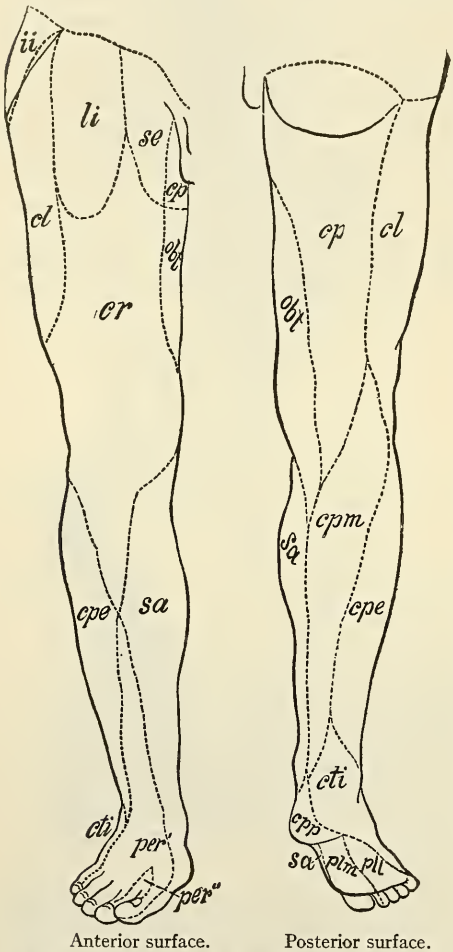
*The Obturator Nerve.*—In obturator palsy the adductor muscles of the thigh are paralyzed, and, in consequence, the patient is unable to cross one leg over the other. Gowers points out that external rotation of the thigh is also interfered with.

*The Superior Gluteal Nerve.*—The division of this nerve paralyzes the gluteus medius and the gluteus minimus muscles, and there is "loss of abduction and circumduction of the thigh" (Gowers).

*The Small Sciatic Nerve.*—Division of this nerve paralyzes the gluteus maximus muscle and produces anesthesia of the upper half of the calf of the leg and of the middle third of the back of the thigh (Gowers) (Fig. 492).

*The Great Sciatic Nerve.*—If this nerve is divided near the sciatic notch there is a paralysis of the flexor muscles of the leg. These muscles, as Gowers points out, are also extensors of the hip. There is likewise paralysis of all the

muscles below the knee. If, however, the injury is below the upper third of the thigh, there is no paralysis of the flexors of the leg. If the nerve is damaged on a level below the small sciatic, there is anesthesia of the outer portion of the leg, of the sole of the foot, and of most of the dorsum of the foot (Fig. 492).




Anterior surface. Posterior surface.

Fig. 492.—Distribution of the cutaneous nerves of the lower extremity: *ii*, N. ilio-inguinal. (plex. lumb.); *li*, N. lumbo-inguinal. (to the genitocrural, plex. lumbal.); *se*, N. spermat. ext. (to the genitocrural); *cp*, N. cutan. post. (plex. ischiad.); *cl*, N. cutan. lateral. (plex. lumb.); *cr*, N. cruralis (plex. lumbal.); *obt*, N. obturator. (plex. lumb.); *sa*, N. saphen. (plex. lumbal.); *cpe*, N. commun. peron. (N. peron. tibial.); *cti*, N. commun. tibial; *per'*, *per''*, N. peronæi ram. superfic. et prof.; *cpm*, N. cutan. post. med. (plex. ischiad.); *cpp*, N. cut. plant. propr. (N. tib.); *plm*, *pll*, N. plantar. medial. et lateral. (N. tib.) (Henle).

*The External Popliteal Nerve.*—When this nerve is divided, there is paralysis of the tibialis anticus muscle, the extensor longus digitorum, the extensor brevis digitorum, and the peronei; and the patient is unable to flex the ankle and extend the first phalanges of the toes. When he tries to walk he cannot lift the foot from the ground; and eventually there is the development of talipes equinus (Gowers). The anesthesia is manifest on the outer portion of the anterior surface of the leg, and also on the dorsum of the foot (Fig. 492).

*The Internal Popliteal Nerve.*—Damage to this nerve paralyzes the posterior tibial muscle, the flexor longus digitorum, the muscles of the calf, the popliteus muscle, and the muscles of the plantar surface of the foot. The toes become flexed at the two distal joints, and extend at the proximal joints. Walking is greatly interfered with. There is loss of the power of rotating the flexed leg inward, if the damage is above the branch to the popliteus muscle; and extension of the ankle-joint is lost. As the consequence, talipes calcaneus develops (Gowers). The anesthesia is variable, but usually involves the sole of the foot and the outer surface and lower portion of the back of the leg (Fig. 492).

*The Plantar Nerves.*—Division of the internal plantar nerve paralyzes the short-toe flexor, the two inner lumbricales, and the plantar muscles of the great toe, except the adductor (Gowers). There is anesthesia of the inner portion of the sole of the foot and of the plantar surface of the three inner toes and of half of the fourth toe (Fig. 492).

Division of the external plantar nerve causes paralysis of the muscles of the little toe, of the adductor of the great toe, of all the interossei, of the two outer lumbricales, and of the flexor accessorius (Gowers). There is

anesthesia of the skin of the outer half of the sole of the foot, of the little toe, and of half of the fourth toe (Fig. 492).

*The Facial Nerve.*—This nerve may be divided during the mastoid operation or may be lacerated by a fracture of the petrous portion of the temporal bone, and a peripheral palsy results. The face is asymmetrical and is drawn to the sound side. Asymmetry becomes more marked on attempting to smile or to show the teeth. Whistling and frowning are impossible. The sense of taste may be less acute or lost on the anterior two-thirds of the tongue. There is relaxation of the palate and deviation of the uvula. Reactions of degeneration can be demonstrated in the palsied muscles. In some cases there are sensory disturbances (hyperesthesia or anesthesia) and in some there are vasomotor perturbations on the palsied side. On the paralyzed side the muscles are relaxed, the nasolabial fold is to a great extent gone, the nostril cannot be dilated. The brow wrinkles have been smoothed out, the eyelids cannot be closed; on attempting to close the eye the globe tilts upward and outward. The cornea and conjuntiva inflame, the lower lid droops, and tears run down the cheek.

**Treatment.**—In every wound in which a nerve or nerves might be damaged make careful search and examination to determine the matter. Always suspect nerve injury in wounds of the wrist. In all recent cases of nerve-section try, if possible, to suture the ends of the divided nerve. The earlier suture is done the better the chance for restoration of function. For instance, in fracture of the humerus, with division of the musculospiral, operate at once. After-care is of the greatest importance in all cases of nerve suture. Primary suture means suture within twenty-four hours of the accident. Suture may fail. There may be partial or complete restoration of function. In 123 reported cases of *primary suture,* 119 were cured in from one day to one year (Willard). The return of sensation may be rapid or may be slow; muscular power returns more slowly than sensation. If the patient is not seen until long after the accident, incise and apply sutures (*secondary sutures*); if the nerve cannot be found, extend the incision, find the trunk above and trace it down, and find the trunk below and follow it up. The results are not nearly so good as are those of primary suture. After-care for months is highly important. In 130 reported cases of secondary suture 80 per cent. were more or less improved (Willard). Even after primary suture loss of function is bound to occur for a time. After secondary suture sensation may return in a few days, but it may not return until after a much longer period; in any case muscular function is not restored for months. After partial section of a nerve the ends should be sutured. In performing secondary suture it may be necessary to effect *lengthening* in order to approximate the ends (see page 759). *Transplantation* of a portion of nerve is sometimes practised (*implantation* or *anastomosis*). *Nerve-grafting* is bridging the gap by means of a portion of nerve from one of the lower animals or from a recently amputated human limb. Nerve-transplantation may fail utterly; it may be followed by great improvement, but absolute and perfect restoration of function cannot be obtained. R. Peterson[1] has made a study of the 20 recorded cases of nerve-grafting; 8 of the operations were primary and 12 were secondary. The periods after the injury at which operation was performed varied from forty-eight hours to a year and a quarter; 4 of the 8 primary cases improved; 8 of the 12 cases of secondary operation showed improvement in motion or sensation. The distance between the nerves did not seem to affect the results. No case recovered completely, but in 1 case sensation returned completely and only the abductors of the thumb remained weak. In most of the cases that were benefited sensation returned by the tenth day and motion within two and a half months. In one of the successful cases, that of A. W.

[1] "Amer. Jour. Med. Sciences," April, 1899.

Mayo Robson,[1] the spinal cord of a rabbit was used. A facial nerve divided in the aqueduct of Fallopius may perhaps be sutured at the site of the injury. This should be attempted as soon as the palsy is observed, as was suggested, I believe, by Frederick Sydenham ("Brit. Med. Jour.," May 8, 1909). If the ends of the divided nerve cannot be approximated, *suture à distance* may be practised, as was done successfully by Sydenham (Ibid.). If suture at the site of injury is impossible the end of the peripheral segment of the divided nerve may be anastomosed to the hypoglossal, glossopharyngeal, or spinal accessory nerve (see page 760).

**Pressure upon nerves** may arise from callus, scars, a dislocated bone, a tumor, or an external body.

The **symptoms** may be anesthetic, paralytic, or trophic.

The **treatment** is as follows: Remove the cause (reduce a dislocated bone, chisel away callus, excise a scar, etc.); then employ massage, douches, exercise, and electricity.

**Dislocation of the Ulnar Nerve at the Elbow.**—This condition is very rare. It may occur as a complication of a fracture or a dislocation, or as an uncomplicated condition. It may be produced by violence or by muscular effort, which ruptures the fascia, the function of which is to retain the nerve back of the inner condyle of the humerus. In some cases the symptoms are slight and transitory, the nerve functionating well in its new situation. As a rule, there are pain, numbness, or anesthesia of the ulnar trajectory, some stiffness of the elbow, and stiffness of the little finger and ring-finger. The nerve can be felt in front of the inner condyle of the humerus. In some cases neuritis follows, with trophic changes.

**Treatment.**—Expose the nerve by an incision, incise the fibrous tissue back of the inner condyle, and press the nerve into the bed prepared for it and hold it in place by sutures of chromic catgut passing through the triceps tendon. Wharton advises suturing also "the margin of the fascial expansion of the triceps tendon superficial to the nerve."[2]

**Contusion of Nerves.**—The **symptoms** of contusion of nerves may be identical with those of section. Sensation or motion, or both, may be lost. The case may recover in a short time, or the nerve may degenerate as after section.

The **treatment** at first is rest, and later electricity, massage, friction, and douches.

**Punctured Wounds of Nerves.**—The **symptoms** of punctured wounds of nerves may be partly irritative (hyperesthesia, acute pain, and muscular spasm) and partly paralytic (anesthesia, muscular wasting, and paralysis).

The **treatment** after the puncture has healed is the same as that for contusion.

## Operations Upon Nerves

**Neurorrhaphy, or Nerve-suture.**—When a nerve is completely or partially divided by accident it should be sutured at the first possible moment. If there is no tension the best suture material is fine plain catgut; if there is any tension, lightly chromicized catgut (Sherren, in "Brit. Med. Jour.," Jan. 15, 1910). Sherren points out that if silk or linen is used it interferes with complete recovery and may cause symptoms months after the operation. In primary suture render the part bloodless and aseptic. Enlarge the incision if necessary. If the ends can readily be approximated, pass two or three sutures through the sheath and connective tissue outside, and tie them (Figs. 493, 494). If the ends

[1] "Amer. Jour. Med. Sciences," April, 1899.

[2] A report of 14 cases of dislocation of the ulnar nerve at the elbow, by H. R. Wharton, "Amer. Jour. Med. Sciences," Oct., 1895.

cannot be approximated, stretch each end and then suture. The sutures do not traverse the nerve, but go through the perineurium and adjacent connective tissue. After suturing, wrap the suture line in Cargile membrane to prevent adhesions to adjacent structures (Sherren, "Brit. Med. Jour.," Jan. 15, 1910). Suture the deep fascia. Remove the Esmarch band, arrest bleeding, suture the skin, dress antiseptically, and put the part in a relaxed position on a splint. After union of the wound continue the use of the splint to maintain muscular relaxation as long as the muscles are paralyzed (Ibid.), and use massage, friction, electricity, and the douche. When voluntary power returns remove the splint and insist on active exercise. The operation in some instances fails, but in many cases succeeds. In some few cases sensation returns in a few days, but in most cases does not return for many weeks or months. Sensation is restored before motor power. After successful suturing of a divided median nerve sensations of skin pain and of extreme heat and cold appeared in fifty-six days and were restored in two hundred and seven days. Sensations of light touch and slight degrees of heat and cold appeared in two hundred and sixty days and were not completely restored for one year (Kenneth A. J. Mackenzie, in "Annals of Surg.," July, 1909). *Secondary suture* is performed upon cases long after division of a nerve. If operation is not done for three years or more after division it is very improbable that complete regeneration will ever occur, and yet it is always worth trying, for muscular control has been regained after suturing in 1 case twenty-nine years subsequent to nerve division (Alfred S. Taylor, in "Jour. of Orthopedic Surg.," Nov., 1908). The part is rendered aseptic and bloodless; an incision is made; the bulbous proximal end is easily found and loosened from

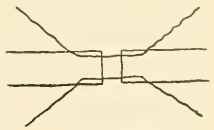


Fig. 493.—Nerve-suture.

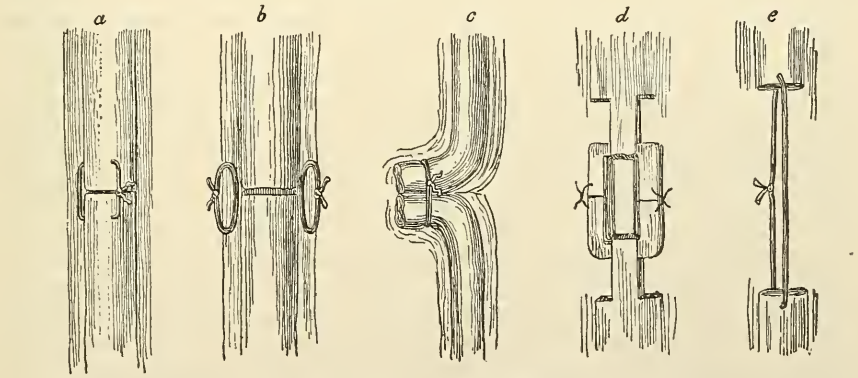



Fig. 494.—Nerve-suture: *a*, Direct; *b*, perineurotic; *c*, paraneurotic; *d*, *e*, neuroplasty (Senn).

its adhesions; the shrunken distal end is sought for and loosened (it may be necessary to expose the nerve below the wound and trace its trunk upward); the entire bulb of the proximal end is cut off; about $\frac{1}{4}$ inch of the distal end is removed. All scar-tissue between the ends is most carefully removed. If the gap between the ends is not wide, each end is gently stretched, and the ends are approximated and sutured together, and the suture line is covered by Cargile membrane. If stretching does not permit of approximation, adopt the expedient shown in Fig. 494, *d*, or in Fig. 495. This operation is *neuroplasty* by the *flap method.* Another method is to make a bridge of strands of catgut running from one divided end to the other. We speak of this plan as *suture à distance* (Fig. 494, *e*). The catgut bridge supports the growing reparative material.

Allis suggested shortening the limb by resecting a piece of bone. This has been done successfully by Keen, Rose, and others.

Letiévant attaches the peripheral portion of a divided nerve into a longitudinal slit in a sound nerve (end to side).

Guelliot suggested *tubulization*, that is, erecting barriers along the path of reparative material to save the ends of the nerve from cellular invasion from the perineural structures (which would block repair) and to guide the new growth. Vanlair uses a piece of artery which has just been removed or surrounds the ends with decalcified bone. Payr uses a tube of absorbable magnesium. Hashimoto and Toknoka use a vein or artery from a recently slaughtered calf, hardened in formalin. *Implantation* or *anastomosis* is advisable in some cases.

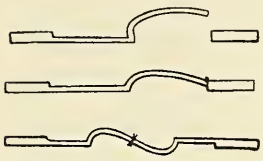


Fig. 495.—Suture of a nerve by splitting the ends (Beach).

Gelatin tubes have been used, silver-foil has been tried, and Cargile membrane has been employed. None of these plans is entirely satisfactory. Murphy covers the line of nerve-suture with fascia, muscle, or fat. In regions where he cannot obtain suitable covering tissue he wraps about the suture line a material composed of equal parts of paraffin and oil of sesame. This mixture can be flattened out very thin ("Surg., Gynec., and Obstet.," April, 1907).

The operation of anastomosis is employed after an exsection which leaves a very large gap, for facial palsy, for infantile palsy, for avulsion of the brachial plexus, and for brachial birth palsy.

**Nerve-grafting** is practised by some. A. W. Mayo Robson used the spinal cord of a rabbit to fill a gap between the ends of the divided median nerve of a man. The restoration of function was almost complete. Some surgeons have grafted in bits of nerve obtained from a recently amputated limb. It makes no difference whether the grafted nerve is motor, sensory, or mixed. The results of grafting are seldom good. Chas. A. Powers ("Transactions of the American Surgical Assoc.," 1904) collected 22 cases from literature, 20 from Peterson's paper, 1 case of Durante's, and 1 of his own. In this series there were 3 good results and 3 "fair" results. The bit of nerve grafted does not participate in repair—it is a mere bridge, and acts as does the suture à distance.

**Neurectasy, Neurotomy, and Neurectomy.**—*Neurectasy*, or nerve-stretching, may be applied to motor, sensory, or mixed nerves. A nerve can be stretched about one-twentieth of its length. Nuerectasy has been employed for neuralgia, neuritis, muscular spasm, hyperesthesia, anesthesia, painful ulcer, perforating ulcer, the pains of locomotor ataxia, and many other conditions. The operation, which was once the fashion, seems to benefit some cases, but it is not now thought so highly of as formerly. The incision for neurectasy is identical with the incision for neurectomy or neurotomy of the same nerve. *Neurotomy*, or section of a nerve, is performed only upon small and purely sensory nerves (in spasmodic wry-neck a motor nerve is cut). It is performed chiefly for peripheral neuralgia or for some other painful malady. It is almost useless in painful conditions, because sensation, as a rule, soon returns. Paget saw complete return of sensation in four weeks after division of the median nerve. Corning endeavors to prevent this regeneration by inserting oil between the ends. He uses oil of theobroma containing enough paraffin to make the melting-point 105° F. The oil is melted, is injected around the nerve, and cold is applied. The nerve is now sectioned with a canaliculated knife, the ends are separated widely, more oil is injected, and cold is again applied. The theory is that this oil, which is solid at the

temperature of the body, devitalizes the nerve at the point of section and acts as a barrier to the passage of regenerating fibers. This method has been applied especially in cervicobrachial neuralgia.[1] *Neurectomy*, or excision of a portion of a nerve-trunk, is applicable to sensory nerves and to painful affections.

**Sympathectomy.**—*Jonnesco's Operation.*—It has long been known that division of the sympathetic nerve in the neck may produce important changes in the eye and in the cerebral circulation. In 1893 Jaboulay divided the sympathetic on each side for the purpose of treating epilepsy. The removal of the ganglia of the sympathetic was proposed by Baracz; and the operation was first performed by Jonnesco, in 1896, for epilepsy. The operation is performed by some surgeons for epilepsy, for exophthalmic goiter, for glaucoma, and for trifacial neuralgia. In operating for glaucoma the superior cervical ganglion on each side is removed, as it is from this that the sympathetic fibers that pass to the eye are derived. If the operation is done at all, it should be a bilateral one.

This operation is used in epilepsy on the theory that there is an anemic condition of the brain in this disease which is corrected by producing a hyperemia, and that the hyperemia improves cerebral nutrition. The operation in epilepsy is largely theoretical, although Jonnesco claims 12 per cent. of cures in a large number of operations. In exophthalmic goiter there seems to be some distinct evidence that the operation may be beneficial, but Curtis shows that the mortality is high. Personally, I have not employed it in epilepsy, and at the present time I should not be inclined to do so. In exophthalmic goiter, if any operation is necessary, I perform partial thyroidectomy or ligation of the thyroid arteries; but in progressive glaucoma, which is always so absolutely hopeless, the operation is a justifiable procedure and occasionally seems to have a distinct influence in retarding the development of the disease.

The incision should be made along the posterior margin of the sternocleidomastoid muscle. I have become convinced, in performing two operations of this kind and through studies made upon the dead body, that the ganglion may be more easily reached from behind the sternocleidomastoid than from in front of it. The internal jugular vein and the carotid artery are lifted upward and forward; and the superior ganglion will usually adhere to the under portion of the carotid sheath and be lifted up with it. Theoretically, it is not necessary to open the carotid sheath in this operation, but, practically, this had better be done, so that one may, without any possibility of doubt, distinguish between the pneumogastric and the sympathetic nerve. The moment the nerve is cut the pupil on that side will contract.

**Stretching of the Sciatic Nerve.**—Some surgeons stretch the sciatic nerve by anesthetizing the patient and holding the leg and thigh in line, strong flexion being made upon the hip, the entire lower extremity being used as a lever. This method, which has caused death, inflicts needless damage, and stretching after an incision has been made is safer and better. The patient lies prone, the thigh and legs being extended. An incision 4 inches in length is made a little external to the middle of the thigh, and going at once through the deep fascia; the biceps muscle is found and is drawn outward; the nerve is discovered between the retracted biceps on the outside and the semitendinosus on the inside, resting upon the adductor magnus muscle. The nerve, which is caught up by the finger, is first pulled down from the spine and then up from the periphery, and finally the hook of a scale is inserted beneath the trunk and the nerve is stretched to the extent of 40 pounds.

[1] "Medical Record," Dec. 5, 1896.

Very rarely is even a single ligature needed. The wound is sutured and dressed. If the incision is made at a higher level, just below the gluteo-femoral crease, the sciatic nerve will be found just by the outer border of the biceps.

**Neurectomy of the Infra-orbital Nerve.**—This operation was first performed by Abernethy in 1793. The patient lies upon his back, the head being raised a little by pillows. The surgeon stands to the outside of and faces the patient. A curved incision $1\frac{1}{2}$ inches long is made below the lower border of the orbit. The nerve lies in a line dropped from the supra-orbital notch to between the two lower bicuspid teeth and is found upon the levator labii superioris muscle. A piece of silk is passed under the nerve by an aneurysm needle and firmly fastened. The upper border of the incision is drawn upward; the periosteum of the floor of the orbit is elevated and held by a retractor; the roof of the infra-orbital canal is broken through; the nerve is picked up far back by the blunt hook and is divided by scissors, and the entire nerve in front of the section is drawn out by making traction upon the silk. The bleeding in the orbit is checked by pressure. The wound is stitched without drainage.

**Neurectomy of the Supra-orbital Nerve.**—Before sterilizing the parts shave off the eyebrow. A curved incision 1 inch long discloses the nerve as it emerges from the supra-orbital notch or foramen at the junction of the inner and middle thirds of the eyebrow. The nerve is pulled forward and cut off above and below.

**Neurectomy of the Inferior Dental Nerve.**—Make a curved incision around the angle of the jaw. Lift the supramaxillary branch of the facial nerve downward (Kocher). Separate the masseter muscle by a periosteum elevator and slight touches of the knife. Chisel an opening in the center of the ascending ramus (Velpeau's rule). This opening exposes the beginning of the dental canal. If necessary, the opening may be enlarged by a rongeur. Pull the nerve out by a hook and remove a piece from it.

**Extracranial Operation for Neuralgia of the Fifth Nerve.**—The operation for removal of the Gasserian ganglion is difficult, bloody, and dangerous. Removal of portions of the pain-haunted nerve-trunks sometimes cures the condition and often ameliorates it for a considerable time. The injection of osmic acid into the peripheral nerves, or of alcohol into the nerves as they emerge from the cranium, may actually cure or secure prolonged relief. The serious operation of removing the ganglion may be performed if peripheral operations and injections fail, or in violent and intractable cases of long standing in which pain is felt in more than one branch. Removal of nerves by ordinary neurectomy often gives comfort for a few months, but rarely gives prolonged relief. If we seek striking benefit by an extracranial operation, it must be thoroughly done.

**Injection of Osmic Acid.**—This method was suggested by Bennett, of London, in 1897. Osmic acid had been used for many years in a sort of haphazard way, being thrown into tissues about the nerves by means of a hypodermatic syringe. Bennett suggested exposure of the nerve and the injection of 5 to 10 min. of a 1 per cent. solution. Acid when so used actually destroys nerve-fibers, and a considerable amount of fibrous tissue forms which intercepts regenerating fibers. It is probable that secondary degenerative changes occur in the nerve-trunks, but they do not ascend and reach the ganglion. Murphy warmly advocates the method. It certainly produces immediate relief by causing anesthesia, but such relief is very seldom, if ever, permanent. I have used it in several cases with satisfaction. In 1 case in which I exposed the ganglion I injected that structure, and the result seemed to be the same as if I had removed the ganglion. In neuralgia of

the fifth nerve the painful nerve or nerves should be exposed, and from 5 to 10 min. of a 2 per cent. solution of osmic acid injected into several different parts of the nerve and also between the nerve-sheath and the bony canal (Murphy). The osmic acid method does not seem to grow in favor.

**Injections of Alcohol Into the Second and Third Divisions.**—This method was devised by Schlösser in 1903. The injections are made into peripheral branches or divisions of the ganglion, and have even been made into the ganglion. Siccard and others have warmly praised the method. It acts similarly to osmic acid. It produces local necrosis of the nerve and fibrosis about it. The necrosis does not tend to ascend (May, "Brit. Med. Jour.," Aug. 31, 1912). It gives relief sometimes after one injection, sometimes after two or more. The permanence of the relief is uncertain. Usually it is complete for six months and then recurrent pain may require renewed injection. Recurrences are milder than the previous condition. Apparent cure may last for three or four years.

I have used the method many times and am satisfied as to its value in proper cases. It is superior to peripheral operations. It is used in very old subjects, feeble subjects the victims of grave organic disease, and in those who refuse radical operation. In strong young persons removal of the ganglion may still be preferred. In some cases it is used before a ganglion extirpation in order to remove pain and permit of such improvement in health and strength as to make the radical operation safer. Never attempt to make a deep injection about the ophthalmic branch. To do so may damage a great vessel, the optic nerve, the third, fourth, or sixth nerve, or the alcohol may enter the orbit and destroy the eye. If there is pain in the supra-orbital and supratrochlear branches, inject them where they are superficial.

*Injection of the Supra-orbital Branch.*—Use an ordinary hypodermatic needle and inject 10 to 20 min. of the fluid recommended below. Throw the fluid about the nerve at the foramen. After withdrawing the needle make pressure at the puncture. Such pressure is advised by Patrick to stop bleeding, prevent a black eye, keep the tissues exsanguine, and add to the effect ("Jour. Am. Med. Assoc.," Jan. 20, 1912). The lids always swell.

I used for a time 80 per cent. alcohol, each dram of which contained $\frac{1}{6}$ gr. of stovain, but this hardens the tissues so much that they become resistant to reinjection. I now use Patrick's formula, viz.: 2 gr. of muriate of cocain, $2\frac{1}{2}$ drams of alcohol, and distilled water sufficient to make $\frac{1}{2}$ oz. The usual dose is 2 c.c. A special needle is used. It is straight, is graduated in centimeters, and carries a stylet which is flush with the point. The stylet is pulled back slightly while the needle is being driven through the skin and fascia; it is then pushed all the way in. I follow the method of Lévy and Baudoin ("Presse Méd.," April 17, 1906), which is as follows:

*Superior Maxillary Division.*—Drop a perpendicular from the posterior border of the orbital process of the malar bone (the beginning of the zygoma) to the inferior edge of the zygoma; $\frac{5}{10}$ c.c. back of this point is where the needle should be introduced. It is carried in and very slightly upward. At a depth of 2 cm. the coronoid process may obstruct: at a greater depth, the external pterygoid plate. Either obstruction may be avoided by inclining the needle very slightly forward (never much or alcohol might enter the orbit). At a depth of 5 cm. the needle encounters the nerve as it emerges from the foramen rotundum. The injection usually causes decided but brief pain. If the injection reaches the nerve there will be immediate analgesia throughout its trajectory. This should last two or three days.

*Inferior Maxillary Division.*—The point for entrance of the needle is at the lower border of the zygoma, 2.5 cm. in front of the anterior root of the zygoma. The needle is carried inward, somewhat backward, and slightly upward. At a

depth of 4 cm. the nerve is reached. Be sure the nerve has been reached by finding analgesia throughout its trajectory after injection.

**Rose's Method of Neurectomy.**[1]—This operation is a modification of the Braun-Lossen method, and is employed when the second division of the fifth nerve is the seat of pain. The infra-orbital nerve is exposed by an incision, a ligature is tied around it, the roof of the infra-orbital canal is opened by a chisel, and the nerve is traced back as far as possible. The wound is then packed temporarily with gauze. The next step in this operation is to open a way into the sphenomaxillary fossa (Fig. 496). The knife is inserted slightly below the external angular process of the frontal bone, is carried back along the zygoma, down in front of the ear to just above the angle of the jaw, and then forward for 2 inches. This flap, which is composed of skin and subcutaneous fat only, is dissected forward, and Steno's duct and branches of the facial nerve are not damaged. The flap is wrapped in gauze and temporarily stitched to the side of the nose. The zygoma is exposed by a transverse incision. At the root of the zygoma two holes are drilled

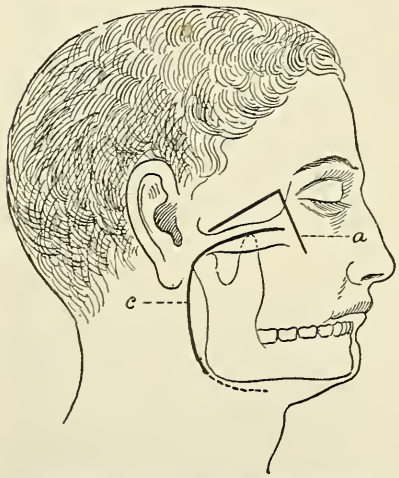



Fig. 496.—*a*, The Braun-Lossen incision; *c*, Rose's incision for reaching the sphenomaxillary fossa (Rose).

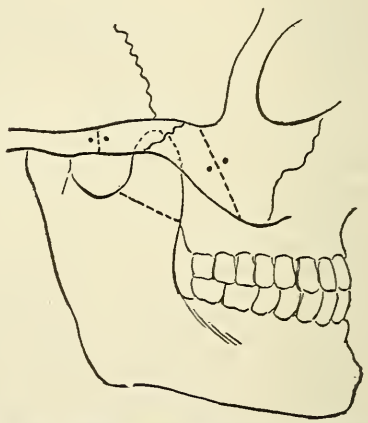


Fig. 497.—Lower jaw and zygoma. Drill-holes and saw-cuts are shown (Rose).

$\frac{1}{4}$ inch apart, and two more holes $\frac{1}{4}$ inch apart are drilled through the zygomatic process of the malar bone. The zygoma is then divided by a saw (Fig. 497). The posterior saw line runs between the two drill-holes at the root of the zygoma. The anterior cut passes between the two anterior drill-holes. The direction of the first cut is directly downward. The direction of the second cut is downward and forward from above. The arch is freed and detached downward and backward. The exposed tendon of the temporal muscle is retracted backward. The removal of a little fat exposes the pterygomaxillary fossa. The internal maxillary artery is exposed, two ligatures are applied, and the vessel is divided between them. The finger feels for the sphenomaxillary and pterygomaxillary fissures. The external pterygoid muscle is separated from the greater wing of the sphenoid and from the root of the external pterygoid process. On the edge of the greater wing of the sphenoid a long prominence is usually detectable. It overhangs the sphenomaxillary fossa and should be cut away by the use of a chisel. The superior maxillary nerve is lifted on a blunt hook, is grasped by forceps, and is twisted off as near the ganglion as possible (Fig. 498). The

[1] See article by Wm. Rose, "Practitioner," March, 1900.

distal end is drawn upon, and the nerve, having been previously loosened, is drawn back through the infra-orbital canal. The zygomatic arch is wired in place, the temporal fascia is sutured with buried sutures, and the skin-wound is closed. If the pain involved not only the second division, but also the third division, the operation previously described should be performed first, and the third division should be attacked a few weeks later. The third division is reached by removing the coronoid process. The inferior dental and lingual nerves are found, and are traced up to the foramen ovale, and are twisted off close to the ganglion, and the distal portions are removed.

**Removal of the Gasserian Ganglion.**—This formidable procedure was first suggested by J. Ewing Mears in 1884, and was first performed by Wm. Rose in 1890. The operation is often bloody and difficult, and is only undertaken in very severe cases of tic douloureux in which the first division is involved, or in cases upon which less grave procedures have failed. Jaboulay and Cavaillin ("Lyon. Med.," May 17, 1908) speak of it as a grave operation of difficult technic which should be left as a final resort. Many operators deny that there is a large mortality after gasserectomy and claim that it is only about 5 per cent. Some operators report a mortality of from 10 to 17 per cent. The greater the experience of the surgeon in this operation, the smaller will be the mortality. Knowledge of the region and parts, dexterity from frequent repetition, and special training count for much. The operation usually cures the pain if the patient recovers from the actual procedure. It is claimed that the pain may recur even after complete removal of the ganglion. I have never seen this occur and am disposed to think that recurring pain is apt to mean that there was a partial removal. Carson collected 100 cases; Murphy and Neff, 42 cases. The mortality in this group of 142 cases was 15 per cent. Most of the cases reported by Murphy and Neff were operated upon during or after 1899, and in this group the mortality was 10 per cent. ("Progressive Medicine," March, 1903). In Lexer's series of 201 cases, referred to below, the mortality was 17 per cent. In many cases a perfect cure is obtained. In some few the pain returns upon the side operated upon. Occasionally it arises on the side not operated upon. In some cases ulceration of the cornea follows operation. Such ulceration may be trivial, may result in opacity, or may destroy the eye. Paralysis of the abducens occurs in some cases. The hemorrhage may be so profuse as to require packing of the wound and suspension of the operation for a few days. The bleeding may come from the meningeal artery, from the sinus, or from the veins of Santorini. Lexer ("Arch. f. klin. Chir.," Bd. lxv, H. 4) gives a table of 201 cases. Of the survivors, 93.4 per cent. were apparently cured. In two-thirds of the cases the trouble was right sided. In 10 the operation was temporarily abandoned because of hemorrhage. The experience of surgeons in general is that after the removal of the ganglion there is apt to be some atrophy of the tongue and the eye usually becomes insensitive and watery. The masseter muscle will be paralyzed.

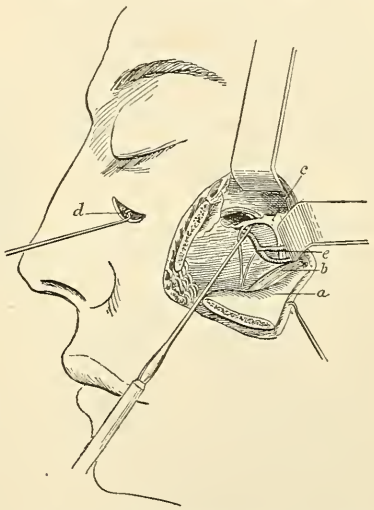



Fig. 498.—*a*, The zygomatic arch, turned down after sawing; *b*, tendon of the temporal muscle retracted; *c*, superior maxillary nerve and Meckel's ganglion; *d*, infra-orbital nerve emerging from canal; *e*, internal maxillary artery.

**The Hartley Operation for Removal of the Gasserian Ganglion.**—This operation was first performed by Hartley in 1891, five months before Krause performed it. An electric forehead-light is required. Long strips of gauze must be ready for packing in case of hemorrhage. The patient is placed recumbent, with head turned to the opposite side. The application of a provisional ligature or clamp to the external carotid artery is advocated by some, but this step will not control the venous bleeding, which is the most harassing hemorrhage encountered. Many operators form a large osteoplastic flap in front of the ear (Fig. 499) and break it out. I do not believe that an osteoplastic flap is necessary. The temporal fascia is so thick and tense that when the wound in it is carefully sutured protection is perfect and safety is secured. Hemorrhage is to be carefully arrested. It may be found that the meningeal artery has been ruptured. If this accident has happened and the vessel lies in a bony canal, plug with Horsley's wax. If the vessel is bleeding upon the dura, ligate by passing suture-ligaments around it. If it is torn off at the foramen spinosum, pack the foramen with iodoform gauze, and postpone the conclusion of the operation for forty-eight hours. It may be

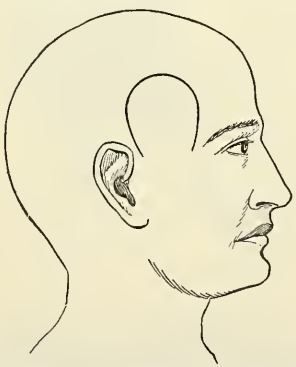


Fig. 499.—Hartley's osteoplastic flap in removal of Gasserian ganglion (Tiffany).

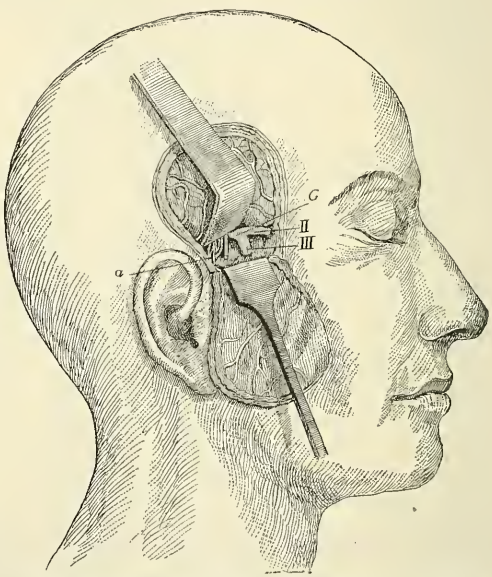



Fig. 500.—Removal of Gasserian ganglion: *a*, Middle meningeal artery; *II*, ophthalmic division; *III*, submaxillary division; *G*, ganglion (Krause).

necessary at any stage of this operation to pack the wound and postpone completion for two days. Some surgeons (Krause, Bergmann) ligate the meningeal artery as a routine procedure, but this operation may be difficult and require much time. If the unligated vessel is divided, the hemorrhage can be arrested by gauze packing or by plugging the foramen spinosum with a bit of sterile wood, but it is best to ligate the vessel. The head and body of the patient should now be elevated. This allows the brain to drop posteriorly and renders forcible retraction unnecessary, and, further, it lessens venous bleeding (Lexer). The next step is to lift up the dura and with it the brain (Fig. 500). Find the inferior maxillary nerve and clamp it with hemostatic forceps. Find the superior maxillary nerve and clamp it. Uncover the ganglion. Loosen the nerves from their beds by a dry dissector and divide each one at its foramen of exit. Twist the clamp forceps so as to reel up the nerves. This pulls out the ganglion intact with the motor root and the root of origin, as far back as the pons (Krause's method). Arrest bleeding; close the flap;

sew together by a couple of stitches the lids of the affected side in order to keep them temporarily closed, and cover the eye with a watch-crystal. The eye is to be frequently washed out. Thus irritants are excluded.

Cushing has modified the Hartley operation so as to permit of extradural manipulation below the arch made by the middle meningeal artery and thus lessen the danger of laceration of the artery ("Jour. Amer. Med. Assoc.," April 28, 1900). The anterior arm of the incision of the soft parts is so placed that it does not cut the nerve to the occipitofrontalis muscle. Thus drooping of the lid and oblation of brow wrinkles on that side are avoided. He trephines the wall of the temporal fossa very low down, opens into the skull below the arch of the meningeal vessels, and thus avoids the meningeal at the foramen spinosum of the sphenoid bone and the sulcus arteriosus of the parietal bone.

**Horsley's Intradural Method.**—An opening is made into the middle fossa of the skull, the dura is opened, and the ganglion is found and removed. This operation is easier than the extradural method, but is believed to be more dangerous.

**The Frazier-Spiller Operation of Intracranial Neurotomy of the Sensory Root of the Trigeminus.**—If experience shows that after division of the sensory root the nerve does not regenerate, and it seems probable that it does not, the operation must be regarded as a valuable addition to our resources. This operation is by many surgeons preferred to removal of the ganglion. In this operation the zygoma is temporarily resected. The temporal fossa is exposed, the bony wall is trephined, and the trephine opening is enlarged by the use of a rongeur. The dura is separated and the ganglion is reached. The dural envelope of the ganglion is opened, separated, and the sensory root exposed. The sensory root is then picked up on a blunt hook and divided. It is frequently possible, Frazier tells us, to separate the sensory root from the motor root. In this operation we avoid the venous hemorrhage from the foramen ovale and foramen rotundum which is apt to be encountered when removing the ganglion.

**Abbe's Operation of Intracranial Neurectomy of the Second and Third Divisions.**—This operation is preferred by Charles A. Ballance, who opposes exposure of the ganglion or division of the sensory root unless the first division of the nerve is the seat of pain. He advocates this in spite of knowing full well that the pain may return in a few years. He advocates it because of its safety, its simplicity, its freedom from serious hemorrhage, its avoidance of opening the intradural space and of all danger of corneal anesthesia, and also because if pain returns the operation can be repeated. The operation is performed as follows: Ligate the external carotid artery of the diseased side, make a vertical incision over the middle of the zygoma down to the bone. An opening into the skull is made by a mallet and gouge, and this opening is enlarged by a rongeur until it is $1\frac{1}{2}$ inches in diameter. The dura is lifted from the middle fossa and the nerves are exposed. Each nerve-trunk is clamped, is divided near its foramen of exit, and is separated from the ganglion by cutting or twisting by the forceps. A strip of sterile rubber tissue, $1\frac{1}{2}$ inches in length and $\frac{3}{4}$ inch in width, is laid over the round foramen and the oval foramen and is pressed into place by gauze. In a few moments the gauze is withdrawn and the ganglion is allowed to descend upon the rubber tissue. The wound is then closed. (See Robert Abbe, in "Annals of Surgery," Jan., 1903.) The rubber tissue is used to block the foramina of exit and prevent future emergence of regenerating nerves. Mayo Robson blocks the foramina by a thin plate of lead or silver, a knob of the plate entering the oval foramen to prevent displacement.

**Division of the Auditory Nerve for Tinnitus Aurium and for Aural Vertigo.**—This operation was proposed for tinnitus by Krause in

1902. Ballance did it on the right side with success in a most distressing case of painful tinnitus. When the cerebellar hemispheres were displaced by sponges the nerves of the posterior fossa were brought into view. He divided the eighth nerve, but made no attempt to preserve the nerve of Wrisberg. Five months after operation the patient was well except for deafness and deviation of the tongue to the left. (See Ballance, in "Lancet," 1908, vol. ii.) Frazier has divided the auditory nerve for aural vertigo, with partial relief.

**Operation for Facial Paralysis of Extracerebral Origin (Facio-accessory Anastomosis and Faciohypoglossal Anastomosis).**—(See "Remarks on the Operative Treatment of Facial Palsy of Peripheral Origin," by Chas. A. Ballance, Hamilton A. Ballance, and Purves Stewart, "Brit. Med. Jour.," May 2, 1903; and also the "Surgical Treatment of Facial Paralysis by Nerve Anastomosis," by Harvey Cushing, "Annals of Surgery," May, 1903.) In 1898 Furet suggested to Faure that he should anastomose the peripheral end of a divided facial nerve to that portion of the spinal accessory nerve which goes to the trapezius muscle. Faure did this, but the operation failed. Robert Kennedy, of Glasgow, did the first successful operation. He divided the facial for the relief of spasm and at once anastomosed to a partly divided spinal accessory. The procedure first employed by Ballance was, after noting by galvanism that muscular fiber still remained, to expose the facial nerve at its point of exit from the stylomastoid foramen, to cut the nerve-trunk across as high up as possible, to expose the spinal accessory, and to suture the distal end of the facial into the trunk of the spinal accessory. The spinal accessory was cut half through to make a bed for the end of the facial. The paper of the Ballances and Stewart above referred to recommends end-to-side anastomosis between the divided facial and the hypoglossal. The authors have operated five times for facial palsy. Cushing, Keen, Hackenbruch, Körté, Currie, Beck, Vidal, Girard, Lund, Alt, Frazier, and others have done similar operations. Marked improvement may follow operation even if palsy has lasted for a considerable time. Improvement followed operation in Currie's case, although the palsy was nearly a year old. The period when improvement should be expected is uncertain. Signs of improvement may not be evident for six months or longer. In Cushing's case they began in thirteen days; in Kennedy's case, in seven days. In most cases operation restores facial symmetry when at rest and in many cases during volitional movements. The patient will often become able to close the eye and raise the angle of the mouth. Curious associated movements may occur. In Currie's case when the patient lifted his shoulder there was contraction of the occipitofrontalis muscle ("South African Med. Record," 1907). Grant operated upon a case of traumatic facial paralysis over four months after the injury. He anastomosed the facial to the spinal accessory and the peripheral end of the accessory to the descendens hypoglossi. At the end of fifteen weeks there were feeble association movements of the face and shoulder, which later disappeared. At the end of a year the result was most gratifying ("Jour. Am. Med. Assoc.," Oct. 22, 1910).

Facio-accessory anastomosis does not restore emotional movements, but faciohypoglossal anastomosis may restore them. Küster reports a case which confirms this.

Operation is indicated when a complete facial palsy is of such duration that recovery is not to be hoped for by longer delay. The Ballances and Stewart believe that when palsy has lasted six months without sign of recovery, operation is indicated. A paralysis due to traumatism gives a much better prognosis after operation than does a paralysis due to a septic process (Chas. A. Ballance, Hamilton Ballance, and Purves Stewart, in "Brit. Med. Jour.," May 2, 1903). Murphy has collected 33 cases of anastomosis of the facial

nerve with the spinal accessory, hypoglossal, or glossopharyngeal, and Joseph Beck has added 5 cases of his own in which he performed faciohypoglossal anastomosis.

The hypoglossal is preferred to the accessory. Its trunk is larger and its cortical center is adjacent to the facial cortical area. After such an operation associated movements are not observed when the mouth is kept closed, and, if Gowers is correct, the fibers of the facial, which supply the muscles closing the mouth, may take origin from the hypoglossal nucleus (John B. Murphy, in "Surg., Gynec., and Obstet.," April, 1907). Murphy points out that an anastomosis may be end to end, implantation of the facial into a slit in the other nerve, implantation of the facial into a partial transverse division of the other nerve, or end to side (Ibid.).

**Operation for Brachial Birth Palsy.**—(See article by L. P. Clark, A. S. Taylor, and T. P. Prout, in "Am. Jour. Med. Sciences," Oct., 1905.) These authors report 8 cases of operation with some notable improvements and with 2 deaths. In these cases they found great thickening of the fascia and in some cases fibrous tissue almost completely obscured the remains of lacerated trunks or roots. They advise that the patient be placed recumbent, with a sand-pillow beneath the shoulders and with the head extended and bent toward the opposite shoulder. An incision is made at the posterior border of the sterno-cleidomastoid and the plexus is exposed and explored. If the lesion is above the clavicle, it is at once attacked; if below that bone, the incision is carried down and the bone is sawed in two. The scar tissue with the lacerated nerves is removed and the nerves or nerve-roots are sutured. The wound is closed, the clavicle being wired if it was divided. After dressings are applied the head is bent toward the shoulder of the damaged side and fixed with plaster of Paris.

I operated on a case of Dr. Charles S. Potts's in the Philadelphia Hospital. The roots were not torn, but were found embedded in a thin layer of scar which it was possible to remove. The result was good. Nerve anastomosis may be necessary if exsection of scar leaves an unbridgable gap or if nerve-roots were divided within the foramina.

**Operation for Avulsion of the Brachial Plexus.**—(See page 750.)

## XXIV. DISEASES AND INJURIES OF THE HEAD

### Diseases of the Head

In approaching a case of brain disorder, first endeavor to locate the seat of the trouble; next, ascertain the nature of the lesion; and, finally, determine the best plan of treatment, operative or otherwise. In all operations upon the brain the surgeon must be able to determine accurately the situations of certain fissures and convolutions, the finding of the situations of these convolutions and fissures comprising the science of *craniocerebral topography*.

The *regional terms* used in craniocerebral topography are derived from Broca (Fig. 501). The middle meningeal artery is found at the *pterion*, $1\frac{1}{4}$ inches posterior to the external angular process, on a level with the roof of the orbit (Fig. 502). The fissures and convolutions of the brain are shown in Figs. 503–505. The *fissure of Bichat* is marked by a line on each side drawn from the *inion* to the external auditory process. A line from the *glabella* to the inion overlies the median fissure and the superior longitudinal sinus. The *fissure of Rolando* is very important, as marking the posterior limit of the motor region of the brain. It begins near the median line, $\frac{1}{2}$ inch posterior to the middle of the distance between the inion and glabella (Thane). This fissure runs downward and forward at an angle of 67.5° for a distance of $3\frac{3}{8}$ inches.

Chiene finds the fissure of Rolando by the following method: He takes a square piece of paper and folds it into a triangle (Fig. 506, 1); the angle B–A–C of this triangle is 45°; the edge D–A is folded back on the dotted line A–E; the angle

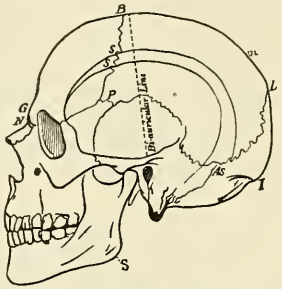


Fig. 501.—Skull, showing the points named by Broca: *As*, Asterion (junction of the occipital, parietal, and temporal bones); basion, middle of anterior wall of foramen magnum; *B*, bregma (junction of the sagittal and coronal sutures); *G*, ophryon (on a level with the superior border of the eyebrows, and corresponding nearly to the glabella, the smooth swelling between the eyebrows); *g*, gonion (angle of the lower jaw); *I*, inion (external occipital protuberance); *L*, lambda (junction of sagittal and lambdoidal sutures); *N*, nasion (junction of the nasal and frontal); *Ob*, obelion (the sagittal between the parietal foramina); *P*, pterion (point of junction of great wing of sphenoid and the frontal, parietal, and squamous bones—this may be H-shaped or K-shaped or "retourné," in which the frontal and temporal just touch); *S*, stephanion (or, better, the superior stephanion, intersection of ridge for temporal fascia and coronal suture); *S'*, inferior stephanion (intersection of ridge for temporal muscle and coronal suture).

D–A–E equals half of 45°, or 22.5°, and the angle C–A–E equals the same (Fig. 506, 2); the paper is unfolded in the line C–A; in the figure thus formed B–A–C = 45° and E–A–C = 22.5°; E–A–B = 67.5°, which is the angle desired. Place the point A in the midline of the head, over the point of origin of the Rolandic fissure; the side A–B is laid along the middle line of the head, and the line A–E corresponds to the fissure of Rolando.[1] Horsley determines the situation of the Rolandic fissure by the use of his metal cyrtometer (Fig. 507). He places the point marked zero over the inioglabellar line and midway between the inion

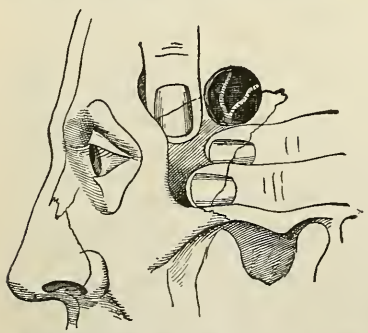


Fig. 502.—The meningeal artery exposed by trephining (after Esmarch).

Fig. 503.—View of the brain from above (Ecker).

and the glabella. To find the *fissure of Sylvius* (Fig. 504, *S*, *S'*, *S''*), draw a line from the external angular process to the occipital protuberance. The fissure of Sylvius begins on this line $1\frac{1}{8}$ inches behind the external angular process;

[1] "American Text-Book of Surgery."

the main branch of the fissure runs toward the parietal eminence; the ascending branch of the fissure corresponds to the squamosphenoidal suture, and continues upward in the same line ½ inch above the suture. The *precentral sulcus* (Fig. 504, F) limits anteriorly the ascending frontal convolution; it runs parallel with and just behind the coronal suture, and a finger's breadth in front of the fissure of Rolando. The *intraparietal fissure* (Figs. 503, 504, *ip*) limits the ascending parietal convolution posteriorly. It begins opposite the junction of the lower and middle thirds of the fissure of Rolando, passes upward in a line parallel with the longitudinal fissure and midway between the Rolandic fissure and the parietal eminence, passes by the parieto-occipital fissure, and downward and backward into the occipital lobe. The motor areas, which on the outer surface are adjacent to the fissure of Rolando, are shown in Figs. 503

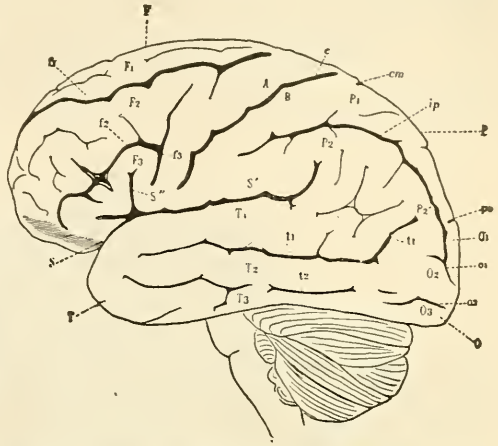


Fig. 504.—Outer surface of the left hemisphere of the brain (Ecker).

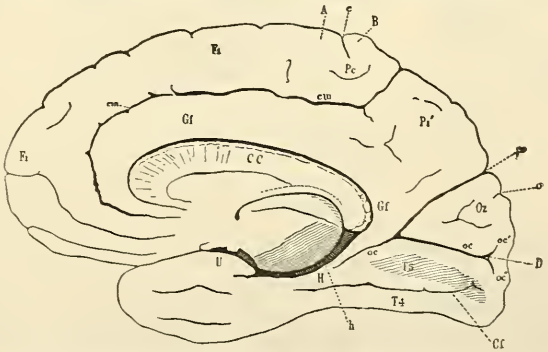


Fig. 505.—Inner surface of the right hemisphere of the brain (Ecker).

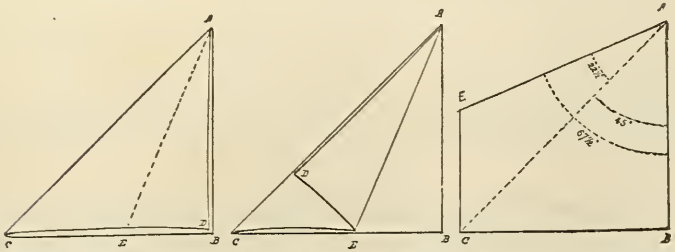


Fig. 506.—Chiene's method of fixing position of Rolandic fissure ("American Text-Book of Surgery").

and 504.[1] The superior longitudinal sinus is overlaid by a line from the inion to the glabella. The lateral sinus is indicated by a line running from the occipital

[1] Recent studies indicate that the motor region is entirely in front of the Rolandic fissure.

protuberance horizontally outward to a point 1 inch posteriorly to the external auditory meatus, and from this point by a second line dropped to the mastoid process. The *suprameatal triangle* of Macewen is bounded by the posterior root of the zygoma, the posterior bony wall of the auditory meatus, and a line join-

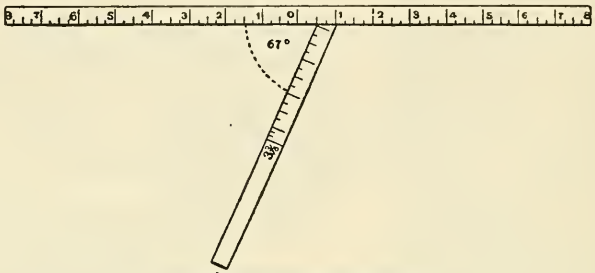



Fig. 507.—Horsley's cyrtometer.

ing the two. The mastoid antrum is opened through *Macewen's triangle* to avoid injury to the lateral sinus. *Barker's point*, the proper spot to apply the trephine for abscess of the temporosphenoidal lobe, is 1¼ inches above and 1¼ inches behind the middle of the external auditory meatus. Fig. 508 shows clearly the main points of craniocerebral topography, obtained by methods approved by many scientists.

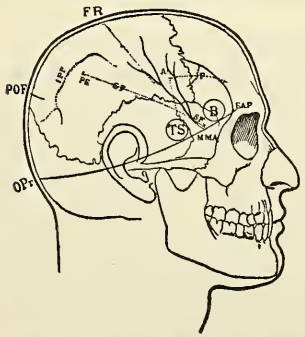



Fig. 508.—Head, skull, and cerebral fissures: B corresponds to Broca's convolution; EAP, external angular process; FR, fissure of Rolando; IF, inferior frontal sulcus; IPF, intraparietal sulcus; MMA, middle meningeal artery; OPr, occipital protuberance; PE, parietal eminence; POF, parieto-occipital fissure; SF, Sylvian fissure; A, its ascending limb; TS, tip of temporosphenoidal lobe. The pterion (to the left of B) is the region where three sutures meet, viz., those bounding the great wing of the sphenoid where it joins the frontal, parietal, and temporal bones (adapted from Marshall by Hare).

*Krönlein's method* for localizing certain areas is the most generally serviceable (Figs. 509, 510). A line, known as the base line, Z–M, is carried horizontally backward from the lower border of the orbit through the upper border of the external auditory meatus. Another horizontal line, K–K′, is drawn parallel with this, on a level with the supra-orbital ridge. A line Z–K is erected from the middle of the zygoma to the supra-orbital line. A vertical line is drawn from the articulation of the lower jaw, A, and is prolonged to R. A vertical line is drawn from the posterior border of the mastoid base (M–K′) and is taken to P, the middle line of the skull. A line is drawn from K to P, and between the points R and P′ it overlies the fissure of Rolando. The angle P–K–K′ is bisected by the line K–S, which corresponds to the fissure of Sylvius from its point of bifurcation to its posterior termination; K marks the bifurcation of the fissure of Sylvius. To reach the anterior branch of the middle meningeal artery trephine at K; to reach the posterior branch, trephine at K′.

**Head Injuries During Labor.—Caput Succedaneum.**—This condition is edema of the scalp due to prolonged pressure. The edema is circular and circumscribed and occupies the part not subjected to continued pressure during the uterine contractions of labor. The ring of tissues which are impressed around project like a cup into the birth canal. The veins become congested and edema results. The parts subjected to pressure may appear normal or may exhibit ecchymoses or even excoriations. The pressure is

usually made by the os, and its situation varies with the presentation, but because the most frequent presentation is left occipito-anterior, the common position of the caput is over the superior and posterior portion of the right parietal bone. In a face presentation great disfigurement may occur. It is seldom that a double caput is encountered. It always means that the presentation has shifted. The worst cases of caput follow prolonged labor. The edematous swelling contains bloody serum, pits on pressure, does not fluctuate, is not limited to the outline of one bone, and the skin above it is usually

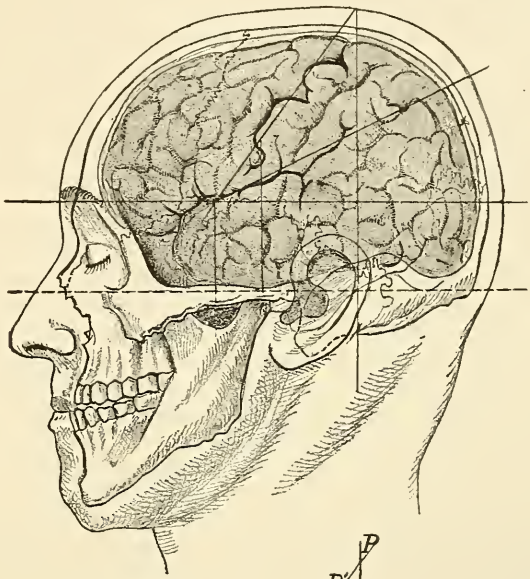


Fig. 509.

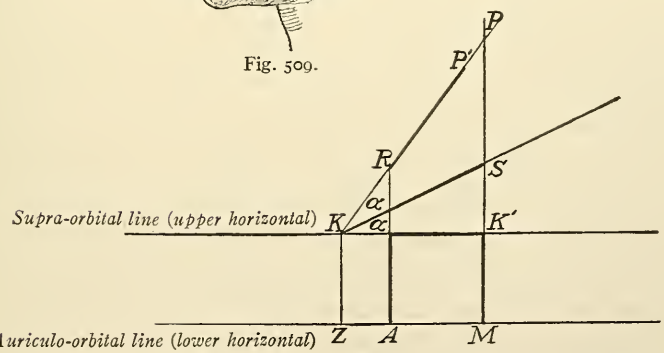



Fig. 510.

Figs. 509, 510.—Krönlein's method of locating the fissure of Rolando ($R$–$P'$) and Sylvius ($K$–$S$); Krönlein's point of trephining for hemorrhage from the middle meningeal ($K$–$K'$); and von Bergmann's region for trephining for abscess of the temporosphenoidal lobes ($A$–$a$–$K'$–$M$) ("American Text-Book of Surgery").

discolored by ecchymoses. No treatment is necessary, as the condition will disappear in from a few hours to three days.

**Cephalhematomata.**—By this term we mean extravasations of blood beneath the untorn pericranium. It is supposed by many to be always due to pressure and venous congestion, as is a caput succedaneum. As Cushing points out, the condition cannot result from the venous stasis of prolonged pressure alone. If it did it would always exhibit above it a caput succedaneum, "and this is far from our actual experience" (Harvey Cushing, in "Keen's

Surgery," vol. iii). It is certainly due in some cases to bending or breaking of a cranial bone. The condition is said to occur in 1 labor out of 200. In most cases there is but one cephalhematoma, but there may be two, three, or even four. The commonest situation is over the right parietal bone (the common seat of caput succedaneum), and caput succedaneum may be associated with a cephalhematoma. The blood begins to flow beneath the pericranium during labor and the swelling increases during the first few days after birth; in fact, it is frequently not noticed for a day or two. The swelling is tense and smooth, with a convex outline. It may cover but a small portion of a bone or an entire bone, but never extends beyond the bounding sutures. This limitation is due to the fact that the pericranium is adherent to the sutures. In the course of a couple of weeks the tumor may become surrounded by a hard ring due to the formation of new bone, and a shell of bone may eventually surround and cover over the clot, an area of permanent bony thickening remaining. In other cases no bone forms, but the clot gradually disappears. Extradural and even subdural hemorrhage may be associated with a subpericranial cephalhematoma.

Cephalhematomata unassociated with cerebral symptoms usually disappear without operation. If there is no sign of subsidence after two weeks, follow Cushing's rule, evacuate by a puncture-like incision and apply pressure. If suppuration occurs, incision is necessary. Suppuration may occur if the scalp was excoriated. In cephalhematoma with cerebral symptoms operation is indicated (incision of the scalp and removal of a piece of bone).

**Diseases of the Scalp.**—The scalp is composed of skin, subcutaneous fat, the occipitofrontalis muscle and aponeurosis, the subaponeurotic cellular tissue, and the pericranium. The scalp is liable to inflammation from various causes, and also to certain diseases—namely, tumors, cysts, warts, moles (local cutaneous hypertrophies), cirsoid aneurysm (see page 434), nevi, and lupus. *Abscesses of the scalp* are common. If an abscess forms beneath the pericranium, the pus diffuses over the area of one bone, being limited by the attachment of the pericranium in the sutures. In cranial osteomyelitis pus may gather between the dura and bone and between the bone and pericranium. The condition is known as *Pott's puffy tumor*. If an abscess forms in the tissue between the occipitofrontalis and the pericranium, it is widely diffused. Treves calls this subaponeurotic connective tissue "the *dangerous area*." Abscess of the subcutaneous tissue is apt to be limited because of the great amount of fibrous tissue. Abscess beneath the pericranium does not spread beyond the suture lines. It is limited by the sutural membrane which runs from pericranium to dura. Abscess is treated by instant incision at the most dependent part and drainage. In abscess beneath the occipitofrontalis aponeurosis it is necessary to open and drain above the eyebrows, above the superior curved line of the occipital bone, and at each side above the line of origin of the temporal fascia.

**Diseases and Malformations of the Bones of the Skull.**—The bones of the skull are liable to caries, necrosis, osteitis, periostitis, osteomyelitis, atrophy, hypertrophy, tumors, etc. (See Diseases of Bones.)

**Cranial Pneumatocele.**—This rare condition is a result of perforation of a bone which permits air to collect beneath the periosteum. It may occur in the mastoid or occipital region or over the frontal region. These protrusions vary greatly in size; and as their shape depends upon the periosteal attachment to sutures in the neighborhood, they vary in shape. The overlying tissues are natural in appearance. The protrusion is tense, but may lessen or disappear on pressure. McArthur ("Jour. Am. Med. Assoc.," May 6, 1905) points out that if diminished by pressure, the patient may hear a sound like rushing air or water in the ear if the protrusion is occipital or

mastoid; and in the nose, if it is frontal. An elevated ridge of bone surrounds a pneumatocele. The protrusion is tympanitic on percussion. The condition is due to perforation of the bony wall of an air sinus by disease, injury, or rupture. McArthur points out that in half of the reported cases the rupture was not preceded by any history of inflammation or injury. The condition is not dangerous.

**Treatment.**—Incision, finding the opening in the bone, enlarging it, removing osteophytes; bringing the walls of the cavity together and applying pressure.

**Microcephalus.**—By "microcephalus" is meant unnatural smallness of the head due to imperfect development. Marked microcephalus is not a common condition, but it is an occasional cause or associate of idiocy. A child may be born with a skull completely ossified even at the fontanels, or the ossification may become complete soon after birth, but in many cases of microcephalus ossification takes place late or not at all. In microcephalus the face is usually fairly well developed; the jaws are prominent; the forehead is flat; the cranium and brain are small; the convolutions of the brain are simpler than is natural; there is apt to be marked asymmetry of the two sides of the brain; internal hydrocephalus may exist; areas of sclerosis and atrophy are common; porencephaly is not unusual. Some patients have perfect motor power; others are slow and incoördinate. Epilepsy, chorea, and athetosis frequently complicate the case. Idiots of this type often present deformities such as cleft palate, strabismus, distorted ears, hypertrophied tongue, deformed genitals or extremities, ill-shaped and irregularly developed teeth. They exhibit irregular muscular movements, are frequently paralyzed in childhood (infantile paraplegia or hemiplegia), and suffer from subsequent contractures. They are active, destructive, excitable, and are liable to be violent and almost demoniacal. As Clouston says, they look impish and unearthly.

**Treatment.**—Skilled training in a school for the feeble minded or in an institution for idiots is necessary in treating microcephalic idiocy. Idiots have but little power of attention, and sensory impressions give rise to but few concepts, and these are feeble and fleeting. In order to educate the idiot it is highly desirable that speech be acquired, and "the more strongly the attention can be aroused, the more perfect does speech become" (Kirchhoff). The principle of the education of idiots is to stimulate, coördinate, and guide sight, hearing, feeling, taste, and smell.

Lannelongue, of Paris, suggested an operation for idiocy with premature ossification (see Linear Craniotomy, page 831). In this procedure the author has no confidence. Idiocy is a general disorder and not a local brain disease. Soft parts mold bone, and bone does not control soft parts. There is no evidence that the brain is being compressed; in fact, the simplicity of the convolutions suggests the contrary. In many typical cases of microcephalic idiocy there is no synostosis even years after birth. The operation has been much abused. It is sometimes fatal, and, although a fatality may gratify the family, a surgeon is not a legal executioner. The remarkable improvement which has been reported in some cases is wrongly supposed to be due to the operation. As a matter of fact, the new surroundings, the strange faces, the firm discipline, the effect of the anesthetic, and the shock of the operation attract the feeble attention and rouse the sluggish senses. Many cases are brought for operation because they are for the time being unusually intractable and excitable, and the return to the usual level of conduct after operation is regarded as a permanent gain, when it is often but a temporary alleviation. We believe that scientific training is the proper treatment, and that the efficiency of training is not increased by the previous performance of craniotomy, and we follow the precept of Agnew, that a

surgeon might as well cut a piece out of a turtle's back to make a turtle grow as to cut a piece out of the skull to make the brain grow. It would be as wise to take a piece out of the dome of a cathedral to increase the stature of the dean and chapter.

**Diseases and Malformations Involving the Brain.—Cephaloceles.**—A cephalocele is a congenital protrusion of intracerebral contents through a defect in the skull. These protrusions are covered with skin. The defect through which the protrusion occurs is always in the median line, although in some cases (as at inner angle of the orbit) the visible protrusion may be at the side. Nearly all such protrusions are either frontal or occipital, although now and then one presents in the pharynx, having emerged from the skull between the body of the sphenoid and the ethmoid.

*Frontal cephaloceles* are divided into:

1. Nasofrontal—those which are in the region of the glabella.
2. Naso-orbital—those at the inner angle of the orbit.
3. Naso-ethmoidal—those below the nasal bone.

Each one of the above forms passes through the horizontal plate of the ethmoid.

*Occipital cephaloceles* are divided into:

1. Superior—those above the external occipital protuberance. In these the bony gap may join the posterior fontanel.
2. Inferior—those below the external occipital protuberance. In these the bony gap may join the foramen magnum.

The above regional classification is that advocated in von Bergmann's "System of Practical Surgery" (translated and edited by Wm. T. Bull and Walton Martin).

The commonest form is hydrencephalocele, and all other forms result from retrograde changes in this.

**Hydrencephalocele.**—This is by far the commonest and is also the most dangerous form encountered. The protrusion consists of arachnoid, a layer of brain tissue, and a cavity containing ventricular cerebrospinal fluid and connected with the lateral ventricle. It is, in reality, a protrusion of the lateral ventricle. It is covered with skin—natural skin—unless the protrusion is very large, in which case the skin is more or less atrophied. Beneath the skin is fascia, and beneath this, arachnoid. The pericranium and dura do not cover it, but each has a gap in it and these two tissues join each other around the bone margins.

**Encephalocele** results from retrograde changes in a hydrencephalocele. The protrusion of the ventricle has become reduced and the hernia consists of a portion of brain covered by arachnoid. Encephalocele is only seen in the nasofrontal region. If there is any fluid in this protrusion it is not in its interior, but on its surface, and results from a cyst of the arachnoid.

**Meningocele.**—We formerly understood by a meningocele a protrusion of the membranes alone; we now regard it as a condition resulting from retrograde changes in a hydrencephalocele. The brain tissue of the latter disappears; beneath the arachnoid is a layer of cells identical with those which line the ventricles; the connection with the ventricle is entirely or almost completely cut off; a cyst forms in the subarachnoid tissue, and thickened pia surrounds the cyst. (See "System of Practical Surgery," by E. von Bergmann, vol. i, translated and edited by Wm. T. Bull and Walton Martin.) The above condition is called by von Bergmann encephalocysto-meningocele.

*Diagnosis.*—The congenital origin and situation make certain that the condition is cephalocele. The bony gap can usually be felt; whether it can or cannot, an *x*-ray picture should be taken. Such a picture may indicate that the mass contains brain matter. The protrusions vary greatly in size and shape.

Some are rounded, some are flattened, some are stalked. The skin covering them may be natural, atrophied, filled with vessels, scarred, or ulcerated. Sometimes the cephalocele is very tense; sometimes it is loose. In naturally hairy regions the skin over the summit of the protrusion is bald, but that around the base is hairy. If there is connection between the interior of the protrusion and the ventricle, the mass can be diminished in size by compression. If it shrinks rapidly from compression, the opening into the ventricle is large. In such cases compression of the mass quickly causes signs of cerebral pressure. Lumbar puncture may cause the protrusion to diminish in size; crying may cause it to increase in size. Large cephaloceles fluctuate and perhaps pulsate. Meningocele feels and looks like a cyst (is translucent and fluctuates); it does not usually pulsate, it has a small base, it becomes tense on forcible expiration, and some cases can be very slowly diminished by compression.

Encephalocele is small, opaque, does not fluctuate, has a broad base, does pulsate, becomes tense on forced expiration, and attempts at reduction fail and cause pressure symptoms.

Hydrencephalocele is larger than a meningocele, is translucent, fluctuates, rarely pulsates, is pedunculated, is rendered a little tense on forced expiration, and can be lessened in size by compression, but cannot be reduced.

*Treatment.*—In von Bergmann's "System of Practical Surgery" we find the wise caution to attempt no operation for an occipital protrusion beneath the protuberance when the cleft enters the foramen magnum and is associated with cleft of the cervical vertebræ—for a condition in which the soft parts are defective and the brain is exposed (*cranioschisis*)—on a case complicated by hydrocephalus or on a case complicated by some other condition which is of necessity fatal. We no longer refuse to operate because the mass contains some brain matter or because it communicates with the ventricle, although if it does so, the prognosis is much worse. For a large hydrencephalocele nothing can be done and early death is inevitable. In rare instances an encephalocele is converted into a meningocele, and the bony aperture closes, thus bringing about a cure. Among the expedients for treating meningocele are electrolysis, injection of Morton's fluid (10 gr. of iodin, 30 gr. of iodid of potassium, 1 oz. of glycerin), pressure, and excision. In cases of cephalocele, when portions of the nerve-centers are not contained in the sac, A. W. Mayo Robson advises the performance of a plastic operation. He ligates the neck of the sac, excises the sac, sutures the skin-flaps separately, and leaves the stump outside the line of superficial sutures. It is usually possible to tell by palpation if nerve-centers are in the sac, but if in doubt, make an exploratory incision, and sweep the finger around inside of the sac.[1] Meningoceles should be operated upon by Robson's plan.

**Spurious Meningocele.**—It occasionally happens, after a fracture of a child's skull, that cerebrospinal fluid gathers beneath the pericranium and bulges the pericranium and scalp. This condition is called spurious meningocele. When a spurious meningocele forms, the bone must have been broken and the dura and arachnoid ruptured. This protrusion fluctuates, pulsates, and is influenced by respiration. In some cases there is communication with the ventricles of the brain. The parietal and frontal regions are the most usual seats of the trouble. The opening in the skull may close; it may remain stationary; it may actually enlarge by bone-absorption. In some cases the spurious meningocele undergoes spontaneous cure; in some cases rupture occurs; in other cases death takes place as a result of the cerebral injury. (See Joseph Sailer on "Spurious Meningocele," "University Med. Magazine," Sept., 1900.)

*Treatment.*—Close the opening by a plastic operation.

[1] "Amer. Jour. Med. Sciences," Sept., 1895.

**Hydrocephalus.**—In *external* hydrocephalus the fluid is on the surface of the brain; in *internal* hydrocephalus the fluid is in the ventricles. Hydrocephalus may be *acute* or *chronic*, *congenital* or *acquired*.

**Acute hydrocephalus** is usually internal, but may be external. It results from meningitis—usually tuberculous meningitis of the base. The symptoms are headache, elevated temperature, delirium, stupor, convulsions, paralysis, and choked disk.

*Treatment* of acute hydrocephalus by medical means is of no avail. Tapping of the ventricles may be tried. Drainage of the cisterna magna has been suggested.

**Chronic internal hydrocephalus** is usually congenital, but may arise after birth in children under seven. In congenital hydrocephalus the condition may be due to circulatory disturbances in the brain of the embryo resulting from uterine disease or injury during pregnancy. Syphilis and alcoholism in parents seem sometimes to be responsible. Chronic acquired hydrocephalus results from inflammation, especially tuberculous inflammation. A tumor pressing on the veins of Galen may cause it. In chronic acquired internal hydrocephalus there is overproduction or underabsorption of cerebrospinal fluid and perhaps both conditions may exist. The usually causative condition is an inflammation of the interior of the ventricles, particularly of the choroid plexuses, and as a consequence venous return is obstructed and oversecretion occurs. In very rare cases one or both foramina of Monro may be closed, and if only one is closed, unilateral hydrocephalus may arise (Alfred S. Taylor, in "Am. Jour. Med. Sciences," August, 1904). The aqueduct of Sylvius, the foramen of Magendie, and the central canal of the cord may be, but seldom are, occluded. Guthrie ("Practitioner," July, 1910) studied 182 cases of meningitis at autopsy. In about 40 per cent. of the tuberculous cases and in 56 per cent. of the non-tuberculous cases hydrocephalus existed. A tumor may cause hydrocephalus by directly obstructing the flow of fluid from the ventricles, but a tumor far away from such a position may cause it by so increasing the intracerebral tension that the brain stem is forced down into the foramen magnum. Such a position of the pons and cerebellum cuts off the flow of cerebrospinal fluid between the subarachnoid spaces of the brain and cord. In hydrocephalus the cranium enlarges enormously and the bones of the skull are widely separated. The brain is distended and thinned and the sulci are obliterated. The broad forehead overhangs the eyes; the fontanels are elevated. The fontanels and sutures are open. The child is mentally weak or is an idiot, and very often does not learn to walk or to talk. Convulsions, palsies, and contractures are common, and blindness is frequent. Such children usually die young.

The *treatment* of chronic hydrocephalus is rarely of much avail. Pressure by strapping with adhesive plaster has been tried. Tappings through a fontanel may be performed by means of a trocar (only 2 or 3 oz. of fluid being withdrawn at a time). If much fluid is allowed to flow out, the head must be strapped with adhesive plaster afterward. If the skull ossifies, the lateral ventricles may be tapped after trephining. It has been proposed to drain by tapping the theca of the spinal cord (Quincke). This last operation is called *lumbar puncture* (see pages 861, 862). It will, of course, fail if the foramina in the floor of the fourth ventricle or the aqueduct of Sylvius are blocked. Even if they are open, it is of little service. The operation which promises most was devised by Sutherland and Cheyne, and is known as *intracranial drainage* ("Brit. Med. Jour.," Oct. 15, 1898). Their theory is that in hydrocephalus fluid distends the ventricles because the channels of communication between the ventricles and the subarachnoid spaces are closed. The subarachnoid spaces communicate directly with veins, hence fluid cannot collect under pres-

sure in these spaces. Intracerebral drainage establishes a communication between the subarachnoid space and one ventricle. It is not necessary to operate on both sides in bilateral hydrocephalus, because the lateral ventricles communicate. A small opening is made in the skull. The dura is incised. A number of strands of catgut, which are tied together, are pushed through the brain so that one end of the catgut mass lies in a ventricle and the other end beneath the dura. The dura and scalp are then sutured. Brewer makes an osteoplastic occipital flap, also a dural flap, lifts the cerebral lobe, and pushes a drain of rubber tissue into a lateral ventricle.

The elder Senn passed a rubber tube into the ventricle and put the outer end of the tube beneath the skin of the scalp.

Alfred S. Taylor ("Am. Jour. Med. Sciences," August, 1904) makes an osteoplastic flap with its base over the right mastoid, cuts a dural flap, passes a slender aspirating needle through the second temporosphenoidal convolution into the lateral ventricle, draws off a *little* fluid, and measures the thickness of the brain. He then takes 6 strands of No. 2 forty-day catgut, each strand $\frac{1}{2}$ inch longer than the thickness of the brain. The strands are tied together with a spiral of catgut, $1\frac{1}{4}$ inches of the loop being left free. Three layers of Cargile membrane are wrapped about the shaft, but the tip remains free. It is carried into the ventricle along the needle track by thumb forceps, and the loops are slipped here and there, but chiefly downward, under the dura. Cargile membrane is placed between the loops and dura and the dura and skin are sutured. Taylor operated on 6 cases and 2 recovered, with relief of all signs of pressure.

The cisterna magna may be drained. Cotterill actually opened the foramen of Majendie, and the patient distinctly improved ("Lancet," Nov. 12, 1910).

Cushing, after determining by lumbar puncture that the ventricles can be emptied, obtains retroperitoneal drainage by a combined laparotomy and laminectomy. Marmion establishes drainage from the ventricles into the highly lymphatic tissues about the parotid gland ("Zentralb. für Chirurgie," August 12, 1911), and Payr, with the internal jugular vein by means of a transplanted artery or vein ("Archiv. für Klin. Chirurgie," August 26, 1911).

Puncture of the corpus callosum has been recommended by Anton and others

## Injuries of the Head

**Caput Succedaneum.**—(See page 772.)

**Cephalhematoma.**—(See page 773.)

**Scalp-wounds** bleed profusely because the scalp is very vascular, because many of the blood-vessels are in fibrous tissue and cannot contract and retract, and because even blunt force splits the scalp almost like an incision. Scalp-wounds are treated as are other wounds. Even a large piece of scalp with only a narrow pedicle may not slough; hence try to save any piece that has an attachment. Always shave a wide area and disinfect the shaved area and the wound. Arrest hemorrhage, and exercise great care in cleansing the wound and the parts about it. Stitch the wound with silkworm-gut. Very few sutures are needed if the wound is longitudinal, but many are required if it is transverse. Deep vessels are ligated. The permanent arrest of hemorrhage from the skin and subcutaneous tissue is rarely affected by ligatures, but rather by sutures judiciously placed. If drainage is required, use a few strands of silkworm-gut; but drainage is rarely used unless we know the wound is grossly infected. Wet antiseptic dressings are used for the first few days and moderate pressure is applied by wet gauze bandages. Avulsion of the Scalp is discussed on page 273.

**Contusions of the Head.**—Scalp swelling from hemorrhage is usually considerable. The patient may be stunned or dazed. The swelling of *hema-*

*toma of the scalp* must not be mistaken for *fracture* with depression. In hematoma there is a central depression; hard pressure on the center finds bone on a level with the general contour of the bone, and the margin of a hematoma is circular, is not quite hard, and is elevated above the general contour. In depressed fracture the edge is on a level with the central depression, is below the level of the general bony contour, and the margin is sharp and irregular. The **treatment** is by bandage-pressure. If suppuration arises, at once incise.

**Concussion, Contusion, and Laceration of the Brain.**—For many years it was customary to regard concussion as a condition produced by molecular vibrations in the nervous substance of the brain. Duret's classical observations profoundly modified surgical thought, and led to the opinion that in concussion of the brain there is injury to the brain itself, a rupture of cerebral vessels brought about by the advance and recession of waves of cerebrospinal fluid. This wave, it is thought, first flows in the direction of the force. Keen says that there may be slight brain injuries which can properly be called "concussions," but it is better to consider concussion as synonymous with laceration of the brain. Kocher considers concussion as identical with *contusion of the brain.* It seems, however, highly improbable that slight cases of concussion are accompanied by vascular rupture or organic mischief; the symptoms are too transitory and reaction too rapid and complete to permit of any such view. Experiments on animals show we can develop concussion without laceration or contusion. Autopsies have been carefully made in some cases of death from concussion, and no organic lesion has been discovered. It is quite true that the same force which causes the concussion may cause contusion or multiple lacerations, and a severe force is apt to do so. But we are not then justified in assuming that concussion is contusion or laceration: we should rather conclude that the individual had both concussion and a demonstrable injury. Both conditions arise from violence, but the two conditions are not identical. I believe, with von Bergmann, that there is such a condition as concussion, which may be pure concussion or may be associated with organic damage, and even if a man dies and is found to have an organic injury, the concussion may have caused or, at least, have hastened the fatal result. I believe, with von Bergmann, that it is not repeated waves of force from the blow, but the concussion of the blow itself that does the harm. The brain is momentarily displaced by the blow. The blow acts on the entire brain; the centers are first stimulated and then depressed, and in fatal cases are not only depressed, but are paralyzed. The cause of concussion is violent force, either direct (as a blow upon the head) or indirect (as a fall upon the buttocks). This force momentarily displaces the brain, giving rise to stimulation and then to exhaustion of the nerve-centers, and perhaps to rupture of vascular twigs, large vessels, or even the membranes. In the less severe cases concussion only exists; in the more severe cases there is also contusion or laceration or compression soon arises.

As von Bergmann points out, the entire cortex in concussion is momentarily stimulated and then depressed. The momentary stimulation exists when a man "sees stars" as a result of a blow. The depression or exhaustion is manifested by heaviness, dulness, stupor, perhaps by unconsciousness. The stimulation of the medullary centers, von Bergmann points out, lasts longer, as a rule, than the stimulation of the cortex, and is manifested particularly by a slow pulse. If the pulse grows rapid and weaker, the pneumogastric center is becoming exhausted and the patient is in danger of death. In slight cases of concussion only the cortex may be involved, the medullary center escaping. In rapidly fatal cases of concussion the medullary centers are quickly paralyzed.

**Symptoms.**—In very trivial cases the patient is slightly and momentarily

dazed and the pulse is temporarily slow and weak, but he is otherwise unaffected. In a rather slight case of brain concussion the patient may or may not fall; his face is pale; he feels weak, giddy, nauseated, and confused, but he soon reacts, and often vomits. The pulse is slow for a time and then becomes normal. In a severe case he lies in a state of complete muscular relaxation. The extremities are cold; the skin is pale and cold; the pulse is small and slow. The slow pulse is due to stimulation of the pneumogastric center; the respiration varies, being sometimes deep, sometimes superficial, sometimes rapid, and sometimes irregular. He seems unconscious, but can usually be roused to monosyllabic response by shouting, pinching, or holding a bright light near his face. Occasionally, however, there is complete unconsciousness. The urine and feces are often passed involuntarily. The pupils may be unaltered, may be dilated or contracted, may be equal or unequal, but in any case they will react to light. Paralysis rarely exists, but if there is paralysis, it is temporary. The temperature at first is subnormal. In a very severe concussion in which there is great danger of death the pulse is very rapid, small, weak, and probably irregular because of exhaustion of the medullary center, and the patient is absolutely unconscious because of depression of the cortex. If there is a severe cortical laceration there will be twitchings or even general convulsions, or the patient will lie curled up with limbs flexed and eyelids shut, and will resist all attempts to open his eyes or mouth or to move his limbs (A. Pearce Gould). Erichsen called this condition "cerebral irritability." If a patient with very severe concussion and very rapid pulse is going to get better, the pulse will become slower. If a patient with severe concussion and a slow pulse is improving, the pulse will become normally rapid; if he is getting worse, it will become abnormally rapid and weaker. How long may concussion last? As von Bergmann well says: Concussion is transient in its manifestations. It is a matter of a few minutes or, at most, a few hours, and any prolongation of severe symptoms beyond this time, especially if they are intensifying as time goes on, indicates an associated injury. When the patient reacts from concussion he will probably vomit. Within twenty-four hours he usually improves, but is feverish and complains of headache and lassitude, sometimes becomes delirious, and in rare cases develops mania. If the patient in concussion recedes from, instead of advances toward, recovery, coma will set in or inflammation will develop. The prognosis is always uncertain. Any concussion producing more than very temporary unconsciousness is almost surely a serious injury, because considerable laceration has probably occurred. Recovery from concussion may be complete and permanent, but, on the contrary, the entire nature may undergo a change. Such a change, which may not be evident for weeks or months, is apt to be manifested by egotism, selfishness, censoriousness, mendacity, great irritability, outbreaks of violent rage about trivial things, and forgetfulness. The forgetfulness is particularly as to recent events. There are headaches, insomnia, attacks of depression, lassitude, and vertigo. Such a patient is very susceptible to alcohol, the heat of the sun, and physical or mental strain. He can do nothing requiring mental effort.

After concussion a patient may develop hysteria, epilepsy, amnesia, or actual insanity. A condition resembling Korsakow's psychosis may develop (a condition of confusion with gaps in memory which are filled up spontaneously by fabrications, the patient also having multiple neuritis), melancholia, confusional insanity, or mania may arise, or a condition like hallucinatory paranoia or mental weakness, which may resemble paresis. Concussion may pervert or wipe out all memory of the causative accident and also, strange to say, of a varying period preceding the accident. The loss of memory of the accident is permanent; the amnesia for a period preceding the accident may

be permanent or may only be temporary. Statements made regarding an accident by one who has had concussion must be received with many grains of salt. A man may tell a story he believes himself, and yet it may be a mass of dream fancies without a word or with scarcely a word of truth.

**Treatment.**—In treating brain concussion bring about reaction by the administration of aromatic spirits of ammonia (no alcohol, as this agent excites the brain), by pouring a few drops of ammonia on a handkerchief and holding it near the nose, by surrounding the patient (who lies in bed with his head on a pillow) with hot bottles, by hot irrigation of the head, by the application of mustard over the heart, and by the administration of enemata of hot coffee or hot saline fluid. Do not pour fluid into the patient's mouth until be becomes able to swallow easily. If he cannot easily swallow, rely on hot enemata and hypodermatic injections of strychnin. Place the patient in bed in a quiet room and watch him. If reaction is inordinate, apply cold to the head, give arterial sedatives and diuretics, and purge. For some days or for some weeks, according to the case, insist on a very quiet life. For many weeks after a grave concussion a patient must be kept away from business and be watched, because of the possibility of an abscess of the brain arising, and because of the liability of such patients to develop hysteria, neurasthenia, or insanity. Give a plain diet containing a minimum of meat, administer an occasional purgative, and secure sleep. Sleep can often be obtained by some simple expedient, such as the administration of warm milk, placing a hot-water bag to the abdomen or feet, or applying a mustard plaster for a short time to the back of the neck. In cases in which obstinate wakefulness exists, it becomes necessary to give bromid, chloral, sulphonal, trional, or some other hypnotic. Morphin is avoided because it is thought to increase venous congestion of the brain, but the elder Gross often used it, especially in cerebral irritation. If signs of compression arise, it is best to trephine, as the compressing agent may be a clot (see page 786). If inflammation arises, some surgeons will not trephine; but most regard it as wise and proper, especially if the damage seems to be localized, to incise the scalp and inspect the bone. If a fracture is discovered and the symptoms are serious, perform an exploratory trephining, open the dura, and secure drainage for inflammatory products. Personally, I believe that trephining for drainage is indicated in such cases even when there is no fracture.

In any severe concussion of the brain with contusion of the scalp the surgeon should at once incise the scalp and inspect the bone.

**Compression of the Brain.**—The combination of symptoms indicative of cerebral compression may be present in a number of different conditions. We find these symptoms in abscess of the brain, tumor of the brain, intracranial hemorrhage, foreign bodies, inflammatory exudate, and fracture of the skull with marked depression. The symptoms of compression are expressive of impairment of the functions of the entire brain by insufficient and imperfect circulation of blood, this impairment of circulation being the result of a lessening in capacity of the cavity containing the brain, its membranes, the blood-vessels, and the cerebrospinal fluid (von Bergmann). Duret injected wax within the cranium of an animal and showed that a diminution of 5 per cent. in the intracranial capacity produced somnolence, and a diminution of 8 per cent. caused death. If a brain tumor, abscess, blood-clot, or portion of depressed bone occupies space previously given to brain matter, vessels, etc., there is less room within the skull to contain the special structures. The bones cannot yield, the brain is incompressible, so the cerebrospinal fluid is displaced, the vessels are squeezed, and the circulation is greatly impeded. Pressure upon either arteries or veins causes compression. This condition of cerebral pressure or compression is one of anemia. In reality it is compression of the vessels which feed the brain with blood, and such compression grievously disturbs

the normal relationship between the blood-supply of the brain and the circulation of cerebrospinal fluid. Compression begins in obstruction to the onflow of venous blood. It extends gradually to the arteries. The circulation is slowed, and because of slow circulation the activity of the centers is finally inhibited. It is stated by Cushing that the rise which occurs in the blood-pressure is conservative and is expressive of Nature's effort to maintain the circulation in the compressed medullary centers. Increased vascular tension is made manifest by estimating the blood-pressure and observing venous stasis in the optic disk. Increased tension of cerebrospinal fluid is shown by lumbar puncture. The fluid flows out rapidly or jets out. The cortex is temporarily stimulated and then depressed, because of impairment of nutrition. The medullary centers are first stimulated. The respiratory center is stimulated by retention of $CO_2$ in the blood, then the vasomotor center is stimulated, then the vagus, and finally, perhaps, the convulsive center (von Bergmann's "System of Practical Surgery"). The stimulation of the cerebral centers is followed after a time by weakening or actual paralysis. The centers are said to suffer in regular order, viz., the cortex, the corona radiata, the gray matter of the cord, and, finally, the medulla (Huguenin). As von Bergmann points out, by the time the convulsive center becomes stimulated the cortex is usually exhausted and the patient is unconscious. In compression the sensitive cortex first feels the effect and feels it most gravely, and the cortical impairment may last long after other trouble has passed. In some cases the cortex alone seems to be distinctly involved. When the vagus center is stimulated the pulse becomes slow; later, as the center becomes exhausted, it becomes rapid and weak, and this change has the same unfavorable significance as in concussion. If death occurs it results from paralysis of respiration and not of circulation. The displaceable cerebrospinal fluid is a great safeguard against compression, but Cushing has shown us that in intracranial obstruction of the venous circulation the flow of cerebrospinal fluid into the space about the cord is prevented because the medulla and cerebellum are jammed down in the foramen magnum.

**Symptoms.**—*Pressure symptoms* are divided into those occurring during the period of stimulation and those occurring during the period of increasing exhaustion. The symptoms of the first stage are headache, vomiting, flushing of the face, contraction of the pupils, choked disk, mental excitement, elevation of blood-pressure, restlessness, and slowing of the pulse. The pulse becomes slow, regular, and strong. The symptoms of the second stage are heaviness, dulness, drowsiness, passing into stupor, and finally into coma. The respirations are stertorous and after a time become Cheyne-Stokes. The pulse is weak, intermittent, compressible, and increasingly rapid. There are involuntary evacuations of feces and urine, and finally paralysis of respiration which causes death, the heart beating for a time after respiration has ceased (von Bergmann's "System of Practical Surgery").

The *headache* usually present in the first stage of compression is intense, persistent, sometimes general and sometimes more or less localized, and often aggravated by percussion of the cranium. It persists even in delirium, and the patient ceases to appreciate it only when unconsciousness begins. The *vomiting* is usually without nausea and is due to stimulation of the medullary center. At first vomiting may arise from taking food, but it soon continues independent of food. The tongue is probably clean. Cerebral vomiting is usually associated with severe headache. *Restlessness* is a pressure symptom in the stage of stimulation, and the patient rolls his head, tosses his body, and groans with pain. The *heart* does not begin to slow until the patient begins to be dull and drowsy, or until stupor arises, when the pulse slows and the

tension rises. Finally it becomes very slow—perhaps less than 40 in a minute. If the condition grows worse, the pulse after a time suddenly becomes rapid and of low tension, instead of slow and of high tension, a most unfavorable sign, indicating exhaustion and approaching paralysis of the vagus. In the stage of stimulation the patient is excited, unstable, delirious, and the condition of delirium gradually gives way to drowsiness, stupor, and coma. In some cases of compression there is distinct protrusion of the eyeballs. Before the patient becomes unconscious the pupils are contracted. When the patient is comatose, they are usually dilated, but may be contracted. In coma the pupils respond slowly to light or not at all. If the conjunctival reflex is gone, they will not respond at all (Gowers). In a lesion making unilateral compression toward the base the pupil on the side of the compressing cause is apt to be much dilated and even immobile. *Choked disk* begins in the stage of stimulation and continues to the end. That choked disk is due to intracranial pressure seems demonstrated by numerous operation reports, especially by Cushing, of Harvard, in which relief of pressure abated choked disk (see page 811). The existence of choked disk is determined by the use of the ophthalmoscope. The respirations become stertorous or snoring as coma develops because of the vibrations of the relaxed palate in the air-current, and the cheeks flap during expiration. As the activity of the respiratory center fails from increasing anemia, the respirations become shallow and infrequent, or, perhaps, of the Cheyne-Stokes type. Gowers defines Cheyne-Stokes breathing as "alternating periods of decreasing and increasing depth of breathing, separated by a pause" ("Lectures on Diseases of the Brain"). The *unconsciousness of compression* may be sudden or gradual, may be partial or complete. Apoplexy and many traumatisms cause immediate unconsciousness: the irritation of such a sudden lesion at once inhibits the cortex. A meningeal hemorrhage causes a gradually increasing unconsciousness. A brain tumor may cause heaviness, dulness, stupor, or, perhaps, after a long time, even coma. If compression comes on gradually, the brain more or less accommodates itself, and unconsciousness, if it occurs at all, is considerably deferred. A sudden increase of pressure may produce immediate unconsciousness. *Stupor* is partial unconsciousness, a condition in which a person lies as though asleep, though he arouses partially and temporarily when positively spoken to. In profound coma the limb reflexes are usually but not always diminished or lost. The superficial reflexes are impaired or lost. The muscles are flaccid and swallowing is impossible. In coma there is incontinence of feces and either incontinence or retention of urine. There may be the incontinence of retention. The *temperature* of a patient suffering from compression varies. In traumatic cases it may be at first subnormal and later normal or elevated. In inflammatory conditions it is elevated, except in abscess of the brain, in which it is subnormal, for a time at least, in half the cases. After an apoplexy it is for a time subnormal, but as shock passes away it becomes somewhat elevated. Any sudden compression causes shock and temporarily subnormal temperature. Lesions of the pons and medulla cause elevation—perhaps remarkable elevation—of temperature. In great or sudden brain compression complete coma always exists and there is no voluntary movement. In cerebral compression *paralysis* may exist, which may be very limited (monoplegia), may be of one side (hemiplegia), or may be general. In hemorrhage into the interior of the brain the unconsciousness is immediate or nearly so. In bleeding from the middle meningeal artery a period of consciousness intervenes between the injury and the coma, during which period blood collects and the coma comes on gradually. In compression from depressed fracture or from a foreign body the symptoms usually come on at once, but they may be deferred for some hours. Compression from inflammation or pus begins gradually after a considerable time has elapsed. The symptoms

described as pressure symptoms are those of pure compression. When traumatism causes the condition, the compression symptoms are mingled with those of concussion, or perhaps of contusion or hemorrhage. The brain adjacent to any lesion causing compression suffers more than the brain distant from it. The blood-supply of the entire brain is affected, but the adjacent brain has its capillaries particularly and directly compressed. Hence limited paralysis is sometimes produced by compressing lesions. The course of compression depends on the nature and persistence of the cause. Great temporary pressure may produce no permanent harm. Moderately severe pressure may be recovered from even after weeks of stupor. Great pressure, sufficient to induce coma, if not relieved quickly, will cause death. Persistent cerebral symptoms after a head injury, when no obvious lesion can be made out, are probably due to edema of the brain.

**Determination of the Cause of Coma in a Patient.**—A diagnosis must be made between coma due to brain injury and the comatose condition of apoplexy, uremia, epilepsy, hysteria, diabetes, opium-poisoning, and alcoholic intoxication. In hospital practice cases of unconsciousness without a known history are frequent. In attempting to diagnosticate, examine carefully for any evidence of traumatism, and inquire as to how and where the patient was found, if any fit occurred, and if a bottle or a pill-box was found near by or in the pockets. The surgeon should himself examine the pockets. Smell the breath to notice alcohol or opium, but always remember that an alcoholic is often a victim of Bright's disease, that a man with Bright's disease is liable to apoplexy, that a man may be stricken with apoplexy while he is drunk, and may fracture his skull by falling when under the influence of opium or of alcohol. The odor of acetone (violets) on the breath or in the urine indicates the existence of diabetes. Draw the urine with the catheter if any water is in the bladder. Examine the urine for albumin, acetone, and sugar, and take the specific gravity. In doubtful cases of coma have an ophthalmologist use the ophthalmoscope. He might find optic atrophy, indicative of Bright's disease, or choked disk, indicating compression. The cerebrospinal fluid obtained by lumbar puncture should contain blood if hemorrhage has taken place beneath the cerebral dura or in a ventricle of the brain. This test is valuable in fracture of the base of the skull, for in this condition cerebrospinal fluid is usually bloody. In *postepileptic coma* the temperature is never below normal, there are no unilateral symptoms, the condition resembles sleep, and the patient can be aroused. *Hysterical coma* occurs in boys and women; there are no objective symptoms, and the patient, though swallowing what is put into his mouth, cannot be aroused. In *uremia*, besides the condition of the urine (and always remember that a person with albuminuria is apt to develop apoplexy), there is a persistent subnormal temperature, and convulsions are prone to occur. There is perhaps edema of the legs, but paralysis and stertor are absent. In *apoplexy* hemiplegia exists, and the initial temperature is for a short time subnormal. A single convulsion may have ushered in the case. *Alcoholic unconsciousness* is often diagnosticated when apoplexy really exists. A man will smell of alcohol who has had one drink, but one drink will not produce coma; hence the smell of alcohol is not conclusive. In any case of doubt some hours of watching will clear up the diagnosis. Regard a doubtful case as serious until the truth is clear. In *opium-poisoning* the pupils are contracted to a pin-point, the respirations are usually slow, shallow and quiet, and may be stertorous, but there is no paralysis. Always remember that hemorrhage into the pons will produce pin-point pupils, but it also causes paralysis (crossed paralysis if in the lower half of the pons) and high temperature with sweating. In opium-poisoning the temperature is subnormal. In *diabetic coma* the pupils will react to a very bright

light, the temperature is subnormal, and the breath and the urine smell of acetone. (See Acidosis, page 1207, and Diabetic Coma, page 175.)

**Treatment of Brain Compression.**—The treatment of brain compression depends on the cause. Hemorrhage (extradural or subdural) requires trephining and arrest of bleeding; coma from depressed fracture demands trephining and elevation; foreign bodies must be removed; abscesses must be evacuated; some tumors are to be removed. In many tumor cases the growth is not removed, but a decompression operation is performed (see page 831). In cerebral compression, if death is threatened by respiratory failure, make artificial respiration and at once trephine over the supposed region of compression. Horsley has shown that irrigation of the head with hot water is of great value in bringing about reaction from shock in cases of brain injury.

**Intracranial hemorrhage** may be either *spontaneous* or *traumatic*. In the vast majority of instances spontaneous hemorrhage comes from the lenticulostriate artery (Charcot's artery of cerebral hemorrhage), and produces apoplexy, a disease belonging to the physician, except in some ingravescent cases, for which ligation of the common carotid on the same side as the rupture has been advised. In adults traumatism is almost always the cause of a meningeal hemorrhage. The blood may flow from a sinus, from the middle meningeal artery or one of its branches, or from vessels of the pia. Traumatism during delivery is an occasional cause of hemorrhage from the middle meningeal artery (Richardiére) and a not unusual cause of hemorrhage from cortical veins. Violent paroxysms of coughing in whooping-cough occasionally produce extradural hemorrhage or subdural hemorrhage. Geo. S. Brown reported such a case. He diagnosticated the condition and operated successfully ("New York Med. Jour.," April 25, 1903).

Fig. 511.—Fracture of skull with middle meningeal hemorrhage. Compression of brain by blood (Scudder).

**Traumatic Intracranial Hemorrhage.**—Hemorrhage may take place—(1) between the bone and the dura (*extradural*); (2) between the dura and the brain (*subdural*), and (3) in the brain substance (*cerebral*).

**Extradural meningeal hemorrhage** arises usually from the middle meningeal artery or from one of its branches. A spicule of bone may penetrate a venous sinus and produce extradural hemorrhage, or a sinus may rupture. Rupture of the meningeal artery or one of its branches is usually, but not always, accompanied by fracture (Fig. 511); in fact, in some cases not even a bruise can be found (Fig. 512). The ruptured vessel may be upon the opposite side to that on which the force was applied, hence the evidence of scalp injury is not a certain sign of the side of the skull involved. The accident may or may not cause temporary unconsciousness; but even if it does, from this unconsciousness the patient almost always reacts unless there are other grave injuries, and there is usually a *distinct period of consciousness* between the accident and the lasting coma, the coma being due to pressure from a continually increasing mass of extravasated blood (Fig. 513). If the main trunk or a large branch is ruptured the period of consciousness is short; if a small branch is ruptured the period of consciousness is prolonged for hours or perhaps for days. As the clot forms and enlarges the patient becomes heavy, dull, stupid, and sleepy; he sleeps so soundly he can scarcely be aroused, and snores loudly, and finally passes into stupor and then into coma.

The other signs of this condition are paralysis of the side opposite the blood-clot (not necessarily of the side opposite the point of application of the force, for the artery may rupture from contre-coup on the uninjured side); this paralysis is apt at first to be localized, but it gradually and progressively widens its domain. If the clot extends toward the base, the pupil on the same side as the clot ceases to react to light, and the immobile pupil dilates widely. If the clot be on the left side, aphasia may be noted. As the clot enlarges adjacent centers become involved. The face becomes paralyzed, then the arm, and finally the leg. Not unusually epileptiform attacks occur, starting in discharges from the centers which are irritated by the advancing clot before their function is abolished by pressure. The pulse becomes full, strong, usually slow, but occasionally frequent; the breathing becomes stertorous; the temperature rises, that of the paralyzed side exceeding that of the sound side. In a compound fracture the pressure of escaping blood may force brain matter out of the wound. In extradural hemorrhage from a sinus the symptoms cannot be differentiated from those produced by arterial rupture.

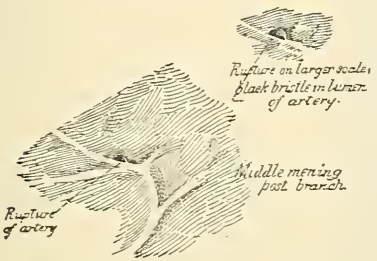



Fig. 512.—A case of rupture of middle meningeal artery. Preparation of dura. In the Warren Museum. The specimen is viewed from the outer side (Scudder).

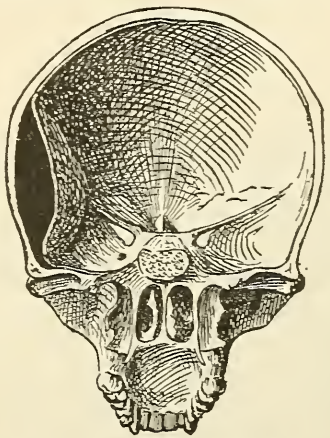


Fig. 513.—Frontal section of skull. Middle meningeal hemorrhage. The dura bulges inward toward the skull cavity (diagram) (Scudder).

*Treatment.*—In treating extradural hemorrhage localize the clot, not by the seat of the wound or contusion, but entirely by the symptoms. In a doubtful case endeavor to bring about reaction; but if the state of shock deepens or does not improve and if pressure symptoms increase, operate at once. To reach the middle meningeal artery or its anterior branch trephine $1\frac{1}{4}$ inches back of the external angular process, at the level of the upper border of the orbit (see Figs. 502, 509, 510). If the incision does not expose the clot, trephine again at the level of the upper border of the orbit and just below the parietal eminence (see Figs. 509, 510). The first incision gives access to the main trunk and to the anterior branch; the second incision exposes the posterior branch. If signs indicate that the clot is traveling to the base, the trephine should be used $\frac{1}{2}$ inch lower than the point first directed. Arrest bleeding by a suture ligament or by packing (see page 452), and always open the dura and inspect the brain. By this procedure a subdural hemorrhage may be discovered which, without it, would have been missed. Drainage must be employed.

**Subdural meningeal hemorrhage** is usually due to depressed fracture and rupture of the middle cerebral artery or of a number of small vessels.

The *symptoms* are identical with those of extradural bleeding, but are usually very rapid in onset and are accompanied by a more distinct drop in temperature and graver depression. The cerebrospinal fluid obtained by lumbar puncture is bloody.

The *treatment* is trephining for exploration at a point 1¼ inches back of the external angular process, enlarging the opening upward and backward by a rongeur, opening the dura, turning out the clot, ligating the bleeding point or packing, elevating any depression of bone, draining, and stitching the dura by catgut. Hemorrhage from internal pachymeningitis requires the same treatment.

**Cerebral Hemorrhage.**—The *symptoms* of cerebral hemorrhage are identical with those of apoplexy. The *treatment* is the same as that for apoplexy, except in ingravescent cases, when the common carotid on the same side as the clot may be ligated.

**Rupture of a sinus** may arise without a bone injury, but is usually due to a compound fracture. A sinus may be wounded during a brain operation. The **treatment**, if the rupture happens from fracture, is trephining. Enlarge the bone opening by the rongeur, pack with *one large piece* of iodoform gauze, or catch the rent with hemostatic forceps, leaving them in place for three or four days, or apply a lateral ligature or a suture ligature. Elevate depressed bone. If during an operation a sinus should be wounded, use a lateral ligature, a suture-ligature, or control hemorrhage by packing.

**Intracranial Hemorrhage in the Newborn.**—Certainly most of the cases of birth palsy seen in children are the result of subdural and subarachnoid hemorrhage at birth and damage of the cortical motor area. In such conditions there is spastic paralysis of the hemiplegic type, or if both hemispheres suffered there is plastic diplegia and usually amentia (Cushing, in "Amer. Jour. Med. Sciences," Oct., 1905). It has not been the custom to operate for hemorrhage in the newborn; most of the cases do not die, but remain for life weakened and paralyzed, epileptic, or idiotic.

The hemorrhage in cases of birth palsy is, as Cushing points out, usually venous and due to "rupture of some of the delicate and poorly supported venous radicles of the cerebral cortex" (Ibid.). It may result from traumatism due to bone overlapping or forceps pressure during parturition, or may arise during asphyxia after birth. Cushing discovered in examining stillborn infants and infants that died soon after birth that many of them died from cortical hemorrhage. In some the extravasations were very large, in fact, completely overlying a cerebral hemisphere. In some they were much smaller. In one the clot was in the cerebellar fossa.

The vessels usually torn are on one side and are the unsupported venous radicles which enter the longitudinal sinus, hence the leg center of one side is the cortical area most apt to be gravely damaged. If the vessels of both sides are torn, a bilateral cortical lesion results.

*Symptoms of Hemorrhage in the Newborn.*—In Cushing's masterly paper (Ibid.) the symptoms of recent hemorrhage are set forth. There is the history of a long and difficult labor, forceps perhaps having been used, or a history of postpartum asphyxiation. The fontanel bulges and perhaps does not pulsate. The fluid obtained by lumbar puncture contains blood-corpuscles. There is usually twitching and, as a rule, convulsions occur. They may occur soon after birth or not for several days. When they occur soon, they may be general; when they occur later, they may be unilateral. Paralysis is rare in the early days after birth. There may be alterations in the circulation and respiration. Pupillary alteration and ocular palsy seldom occur. If the child is not operated upon it may die or it may apparently recover. If it apparently recovers after a considerable hemorrhage, several months may pass before ominous symptoms are recognized. The late manifestations of the disease may be "spastic palsies, or blindness, or deafness, or feeble-mindedness, or, in severe cases, even complete amentia" (Cushing). Epilepsy may be a result.

*Treatment.*—Osteoplastic craniotomy in the parietal region, on one side or both, according to the unilateral or bilateral nature of the hemorrhage;

opening of the dura; washing out and turning out the clot; suturing the dura and closing the scalp without drainage. Cushing reports 4 cases, in 1 of which operation was done on both sides. He says chloroform should be given and that the parietal bone can be cut with blunt, curved scissors.

**Fractures of the skull** may be *simple, compound, depressed, non-depressed,* or *punctured. Fracture by diastasis* means separation of a suture or of sutures by violence. A fracture of the skull may be produced by a *bending* force, by a *bursting* force, or by an *explosive* force (see Gunshot-wounds).

A bending force is usually applied by the forcible impact of a body of small area. It produces a fracture and seldom causes distant injuries. The fracture may be of the inner table only, or of both tables. If both tables are fractured the broken bone is displaced and remains so.

In fracture by bursting, lines of fracture run to distant points from the seat of application of the force. Such an injury is inflicted by the impact of a flat surface of considerable area. In some cases we have a force which first is bending in character, but causes bursting to also occur, because there is no rebound.

Falls of large heavy objects or falls on the head, blows on the head from large flat objects, crushes in railroad accidents, etc., may burst the skull. In bursting fracture there is often widespread injury. The skull may be fractured during labor.

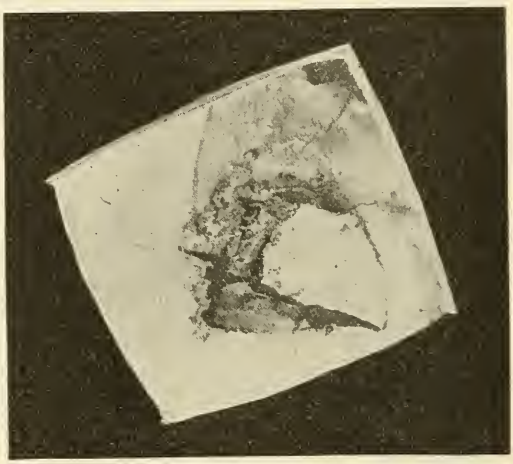


Fig. 514.—Fracture of the vault with extensive depression of the inner table ("American Text-Book of Surgery").

Fractures are divided into fractures of the *vault,* usually due to direct force, and fractures of the *base,* due to extension of fractures of the vault, to indirect violence (a fall upon the feet, the buttocks, or the vault), to forcing of the condyles of the lower jaw against or through the base, or to foreign bodies breaking through the orbit, vault of the pharynx, the ear, or the roof of the nostrils. *Fracture by contre-coup* was the name long given to a fracture which was supposed to occur on the side opposite the point of application of the violence. It is very doubtful if such a fracture ever occurs. I have seen meningeal hemorrhage by contre-coup, but not fracture. Fractures of the skull are uncommon in early youth, but they are much more frequent in the aged. Usually the entire thickness of the bone is fractured, but either the outer or the inner table (Fig. 514) may be broken alone. In complete fractures the inner table is broken more extensively than is the outer table, because the inner table is the more brittle, because the force diffuses, and also, as Agnew taught, because the inner table is part of a smaller curve than is the outer table, and violence forces bone-elements together at the outer table, but tears them asunder at the inner table (Figs. 515, 516).

**Fractures of the Vault.**—A fracture may involve the vault alone, but in over 60 per cent. of such fractures the base is involved. A fracture of the vault of the skull may be simple and undepressed, or it may be depressed (Fig. 514), compound, or comminuted (Fig. 517). A mere crack may exist in a bone, and if a rent exists in the soft parts, a bit of dirt or a hair may be caught in the crack. Fractures of the vault arise from direct force. A *fissure* may escape recogni-

tion, although in some cases percussion gives a "cracked-pot" sound. Any considerable depression can be detected. In a simple fracture occasionally the cerebrospinal fluid collects under the scalp and forms a tumor which pulsates and becomes tense on forcible expiration (see Spurious Meningocele, page 777). Compound fracture can be readily recognized, but do not mistake a suture, a

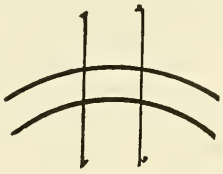


Fig. 515.—Section of outer and inner tables, with two parallel lines (after Agnew).

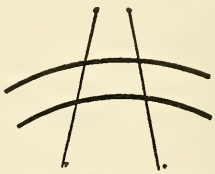


Fig. 516.—Greater yielding of the inner table than of the outer after the application of violence (after Agnew).

Wormian bone, or a tear in the pericranium for a fracture. A fracture bleeds, a suture does not. Even a narrow fracture is marked by a dark line of blood which *sponging will not remove.* Fracture of the inner table alone can only be suspected unless the *x*-rays make it evident. The prognosis of fracture of the vault depends upon the extent of intracranial injury rather than upon the extent of bone injury. Simple fractures may unite by bone; compound fractures with loss of bone unite only by fibrous tissue. The dangers may be *immediate* (hemorrhage, brain injury, and septic inflammation) or be *distant* (epilepsy, insanity, and persistent headache). In an open fracture the danger of infection is added to the danger of brain injury.

Fig. 517.—Fracture of skull with depressed fragments. Compression of brain by bone (Scudder).

*Treatment.*—The mortality of fracture of the skull was formerly much greater than at present. Before the days of antisepsis it was 51 per cent. (Harte). Trephining is performed much oftener than was once the custom, and is vastly safer. Out of 26 trephined cases, 3 died (Harte). In any case of fracture of the skull endeavor to bring about reaction before operating, unless the signs of pressure continually increase or the evidences of shock remain unimproved or become graver. A *simple fracture without depression* and *without brain symptoms* is treated expectantly (by rest, quiet, low diet, purgation, moderate elevation of and cold to the head, and arterial sedatives). A *simple fracture with moderate depression* and *without cerebral symptoms* is treated expectantly, and so also is a *simple fracture* in which *symptoms existed but are abating. Simple fracture with marked depression* requires immediate trephining, even when brain symptoms are absent. We make an exception in young children, and wait a while before trephining, in the expectation that the expansile brain will lift the depressed but elastic bone up to the level. Trephining in cases in which no symptoms exist, although there is marked depression, often prevents disastrous consequences arising in the future, and is known as *preventive trephining* (Agnew, Keen, Horsley, Macewen, von Bergmann, and others). In all *compound fractures* shave and asepticize the entire scalp, enlarge the incision, and explore the bone. If a fissure exists, it must be asepticized, and if a hair or other foreign body is found in it, in order to effect removal and secure asepsis the outer table of the

skull at this spot must be cut away by a chisel, the fissure being thus converted into a broad groove. In a *compound fracture with much depression* trephine, elevate, and irrigate. In any fracture trephine if distinct symptoms exist. In punctured wounds of the brain (*punctured fractures*) *always* trephine, open the dura, and disinfect. In a comminuted fracture the usual custom is to remove loose fragments. Schaak has recently advocated their reimplantation ("Archiv. für klin. Chir.," April 6, 1912). My usual custom has been to remove them. In any case of fracture of the vault in which trephining has been performed it is wise to open the dura and examine the brain. In an open fracture and after every operation of trephining in which the dura was opened administer urotropin in order to make the cerebrospinal fluid bactericidal.

**Fractures of the Base.**—A fracture of the base of the skull may exist in only one of the three fossæ, in two of them, or it may involve all. Figure 518 shows an extensive fracture of the base of the skull. The middle fossa is oftenest involved. Fracture of the posterior fossa is the most fatal. These fractures may be due to direct violence, to indirect force, and to extension of a fracture of the vault. Extension from the vault is always by the shortest route. Fractures of the base may extend up into the vault, and do so in over 80 per cent. of cases. Fracture by direct violence may arise from the penetration of the nasal roof, the orbital roof, or the pharyngeal roof by a foreign body. The posterior fossa may suffer from a fracture by direct violence applied to the neck. Fractures by indirect force may arise from blows upon the frontal bone (the orbital portion of the frontal or the cribriform process of the ethmoid breaking), from falls upon the chin (the condyle of the jaw breaking the middle fossa), or from falls upon the buttocks, the knees, or the feet (fracture occurring in the posterior fossa). The base is not broken by contre-coup.

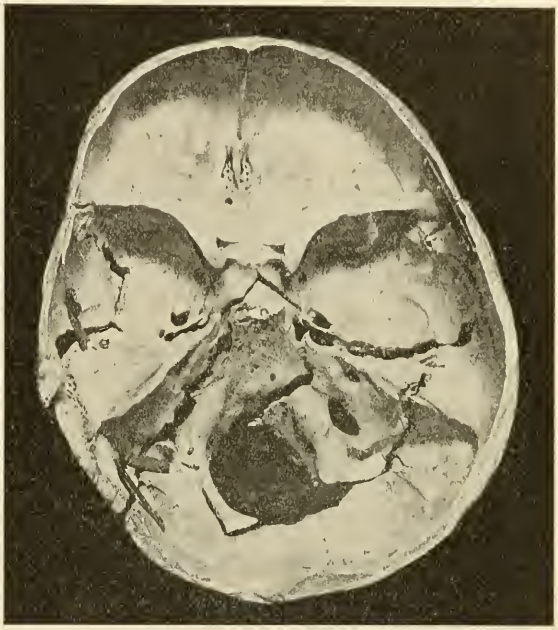


Fig. 518.—Extensive fracture of the base of the skull ("American Text-Book of Surgery").

*Symptoms.*—Fractures of the base of the skull are apt to be compound. A solution of continuity in the pharynx, roof of the nares, orbit, or ear permits access of air to the seat of fracture and allows blood and cerebrospinal fluid to flow externally. In *fracture of the anterior fossa* the fracture may be compound because of laceration of the mucous membrane of the nares or of the conjunctiva. Blood may run from the nose, its source being the vessels of the mucous membrane or the dura, the fracture being compound. Epistaxis does not prove the fracture to be compound, but only suggests it; but if the epistaxis is prolonged, the probability is greatly increased; and if the flow of blood is succeeded by a flow of cerebrospinal fluid the diagnosis of compound fracture is positive. Cerebrospinal fluid appears only when the mucous

membrane, the dura, and the arachnoid are each lacerated. In fractures of the anterior fossa blood is apt to flow into the orbit, producing *subconjunctival ecchymosis*, and perhaps pushing the globe of the eye forward. Some blood is often swallowed and vomited. In *fractures of the middle fossa* blood may flow from the ear through a tear in the tympanum, its source being the vessels of the tympanum, the meningeal vessels, or a sinus. Blood may flow through the Eustachian tube and come from the nose, may be spat up, or may be swallowed and vomited. In some cases a quantity of cerebrospinal fluid flows from the ear, the discharge being increased by expiratory effort and a position which favors gravity. Cerebrospinal fluid is at first blood-stained, but later becomes clear. The cerebrospinal fluid must not be confused with either blood-serum or liquor Cotunnii. The cerebrospinal fluid, if it flows at all, is always present in large amount; the liquor Cotunnii can be present only in minute amount. Blood-serum is highly albuminous; cerebrospinal fluid is a serous fluid of very low specific gravity, never shows more than a trace of albumin, and contains considerable chlorid of sodium and a carbohydrate now known to be a dextrose which reduces the copper of Fehling's solution and reacts to Trommer's and to Moore's tests, but does not refract polarized light nor easily ferment with yeast. Treves[1] states that cerebrospinal fluid cannot flow from the ear in fractures of the middle fossa —(1) unless the line of fracture crosses the internal meatus; (2) unless the prolongation of the membranes into the meatus is torn; (3) unless a communication exists between the internal ear and tympanum, and (4) unless the drum-membrane is torn. Miles, of Edinburgh,[2] claims that bleeding from the ear followed by a flow of cerebrospinal fluid is not pathognomonic of fracture of the middle fossa of the base. He maintains that when the drum is ruptured we may have these signs; when bone is not broken the chief source of the blood being the vessels of the pia and temporosphenoidal lobe, the blood and cerebrospinal fluid flowing inside the sheath of the auditory nerve, passing into the vestibule, through the lamina cribrosa, and from the vestibule into the middle ear, finding exits from this space by way of the Eustachian tube and also through the rent in the drum-membrane. Profuse mucous discharge may flow from the ear after an injury without fracture when the drum is ruptured, the fluid coming from the cells of the mastoid. It must be understood that fracture of the base may exist when there is no flow of blood or of serous fluid. A fracture of the middle fossa is usually compound, made so, even when the drum is not ruptured, by the Eustachian tube, and there is often paralysis of the seventh or eighth nerve or of both of them. In *fracture of the posterior fossa* there is usually respiratory derangement and blood accumulates beneath the deep fascia and produces discoloration in the line of the posterior auricular artery (*Battle's sign*), the discoloration first appearing near the tip of the mastoid. The discoloration appears in the line of nerves and vessels which emerge from the deep fascia, the vessels passing through openings and the extravasated blood emerging from the same openings. Fractures of the posterior fossa are apt to be compound through the pharynx, and in such cases the patient spits or vomits blood. Fractures of the posterior fossa are more fatal than fractures in either of the other fossæ because of the adjacency of vital centers. Fractures of the base are apt to be associated with *paralysis of cranial nerves*. The palsy indicates the situation of the fracture. In fracture of the anterior fossa the olfactory nerve may suffer. In fracture of the middle fossa the fascial nerve most often suffers. The eighth is sometimes injured. Other nerves which may suffer alone or in combination in fracture of the base are the abducens, the motor oculi communis, the trigeminus, the pneumogastric, the optic, the spinal accessory, the hypoglossal, and the glossopha-

[1] "Applied Anatomy." [2] "Edinburgh Med. Jour.," Nov., 1895.

ryngeal. *Optic neuritis* often arises after the first week. In fractures of the base the temperature is subnormal during the shock, rises to 100° to 101° F., falls again to about normal, and remains normal or subnormal unless there is inflammation or sepsis. Lumbar puncture may obtain bloody fluid. Such a finding means subarachnoid bleeding and indicates fracture. In any fracture injury of the brain may exist. Such an injury will be made manifest by symptoms, and we may or may not be able to diagnosticate and localize it. The prognosis is greatly influenced by the nature and extent of the intracranial damage. Harte ("Annals of Surgery," Oct., 1901) has collected 46 positive cases of fracture of the base of the skull from the records of the Pennsylvania Hospital; 35.5 per cent. recovered. Ransohoff collected 190 cases of fracture of the base of the skull. The mortality was 65 per cent. Over one-half of the fatalities were within twelve hours. Only 15 per cent. died after the second day. Of 98 cases with profound coma and respiratory disturbance 70 per cent. died ("Annals of Surgery," July, 1910). According to Hartley, in cases treated expectantly the mortality is 90 per cent., in cases treated by operation it is less than 35 per cent. ("Am. Jour. of Surgery," Dec., 1910).

*Treatment.*—In fracture of the base I now always do a subtemporal decompression, usually on both sides, as Cushing advocates. This is, first of all, exploratory, and may disclose a bleeding meningeal artery. After the dura is opened it enables us to evacuate fluid causing pressure and in which bacteria could multiply, and to prevent recurrence of pressure after the wound has been closed. If there is bleeding under the dura the brain should be lifted to let the blood out, and a drain of rubber tissue should be inserted. If there is only brain edema no drain is required. In some cases drainage has been obtained from the anterior fossa by breaking through the cribriform plate and introducing a tube by way of the nostril (Allis), and from the middle fossa by trephining above and behind the external auditory meatus. In a compound fracture of the orbit disinfect and drain. It may be necessary to trephine the roof of the orbit to secure drainage.

In addition to performing decompression I always give urotropin, as advised by surgeons in Johns Hopkins Hospital (S. J. Crowe, in "The Johns Hopkins Hospital Bulletin," April, 1909). This drug renders the cereborspinal fluid bactericidal. Other methods of treatment are secondary to the above. My experience is that this plan saves many cases which would otherwise perish. In treating a compound fracture of the base of the skull disinfect any cavity involved. In fractures of the middle fossa with ruptured drum clean the ear mechanically, wash it out by a stream of warm salt solution (turn the head toward the affected side while washing, so that the solution will not run down the Eustachian tube), insufflate iodoform, insert a piece of iodoform gauze, and apply an antiseptic dressing. Several times daily the ear is to be irrigated and insufflated with iodoform. The nasopharynx must be frequently irrigated by normal salt solution or boric acid solution and insufflated with iodoform. The conjunctival sac is frequently irrigated by boric acid solution. If after a head injury blood accumulates back of the drum, this membrane should be incised to permit of drainage and disinfection. In fractures of both the middle and anterior fossæ and in fractures of the posterior fossa communicating with the pharynx the nasopharynx must always be cleaned. The exact method depends on the choice of the surgeon. We may wash out these cavities frequently by hot water, next by peroxid of hydrogen, and finally by boric acid solution, or can simply use normal salt solution. After washing, insufflate the nasopharynx with iodoform. Repeat the cleansing at regular intervals and also cleanse the conjunctival sac frequently. In fracture of the posterior fossa examine to see if the fracture is compound, into the pharynx, and if it is, cleanse with great care the nasopharynx

and mouth, as previously directed. In a very extensive fracture of the base, besides use of the methods set forth above, the entire head should be shaved and a plaster-of-Paris cap be applied. A patient with fracture of the base must be put into a quiet and darkened room and be kept upon a low diet, sleep being secured, and the bowels and bladder being attended to. If we are uncertain as to whether a fracture exists or not, keep the patient quiet, in a darkened room and on a low diet. Attend to the bladder, keep the bowels loose, examine the nasopharynx with a mirror and the ear-drum through a speculum, and make a lumbar puncture.

**Obstetric Depressions of the Skull.**—These lesions seem to have been first studied by Danyau in 1849. The depression may be of the parietal or frontal bone and may or may not be accompanied by fracture. It may have been caused by the promontory of the mother's sacrum or by obstetric forceps. A slight depression does no harm, because it is gradually and spontaneously corrected. A marked depression, especially if accompanied by fracture, places the child in danger of epilepsy, idiocy, and non-development of body, and requires treatment. The usual treatment is trephining and elevation. Some claim to elevate by an apparatus making pneumatic suction. Some make a small incision, insert a screw (Heine's screw), and elevate by making traction on the screw. Hanch elevated by means of a corkscrew. (See Frazier, in "Progressive Medicine," March, 1913.)

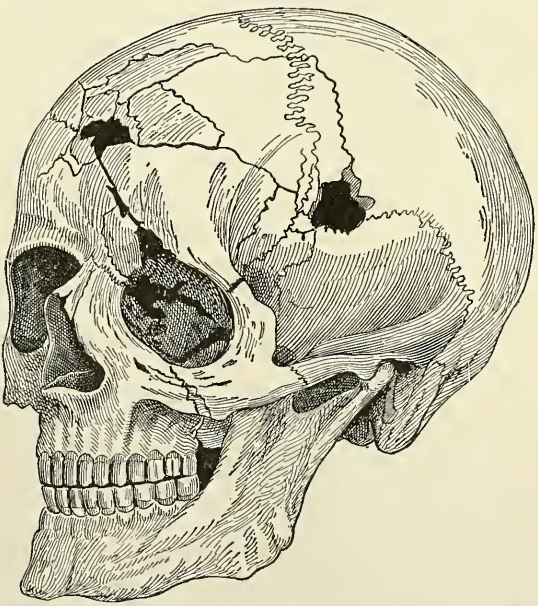


Fig. 519.—Extensively comminuted gunshot-fracture of the skull (after von Bergmann).

**Wounds of the brain** are produced by violence and especially by foreign bodies (knives, bullets, etc.). Except when due to penetration of a fontanel in a child or of a parietal foramen in adults, wounds of the brain are accompanied by fracture of the skull. These wounds are very dangerous; foreign bodies (bone, hair, clothing, etc.) are often lodged in the brain, hemorrhage is usually severe, and without proper treatment sepsis is almost inevitable. Such cases are very fatal, though some astonishing recoveries are on record. Figs. 519 and 520 show gunshot-fractures of the skull.

The **symptoms** of a brain wound may be slight and long deferred or may be immediate and overwhelming; they depend upon the site and extent of the injury. Localizing symptoms may exist, and encephalitis with coma is apt to arise. Abscess may follow.

In treating wounds of the brain always shave the entire scalp and examine the weapon, if possible, to see if a piece were broken off. Asepticize, enlarge the wound, trephine, arrest bleeding, elevate any depression, remove foreign bodies, irrigate the wound with salt solution, drain by gauze, suture the dura, and dress.

**Wounds in War.**—When the bullet of a military rifle, fired at very close

range, crosses the brain it may blow the skull into fragments, but often it does not, but produces fracture of the skull and wound of the brain. The "explosive effect" is far less marked on the head of a living man than on the head of a corpse and may even be absent when the range was only 100 yards. At moderate range, at the point of initial contact of the bullet with the skull, a fracture is produced, the opening is slightly larger than the bullet, and short fissures commonly radiate from it. Fragments from the internal table are usually displaced and driven into the brain. The wound of exit is more irregular and is apt to exhibit more and longer fissures than the wound of entrance. When a bullet strikes a glancing blow it may fracture the outer table alone; it may produce a "gutter-fracture" (two scalp openings, and "a gutter ploughed" through both tables of a portion of the skull, as O'Reilly expressed it), *very seldom* a fracture of the inner table only, *penetration* of the skull, and lodgment of the bullet, or *perforation* of the skull, the bullet passing through the head and emerging. Nearly half the cases are instances of perforating wound.

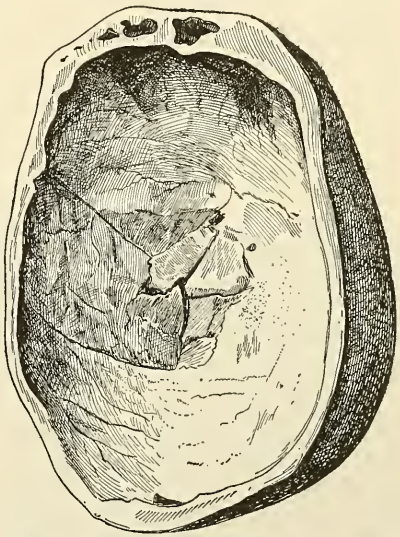


Fig. 520.—Gunshot-fracture of internal table of the skull (after von Bergmann).

In all of these injuries there is great shock and usually concussion, but concussion symptoms may be absent. The patient may die at once or almost at once, but if he is alive a few hours after the injury he has still to face the danger of infection and resulting inflammation. The danger depends on the brain injury and the amount of infection and not on the extent of the bone damage. The symptoms vary according to the part of the brain injured and the extent of the damage.

In practically all cases bone-fragments are driven into the brain, and as the scalp is a dirty region, the wound is more or less infected.

The mortality from these injuries is very large. In the American Civil War it was 61.2 per cent. In the Franco-Prussian War it was 51.3 per cent. In the Boer War, among the British it was only 33.1 per cent., a very notable improvement. (See "Military Surgery," by Surgeon General Robert M. O'Reilly, U. S. A., in "Keen's Surgery," vol. iv.) In the Russo-Japanese War the mortality seems to have been something over 37 per cent.

In estimating mortality those killed outright and those dying before reaching the hospital are not counted by makers of statistics. Much brain matter may ooze out from such a wound. Cerebral hernia (Fig. 522) is common after these injuries, especially if much bone was destroyed by the injury or removed by the surgeon.

**Treatment of Wounds in War.**—At the first-aid station antiseptic dressings are applied. On reaching the field hospital the wound is explored, if time, situation, and military necessities permit. Every wound of this sort is regarded as being complicated by infection and by depression or wide dispersion of bone-fragments. The patient is reacted from shock and a flap of scalp is turned down to permit of exploration. Depressed fragments of bone are elevated or removed and loose pieces are removed. Treatment is the same as for wounds from revolver bullets (see page 796).

**Wounds from Revolver Bullets.**—The bullet may strike the skull and glance (if fired at an angle) with or without the production of a fracture. A small bullet (No. .22) may even strike perpendicularly and fail to enter, sometimes causing a fracture and sometimes not doing so. Even a No. .22 may enter the skull. I removed a bullet of this size which had entered and crossed the skull and lodged beneath the cortex on the opposite side of the brain. A bullet may cause a "gutter-fracture"—may enter the cranium and lodge—or may cause a complete perforation. A revolver bullet is much more apt to lodge than a military bullet.

The wound of entrance is small; the wound of exit is larger. At the wound of entrance the inner table is more extensively fractured than the outer table; at the wound of exit the outer table is more widely broken than the inner table. In these cases there is always great shock and usually concussion, and concussion symptoms may exist even when the bullet has not entered the brain. In moderate concussion the action of the heart is retarded; in severe concussion it is accelerated (see page 781). A bullet may be lodged within the cranium when merely a fracture without a bullet-hole can be detected. In these cases the bullet produced a fracture and entered the cranium, and then the depressed bone flew back into place (von Bergmann). In such cases, if complete perforation occurs, the one existing opening in the bone is the opening of exit. A bullet may lodge in the bone, between the dura and the bone, between the dura and brain, in the brain, between the dura and the brain or the dura and the bone of the opposite side, or in the bone of the opposite side, in the nasal fossa, maxillary antrum, or orbit. Always examine the side of the head opposite to the wound of entrance to determine if there is a wound of exit or any bulging or fracture. A bullet may pass across the brain and be deflected from the inner surface of the skull. Ruth does not believe the bullet can rebound from the opposite wall.[1] If certain regions are injured, localizing symptoms may arise. Much brain matter may ooze out from the wound. Loss of brain matter sometimes causes great impairment of function, sometimes little or none. The secondary **symptoms** of gunshot-wounds of the head are varied and uncertain, and may not be observed at all before death. Fowler wisely points out that a patient with a gunshot-wound of the head may have also received other injuries, and the other injuries may be in part, at least, responsible for cerebral symptoms.

**Treatment of Wounds from Revolver Bullets.**—Endeavor to bring about reaction (see Concussion). In severe cases apply heat to the head and make artificial respiration. It will sometimes be necessary to operate while artificial respiration is being made. In treating gunshot-wounds of the head shave and asepticize the whole scalp, disinfect the entire track of the ball, and arrest hemorrhage at the wounds of entrance and exit, using the rongeur to expose the bleeding points if the bullet be large, employing the trephine if it be small. If the bullet has emerged and has been picked up, examine it to see if it is entire. The bullet, if retained, is to be sought for. The *x*-rays are invaluable in locating the missile. Place the head in such a position that the track of the ball will be vertical, then introduce Fluhrer's aluminum probe or Senn's probe, and let it find its way by gravity. The probe may find the ball near the wound of entrance, in which case extract the ball with forceps; or the probe may find the ball near the opposite side of the head, in which case make a counteropening through the bone at a point the probe would touch if it were pushed entirely across. Take a new and *clean* rubber catheter (No. 9, French), insert a stylet, and carry the catheter through the wound (Keen). Knowing the depth of the ball, search for it around the catheter-tube as an axis, and when found, extract it. After extraction drain the wound by means of a tube. When

[1] See the instructive article by Fowler, in "Annals of Surgery," Nov., 1895.

a counteropening exists, drain through and through. If the ball cannot be detected, drain by a tube carried to the depths of the wound. After dressing always place the head in a position favorable to drainage. Fluhrer tells us that when a counteropening fails to disclose the bullet, use the new opening as a doorway through which to search for the ball. He believes the bullet is not unusually deflected. The angle of deflection is somewhat greater than the angle of incidence, and the bullet is apt to fall a little toward the base. Splinters

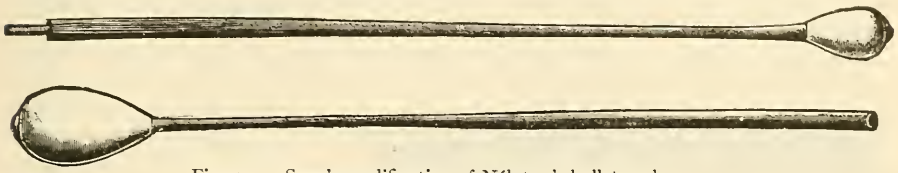


Fig. 521.—Senn's modification of Nélaton's bullet-probe.

of bone are often driven into the brain by a bullet, and these should be removed whether the ball is found or not. Several varieties of probes have been recommended. Fluhrer uses a large-sized aluminum probe. Senn used an instrument shaped like the Nélaton probe, but of the same diameter as the bullet (Fig. 521). (Of course, the porcelain probe will not show a black mark from contact with a hard-jacketed bullet.) Fowler uses a graduated pressure probe; so long as the pressure is within the limits of the spring, as shown by the scale, the probe is in the bullet-track. Girdner's telephonic probe has been used as an aid to localization. Bullets are now certainly located by the Röntgen rays. There can be no doubt that many gunshot-wounds have been recovered from without operation, and it is beyond question that many deaths follow operation (about $33\frac{1}{3}$ per cent., according to Hahn). Von Bergmann is so impressed with these facts that he does not operate when cerebral symptoms are absent. I usually operate.

Fig. 522.—Hernia cerebri under scalp after operation for brain tumor (W. W. Keen).

**Prolapse of the Brain and Hernia of the Brain.**—In a compound fracture, especially a gunshot-fracture, with torn dura and pia, brain matter may emerge from the wound. In fracture of the base brain matter may enter the orbit, the nose, or the ear. A flow of brain matter may continue from a wound for many hours. A week or more after an injury a portion of the brain may protrude or prolapse. To this condition the term *prolapse of the brain* should be applied. In many instances the protrusion is covered with pia, but if the pia were torn or cut, it will not be a covering. This protrusion emerges from the opening in the skull, mounts up, growing larger and larger, until it may become the size of a fist. It usually pulsates. When bare it is soft, lobulated, of a dirty-white color, pulsating, painless to the touch, often bleeding, and sometimes discharging cerebrospinal fluid. Death may soon follow such protrusion, but the protruding mass may become necrotic and be sloughed off, a granulating surface

remaining, which heals. *Hernia cerebri* (Fig. 522) sometimes follows operations upon the brain or injuries of the skull and dura, when large pieces of bone have been removed or when the dura has been widely cut or torn and has not been carefully sutured. The condition is due to increased cerebral pressure. Hernia of the brain is protrusion through the dura, but not through the scalp, the scalp wound being healed above the protrusion. In a decompression operation we deliberately create a hernia of the brain. Prolapse of the brain is treated by antiseptic dressings and perhaps by a decompression operation. Skin-grafting benefits some cases. Pressure is dangerous. Excision by the knife or cautery seldom does any good. Hernia in some cases can be treated by repeated lumbar punctures, in some others by craniotomy of the opposite side of the skull.

Fig. 523.—Fungus cerebri (W. W. Keen).

**Fungus Cerebri** (Fig. 523).—When the brain is exposed, a granuloma may grow from the neuroglia and fungate through the opening in the skull. This condition is fungus cerebri and is not composed of brain matter. It is due to infection of the brain, and is most frequent when a bit of bone or some other foreign body is retained. A fungus is soft to the touch, is livid in hue, bleeds easily, frequently contains multiple foci of suppuration, and pulsates. It often attains the size of a small orange. It is treated by removing the granulations and any foreign body, and applying, with moderate pressure, aseptic dressing soaked in alcohol. After healing, a depression marks the site of the fungus.

**Traumatic inflammation of the brain and its membranes** is divided into *encephalitis* or *cerebritis*, inflammation of the cerebrum; *cerebellitis*, inflammation of the cerebellum; *meningitis*, inflammation of the meninges; *arachnitis*, inflammation of the arachnoid; *pachymeningitis*, inflammation of the dura; and *leptomeningitis*, inflammation of the arachnoid and pia.

**Meningitis.**—Of recent years our views regarding meningitides have changed. We no longer regard each form as a separate and distinct disease, but we regard all the forms as different phases of one disease, that disease being a reaction to infection on the part of the membranes of the brain and cord. We must still, for clinical convenience, classify meningitides into many types. We speak of meningitis as serous or purulent, local or diffuse. We speak of it as basilar, leptomeningitis, etc., or as tuberculous, meningococcic, etc. But, after all, meningitis is an infection for which many varieties of bacteria may be responsible, and in which there are many different routes for the entrance of the causal micro-organisms, but the effects of these organisms upon the tissues are similar and the variety of the symptoms produced depend upon the region diseased and the power and nature of the toxins.

This view has been most ably set forth by Kopetzky ("The Laryngoscope," June, 1912). The same careful observer points out that in every form of meningitis there is increased tension of the cerebrospinal fluid and poisoning of the central nervous system by toxins and products of tissue metabolism.

The chief factors in causing increased tension of cerebrospinal fluid are edema of brain tissue and edema of the membranes.

Kopetzky ("The Laryngoscope," June, 1912) further points out that very early in the progress of every case of meningitis there is a disappearance from the cerebrospinal fluid of the copper-reducing substance dextrose. Bacteria are greedy for carbohydrates and eat up dextrose. The disappearance of dextrose is highly significant of the existence of meningitis. Its reappearance indicates abatement of the inflammation.

**Treatment.**—Kopetzky (Ibid.) in acute cases advocates the administration of serum or antitoxin and the early performance of an operation to reduce intracranial tension. Lumbar puncture is of high diagnostic, but small therapeutical, value. The most promising operation is one which drains the cisterna magna (see Haynes's Operation, page 832).

**Pachymeningitis Externa.**—Inflammation of the external layer of the dura is called *pachymeningitis externa*. It may arise from tumor, caries, necrosis, middle-ear disease, sunstroke, or traumatism. Syphilis is a not unusual cause. The other membranes may become involved. Suppuration may arise, having extended by contiguity from neighboring parts.

The **symptoms** of pachymeningitis externa are uncertain. They resemble often those of leptomeningitis (see page 800). Pressure symptoms may arise. Headache is always present. Paralysis may or may not exist. If pus forms, the ordinary constitutional symptoms of suppuration are evident (high temperature and sweats), not the usual symptoms of abscess in the brain. In a severe case the other membranes become involved.

The **treatment** consists in removing the cause (carious bone, pus, middle-ear disease). In pachymeningitis from traumatism it is sometimes advisable to trephine in order to drain inflammatory products; in a case with localizing symptoms always trephine; in an ordinary case, without pus and with no evidences of traumatism, use wet cups back of the mastoid processes, apply an ice-bag to the head, and purge by means of calomel. Administer iodid of potassium in most cases. If sunstroke is the cause, treat according to ordinary medical rules.

**Pachymeningitis Interna.**—This term means inflammation of the inner layer of the dura. Inflammation may extend from the pia or from the outer layer of the dura. The disease is most often met with in infants and in the chronic insane, but may occur in those not insane in late middle age or beginning old age. The form known as *hematoma of the dura mater*, or *pachymeningitis interna hæmorrhagica*, may arise during infectious disease (typhoid fever or rheumatism), in persons of the hemorrhagic diathesis, in diseases causing atrophy of the brain, in chronic diseases of the heart and kidneys, and in syphilitics. Among the exciting causes are traumatism, inflammation in adjacent parts, and, especially, the abuse of alcohol. In this disease blood is extravasated on the inner surface of the dura. Many observers do not class hemorrhagic pachymeningitis as inflammation, but regard the hemorrhage as primary.

The **symptoms** of internal pachymeningitis are very chronic, come on gradually, are not characteristic, and may be absent. They consist usually of mental irritability or excitement, followed perhaps by hebetude and persistent headache; and apoplectiform attacks, with contraction of the pupils, slow pulse, and vomiting; there may also be muscular rigidity and spasm of the extremities. Choked disk is not infrequent; localizing symptoms may be made out, and coma is apt to arise. Cranial nerves are seldom affected.

The **treatment** is operation, which removes the clot. This is unpromising, but Munro saved 1 case out of 5 ("Chicago Med. Recorder," Dec., 1902).

**Acute leptomeningitis** is a purulent inflammation of the soft membranes of the brain. The pathological changes can be noted in the pia and in the brain substance. The brain is edematous, the pia purulent, the convolutions are flattened, the ventricles are distended with fluid, and hemorrhages occur into the brain substance. Pus may be localized upon the pia, but it is usually diffused over one hemisphere or over both. Various organisms may be found, especially streptococci, staphylococci, and diplococci. In some cases we find the Bacillus pyocyaneus or the Bacillus pyocyaneus fœtidus, which is identical with the colon bacillus and with the Bacillus meningitidis purulentæ (Park). Saprophytic organisms are occasionally present. This disease may be acute or chronic, and a severe case is spoken of as *encephalitis*. *Secondary leptomeningitis* is apt to affect the convexity; *primary leptomeningitis* is apt to affect the base.

The **causes** of leptomeningitis are epidemic cerebrospinal fever, tuberculosis, acute general diseases (pneumonia, typhoid, erysipelas, and rheumatism), bone diseases, traumatism, middle-ear disease, syphilis, and sunstroke. In diplococcic meningitis the tissues of the *pia* and the cerebrospinal fluid contain diplococci identical with pneumococci. Infection may take place by various avenues. It may pass from the pharynx by way of the Eustachian tube to the ear, or from the nose to the frontal sinus or ethmoid sinuses (Hirt), and from these situations to the brain. It may pass from the middle ear or mastoid to the membranes of the brain. In fractures at the base the organisms enter by way of the pharynx and the Eustachian tube, or the ear. The **symptoms** of acute leptomeningitis early in the case are: rising blood-pressure (determined by the sphygmomanometer), edema of the optic papillæ, absence of carbohydrates from the cerebrospinal fluid (obtained by lumbar puncture), "an irritable or clouding sensorium" (Irving S. Haynes, in "The Laryngoscope," June, 1912). The same author adds that vagus pulse and respirations irregular in depth and rate may also be present. As the case progresses there are violent headache persisting during delirium, flushing of the face, rigidity of the neck, cerebral vomiting, a pulse small and irregular in force and frequency, but often slow, elevated temperature, leukocytosis, photophobia, contraction and perhaps inequality of the pupils, intolerance of sound, hyperesthesia of the skin and muscles, and delirium passing into stupor and coma. There are rigidity of the muscles of the neck, retraction of the head, retraction of the abdominal muscles (boat-shaped abdomen), inability to extend the legs when sitting up, though it can be done when recumbent. If, while lying down, the thigh is flexed on the belly the leg cannot be extended on the thigh (*Kernig's sign*). If a dull point is drawn along the skin a red line follows it (*tache cérébrale*). A chill or a succession of chills may occur. Choked disk, strabismus, and nystagmus are not unusual. Twitching convulsions or paralyses may occur. Death is the rule within one week. In a case of meningitis the fluid obtained by lumbar puncture may contain bacteria and may be actually purulent. The presence of numerous leukocytes is usual, there being an excess of polymorphonuclear forms in pyogenic infections.

**Treatment.**—Flexner's serum is valuable in cerebrospinal meningitis, a condition caused by the Diplococcus intracellularis, but is useless in all other conditions. Lumbar puncture is of immense diagnostic importance, but is useless therapeutically. It is not entirely safe. The rapid withdrawal of fluid may cause death by allowing the foramen magnum to be plugged by the brain stem. Ordinary trephining with tapping of the ventricle is practically useless. An operation which promises is the exposure and drainage of the cisterna magna (see page 832). If employed it should be done early, that is, when what we regard as early symptoms are present. Late operations are of no avail. Any causal condition, for instance, suppurative otitis media, must also be treated by

operation. After operating administer urotropin. Should the patient recover, he must be guarded for a long time from physical exertion, mental excitement, worry, irritation, constipation, and insomnia.

**Chronic Leptomeningitis** (*or Chronic Encephalitis*).—The causes of chronic leptomeningitis are the same as those of the acute form. If traumatism is the cause, the inflammation arises at a later period than it would in acute encephalitis. The symptoms of concussion follow a head injury. Days, or even weeks, after the accident a series of symptoms may occur, namely, localized pain at the seat of injury, often accentuated by tapping; listlessness; irritability; apathy regarding business affairs and home obligations, or profound depression and hypochondria with inability to attend to business. Choked disk may exist. In any case acute encephalitis may arise, with or without a chill. The **treatment** of this disease is symptomatic unless local symptoms exist. Always trephine if localizing symptoms are found. Intense local pain justifies trephining.

**Tuberculous Meningitis (Acute Hydrocephalus; Water on the Brain).**—This inflammatory condition is due to the bacilli of tuberculosis. In a child affected with tuberculous meningitis there is often a record of a fall, the injury acting as an exciting cause by establishing an area of least resistance. Prodromal symptoms lasting several weeks are common (restlessness, irritability, anorexia, loss of flesh, change of character). The attention of the physician is attracted to the meninges by a convulsion or, what is more common, by headache, fever, and vomiting. The fever is persistent, but irregular. The child cries out from pain (*the hydrencephalic cry*) and the bowels are constipated. The pulse is rapid in the beginning, but later becomes slow, irregular, and of high tension. The pupils are contracted, there is muscular twitching, and the sleep is impaired. The temperature is about 103° F. There is usually edema of the optic papillæ and carbohydrate disappears from the cerebrospinal fluid (see page 799). In the second period of the disease the vomiting ceases, constipation becomes more marked, the belly retracts, headache is not so violent, and the patient lies in a soporose condition interspersed with episodes of delirium. In this stage the pupils dilate and are often unequal, the head is retracted, convulsions occur or limited rigidity is noted, the respirations are sighing, and if a finger-nail is drawn along the skin, a red line develops (the *tache cérébrale*, due to vasomotor paresis). Kernig's sign is present (see page 800). Squint and consequent double vision are usual. In the last stage coma becomes absolute and general convulsions or limited spasms are apt to occur. Optic neuritis exists, and the child passes to death along a road identical with that of typhoid collapse. In some cases the examination of cerebrospinal fluid withdrawn by lumbar puncture throws light upon the diagnosis. The fluid is devoid of carbohydrate and usually contains an excess of lymphocytes. It may contain the bacilli. It may be sterile. Sterile fluid from a patient with symptoms of meningitis suggests that the condition is tuberculous. In children the base of the brain is usually involved, and the disease is apt to last from two to four weeks; in adults the convexity is usually involved, and death is apt to occur in a few days. When the meningitis is basilar, occipital headache, rigidity of the neck, and vomiting are severe, paralysis of various cranial nerves may occur, and there may be hemiparesis from pressure on the crus. Absence of leukocytosis points to a tuberculous cause for a meningitis. The existence of leukocytosis does not disprove the tuberculous cause of the disease.

The **treatment** is like that for traumatic meningitis. The operation of trephining, perhaps with tapping a ventricle, seldom offers any chance of improvement, and never does unless the process is limited in area and confined to the convexity. Lumbar puncture is performed for diagnostic rather than

for therapeutic reasons. Draining the cisterna magna should be tried (see page 832).

**Abscess of the brain** is a localized collection of pus. The bacteria which may be found are noted upon page 800 (Acute Leptomeningitis). The **causes** are suppurative otitis media (in half of all the cases), fracture of the skull, osteomyelitis of the cranial bones, erysipelas of the scalp, subaponeurotic abscess of the scalp, abscess of the lung, gangrene of the lung, empyema, concussion or wound of the brain, and general infections. As Ballance points out, abscess of the brain complicating head injury is not really an abscess of the brain unless the wounding material entered into the brain substance. In most cases the abscess is "a local meningeal suppuration with participation of the adjacent brain cortex, a meningocortical abscess rather than a brain abscess proper" ("Some Points in the Surgery of the Brain," by Chas. A. Ballance). General infections may cause abscess (pyemia, tuberculosis, and specific fevers). A tuberculous mass may caseate (tuberculous abscess). The abscess may be between the dura and the skull (extradural), adhesions forming and preventing general leptomeningitis, between the dura and brain (subdural), or in the brain substance (cerebral or cerebellar). Leptomeningitis may arise because no adhesions are created, because septic clots form in veins or sinuses, or because infected blood regurgitates into the sinuses (Park). A traumatic abscess is generally beneath the area to which the traumatism was applied, but it may be on the opposite side. Source of infection may be the nose, the orbit, or the middle ear (see page 800). Roswell Park says infection may pass along blood-vessels, lymph-vessels, nerve-sheaths, or the prolongations of the membranes which extend outside of the skull. An acute inflammation of the middle ear rarely causes abscess, because an acute inflammation in sound tissue causes the formation of granulation tissue, which acts as a barrier to infection. Chronic inflammation of the middle ear is the most frequent cause of abscess. Park tells us that if the roof of the tympanum is involved, it may perforate and abscess of the middle fossa may form; if the tympanum is perforated toward the mastoid antrum, the abscess arises in the temporosphenoidal lobe; if the perforation is toward the sigmoid groove the abscess forms in the cerebellum.[1]

Chronic bone disease is seldom followed by spreading meningitis, often by abscess. When infection reaches the brain by direct extension from a suppurating bone it must pass through the membranes, but it is usually limited by adhesions. The cortex is very vascular, strongly resists infection, and is seldom extensively destroyed, but the white matter is far less resistant and abscess tends to form in it (Ballance, Ibid.). In some cases of abscess of the temporosphenoidal lobe following ear disease the cortex seems normal, in others the membranes and cortex are fused over a narrow area which constitutes the stalk of an abscess in the white substance of the lobe. This is the *mushroom abscess* of Ballance. An abscess may increase rapidly in size and finally break into a ventricle or through the cortex. It may become encapsulated and latent. A slow-growing abscess may push aside nerve-fibers as does an encapsulated tumor, but a rapidly growing abscess destroys them.

In the cerebrum, multiple abscesses, except in cases of general infection, are seldom seen. In the cerebellum they are not uncommon. One or several abscesses may arise from a primary one. If they are adjacent to the primary one they are called satellite abscesses and tend to break into the older purulent area.

Ballance, in considering the onset of abscess, adopts the views as to the five types set forth by Brissaud and Souques. These types are as follows (Ballance, Ibid.):

(1) A subacute evolution. In this there is a febrile onset, like the onset of a

[1] Park, in "Chicago Med. Record," Feb., 1895.

specific fever, with headache, vomiting, and elevated temperature. After a few days there comes a remission, the period of delusive calm. In this period symptoms are absent or trivial. Though there may be progressive emaciation, there is no elevation of temperature in this period.

Suddenly convulsions occur which are followed by coma, or coma arises without antecedent convulsions. The patient may die in coma or the coma may pass away, "the symptoms indicating a local brain lesion" (Ballance, "Some Points in the Surgery of the Brain"). In this stage elevated temperature may appear again.

(2) Evolution with violent general infection, the symptoms of abscess being merged and usually lost in the symptoms of general infection.

(3) Evolution with complete latency. The patient presents no symptoms until a few hours before death, or he may die suddenly without a symptom having been observed. In this connection Ballance speaks of the difference between "symptoms not noticed and symptoms not present" (Ibid.).

(4) Only when symptoms were not present does he use the term "complete latency" to indicate the condition. Ransohoff has reported the case of a boy in whom an abscess in the frontal lobe was latent for three years, and another case in a man due to gunshot-injury, which was latent for four and a half years and in which nearly ten years elapsed between the injury and death ("Annals of Surgery," July, 1909).

(5) Onset not to be distinguished from a brain tumor.

(6) Onset with headache and fever, or with mental excitement. Then the patient appears to get completely well and remains so for weeks, for months, or for a year or more. This condition may occur in abscess secondary to influenza (Ibid.).

**Symptoms of Abscess of the Cerebral Substance or of the Cerebellum.**—The symptoms due to pus formation are as follows: There is an initial rise of temperature, but (except in extradural abscess) the temperature may quickly become normal or even subnormal. Years ago Sir Samuel Wilks called attention to the depression of temperature frequently noted in cerebral abscess. Subnormal temperature is not nearly so common as is supposed. It has been present in about one-half of the cases I have seen. Toward the end of the case the temperature may rise and the fever become linked with delirium. Surface elevation of temperature over the seat of the abscess is occasionally observed. A chill may occur, but seldom does. Anorexia and vomiting are present. Urinary chlorids are diminished and the phosphates are increased (Somerville). Certain symptoms are due to pressure: Headache begins (which at first is general, then local, and grows worse later in the case, and exists even in delirium; this fact distinguishes it from the headache of fever, which ceases in delirium); pulse is full, regular, and, in the absence of complications, becomes very slow; respiration tends to alterations of rhythm and the Cheyne-Stokes type; drowsiness lapses into stupor and stupor passes into coma; paralysis of the sphincters takes place; superficial reflexes gradually disappear on the side opposite to the lesion; convulsions are common; sensation is rarely impaired, and paralysis of the basal nerves may occur (third and sixth especially). The pupil on the same side as the abscess is sometimes dilated and fixed. Choked disk is not invariably found. It may be more marked on the same side as the abscess. It is more moderate in degree than in meningitis. Localizing symptoms, spasmodic and paralytic, depend upon the center which is irritated or destroyed. In abscess of the temporosphenoidal lobe hemiplegia of the opposite side is apt to develop. The face is most and first involved, next the arm, next the trunk, and finally the leg (Sir Victor Horsley, "Lancet," Jan. 27, 1912). In an abscess far posterior the motor palsy may be so slight as to almost escape recognition, but there is loss of the sense of position of a limb

and loss of power to localize touch. In cerebellar abscess there are vertigo, vomiting, occipital headache, rigidity of the postcervical muscles, and incoordination. Choked disk may be present or absent. A cerebral or a cerebellar abscess causes a decidedly high leukocytosis.

*Meningitis* arises soon after an accident; a *traumatic abscess* cannot arise until more than a week has elapsed after an accident, and many weeks may elapse. Meningitis presents high temperature and the general symptoms before outlined. *Mastoid disease* may occasion cerebral symptoms without abscess, or it may cause abscess. It is curious that in some cases of mastoid disease without brain abscess choked disk arises. In *sinus-thrombosis* there is septic temperature, the veins of the face and neck are enlarged, and a clot can usually be felt in the jugular. A *tumor* grows slowly, may present localizing symptoms, and double choked disk is frequently present. In tumor the temperature is apt to be normal.

**Treatment.**—If abscess is due to ear disease with implication of the mastoid cells, at once open and clear out the mastoid (see Fig. 535), and after doing this proceed to trephine the skull in order to reach the abscess. In any case, if symptoms of abscess exist, trephine the skull at once. If localizing symptoms are present, open over the suspected region. If localizing symptoms are not present, and the cause is ear disease, trephine at Barker's point (see Fig. 535). If no pus is found between the bone and dura, open the membrane. When the dura is opened, if the abscess is subdural, pus will be evacuated; if the abscess is in the brain substance, the brain will bulge very much and will not pulsate. A grooved director is plunged into the brain, in the direction of the abscess, for 2 or $2\frac{1}{2}$ inches. It is pointed to the external angular process of the opposite side. If pus is not found, withdraw the director and introduce it at another point, pointed to the nostril of the opposite side. If pus is not found, withdraw the director and introduce it again, pointing to the angle of the jaw of the opposite side. When pus is discovered, incise the brain with a knife, enlarge the opening by inserting a closed pair of forceps and withdrawing the instrument with the blades open. Scrape away the granulation tissue lining the abscess-cavity, irrigate with hot salt solution, and introduce a rubber drainage-tube and suture it to the scalp; stitch the dura, but leave an ample opening for the tube; bring the tube out through a buttonhole in the scalp, and after the first two days pull the tube out a little every day and cut off a piece. If the first trephining does not find pus, trephine at another point. If we are seeking for an abscess due to middle-ear disease and fail to find it in the temporosphenoidal lobe, seek for it in the cerebellum. In cerebellar abscess make a flap with the base up, and trephine or gouge away the bone just below the line of the lateral sinus. Puncture the brain for exploration as for cerebral abscess.

**Brain Disease from Suppurative Ear Disease.**—Acute otitis media sometimes, and chronic otitis media much more often, cause meningitis (*otitic meningitis*). This may be a circumscribed inflammation of the dura eventuating perhaps in an extradural abscess, a serous leptomeningitis, or a purulent leptomeningitis. There may or may not be involvement of the mastoid. There is always fever and pain and tenderness over the region of meningitis. The pain and tenderness are above the zygoma or back of the mastoid (Dench, in "New York Med. Jour.," August 27, 1910). With these symptoms there may or may not be signs of mastoid involvement. Dench points out that the occurrence of sudden and profound deafness during middle-ear suppuration usually means beginning meningitis (Ibid.). Chronic disease of the middle ear is apt to destroy the bone between the tympanum and the middle fossa of the skull, and thus produce meningitis, thrombosis of the petrosal or lateral sinuses, abscess of the temporosphenoidal lobe or of the cerebellum, or extra-

dural abscess. In chronic otitis media the reflexes of the opposite side of the body are frequently altered (Russell Reynolds, Sir Victor Horsley). In some cases, even without any other evidence of cerebral involvement, there is choking of the disk. In many cases the infection is direct, by bone involvement or by the lateral sinus. In many other cases the infection is through the labyrinth. If labyrinthine infection arises, it produces symptoms (disturbances of equilibrium and nystagmus). Chronic otitis media is apt to induce inflammation or suppuration of the mastoid cells (*empyema of the mastoid*). Pus in the mastoid may discharge itself into the middle ear, and from this point into the external auditory canal, through a perforation in the drum-membrane (especially in acute cases). In some cases the pus becomes blocked up within the mastoid process. Pus in the mastoid may after a time break into the cavity of the cranium or into the groove for lateral sinus, or may find its way externally and open into the sheaths of muscles arising from the mastoid. It not unusually opens into the sheath of the digastric muscle (*Bezold's abscess*). These facts teach the surgeon that inflammation of the middle ear should never be neglected, but should, if possible, receive the closest attention of the specialist. If no perforation exists in the drum, the surgeon must make one. In ordinary cases cleanliness and antisepsis are sufficient, the ear being syringed every day with a warm 2 per cent. solution of common salt. If only a small drum-perforation exists, 10 drops of pure alcohol or of corrosive sublimate solution (1:5000) are dropped into the ear daily; but if a large drum-perforation exists, boric acid and iodoform (7 to 1) are insufflated. Never inject alum. A strong silver solution is not safe; if it is used, wash the ear out afterward with warm salt water. If granulations or polypi exist, they must be removed. Some cases require the removal of the drum-membrane and the ossicles of the ear. Some cases of mastoid necrosis are due to tuberculosis. If headache, vomiting, and mastoid tenderness exist, open the mastoid (see page 830) in order to prevent abscess of the brain. In acute otitis media it is very rarely necessary to open the mastoid. The middle ear is on a lower level than the antrum of the mastoid, and in most acute cases both the middle ear and mastoid cells drain safely through a drum-perforation. Because a man has chronic otitis media it is by no means always necessary to trephine the mastoid. In many cases removal of the ossicles and drum-membrane effects a cure. In chronic otitis media, even if the mastoid is trephined, the ossicles and membrane ought to be removed in most cases.

**Cerebral abscess from ear disease** (see page 804) is almost always in the temporosphenoidal lobe, but may arise in the cerebellum. The **symptoms** are a regular, full, slow pulse (except in complicated cases), a transient rise of temperature, followed in many cases by a normal or subnormal temperature; vomiting; mastoid, frontal, and temporal pain. The mind is dull, and stupor arises which passes into coma; the bowels are constipated; choked disk may be present; and convulsions or spasms or paralyses may exist. Trephine and clean out the mastoid, and asepticize (see Operations Upon the Skull and Brain). Also trephine at Barker's point, 1¼ inches behind and the same distance above the middle of the external auditory meatus, open the dura, and seek for pus in the brain. If pus is not found, open the cerebellum.

**Extradural Abscess.**—The eye-symptoms and pain are the same in this as in cerebral or subdural abscess, but the temperature is different, rising to 103° or 104° F., and never being subnormal. There is often considerable tenderness above and behind the mastoid. In extradural abscess following disease of the middle ear trephine and clear out the mastoid; follow up a bone-sinus to the abscess, rongeur away the bone, being careful to avoid injuring the lateral sinus; curet, irrigate, and drain.

**Infective Sinus-thrombosis.**—Any sinus may be attacked. The dis-

ease may result from scarlet fever, small-pox, diphtheria, influenza, typhoid, or any acute suppuration. In erysipelas of the scalp, subaponeurotic abscess of the scalp, and cranial osteomyelitis septic clots may form in the veins which pass through the bone and reach the longitudinal sinus. Infective thrombosis of the superior longitudinal sinus is thus produced.

In carbuncle of the lip and orbital suppuration the cavernous sinus may become involved.

In caries of the basilar portion of the occipital bone the circular sinus or the cavernous sinus may suffer. In caries of the petrous portion of the temporal bone, and in suppuration of the middle ear and mastoid process, infective thrombosis of the lateral sinus may occur.

In any case the symptoms are those of pyemia. The lateral sinus is the one most frequently attacked. In infective thrombosis of the lateral sinus there is usually a history of an old discharge from the ear.

*Infective thrombosis of the lateral sinus* may result from a specific fever, but is usually due to chronic suppuration of the middle ear associated in most cases with carious bone and pus in the mastoid process. Thrombosis of the lateral sinus occasionally follows an operation upon a suppurating mastoid, or develops in an individual who suffers from middle-ear disease, who has been struck upon the head, who has had the ear syringed with force, or who has had injected a corrosive or very irritant fluid. Tuberculous bone disease is an occasional cause.

**Symptoms.**—In most cases there is a history of chronic ear disease. In children the symptoms are more acute than in adults. In any case the symptoms may rapidly become violent. In some cases there are preliminary symptoms of extradural abscess, pus being lodged in the groove of the sinus. It has been pointed out that pus in the jugular foramen may make pressure upon the pneumogastric, spinal accessory, and glossopharyngeal nerves, producing aphonia, hoarseness, dyspnea, dysphagia, and slow pulse (George F. Cott[1]). Marked headache and often an initial chill usher in sinus-thrombosis. The pain is apt to be localized about the ear and mastoid process, but may become general. There is usually acute tenderness of the mastoid. There is high fever from the start, but when the clot begins to soften and break down, hard rigors develop and the temperature fluctuates violently. The temperature varies greatly each day, fluctuating it may be between subnormal and 106° to 107° F. A chill may occur once or even twice a day, and it lasts from ten to twenty minutes. The pulse is soft and usually rapid. The patient is nauseated, labors under vertigo, is very restless, is sometimes delirious, may become dull and stupid, and the muscles of the neck are stiff. Tenderness and marked edema are detected over the mastoid, and the veins of the neck and mastoid region may be enlarged. When the clot extends into the jugular vein there is pain on moving the head and on swallowing, the cervical glands are swollen, and a cord-like clot may be felt in the neck. Choked disk exists in about half of all cases. There is often a profuse discharge of pus from the ear, but in some cases a discharge is found to have abated or ceased. Exophthalmos and swelling of the eyelids point to involvement of the cavernous sinus in the process. In early cases there is thrombosis of the lateral sinus alone, or of the lateral sinus and jugular vein. In advanced cases other sinuses become involved (superior petrosal, inferior petrosal, both cavernous, the lateral sinus of the opposite side, the ophthalmic veins, and the torcular Herophili). In sinus-thrombosis there is leukocytosis unless the patient is profoundly septic. A patient with sinus-thrombosis is in great danger of developing pulmonary metastasis and septic meningitis (Jansen). Septic meningitis is accompanied by abscess about the sinus. Infective sinus-thrombosis is a very fatal disease

[1] "Am. Med.," April 19, 1902.

and usually runs its course in from seven to ten days, but occasionally lasts for several weeks. It is a form of pyemia, and death arises from the causes which have been referred to in discussing that disease.

*Infective thrombosis of the cavernous sinus* occurs when an infected clot comes from another sinus, from disease of the nasal sinuses, from orbital infection, or from a pyogenic process of the face or lids. It causes pain and the general symptoms of pyemia, and also edema of the lids, chemosis, and an extreme degree of exophthalmos. Choked disk exists. Vision may be normal or impaired. The condition almost invariably spreads to the other eye along the circular sinus.

*Infective thrombosis of the petrosal sinus* produces pyemic symptoms, but no characteristic signs.

The **prognosis** largely depends upon early recognition. The surgeon should, whenever it is possible, open a mastoid before sinus-thrombosis arises, and should evacuate an abscess about the sinus before a clot forms in the venous channel, or at least before that clot becomes septic (Jansen).

**Treatment.**—In 1880 Zaufal proposed the operation now practised, and Horsely did it in 1886. (See article by Geo. F. Cott, in "American Medicine," April 19, 1902.) Infective thrombosis of the lateral sinus is treated as follows: Open and clear out the mastoid, and expose the sinus by the use of the chisel or rongeur (see Fig. 535). Follow Mr. Ballance's advice and expose the sinus from the bulb to the torcular. The jugular vein should now be exposed at the level of the cricoid cartilage and ligated below any clot which may exist. This is done to prevent propagation of an infected clot and diffusion of sepsis. Even if a clot does not exist in the jugular, the vein should be tied in two places and divided, because the sinus contains infected clot or putrid material even when the vein as yet does not. According to Ballance, the portion of the vein above the point at which it was divided should be extirpated. Some surgeons after ligating the jugular do not excise it, but if it contains or comes to contain a septic clot, incise the vein up to the base of the skull and pack the wound. After attacking the vein open the sinus, and if a clot is found to exist, cut away the wall of the sinus. Introduce a small spoon into the lumen and carry it toward the torcular Herophili, and scrape away the clot until blood flows. Arrest hemorrhage by forcing a piece of iodoform gauze into the wound and toward the torcular. Jansen opposes removing the entire clot toward the jugular, and does not tie the jugular, believing that to do so increases the danger of thrombosis of the inferior petrosal and cavernous sinuses. He simply removes the soft clot, but does not disturb the solid clot toward the heart. Most surgeons differ from him. Surgeons are of the opinion that it is futile to do any operation if pulmonary metastasis has taken place. In a case of the author's in the Jefferson Medical College Hospital the patient recovered after operation in spite of the fact that endocarditis had developed.

Until recently it was thought that the lateral sinus was the only sinus which should be attacked surgically, but in one case Knapp, of New York, requested Hartley to remove from the cavernous sinus a clot which was causing blindness and was due to sarcoma. The operation was successfully executed by Hartley, the incision being the same as is employed to reach a Gasserian ganglion in the Hartley operation. This patient lived several months. Dwight operated upon another case by incision of the sinus (E. W. Dwight and H. H. Germain, "Boston Med. and Surg. Jour.," May 1, 1902). Some surgeons advise removal of the eyeball and curettement of the sinus.

**Postoperative Insanity.**—Various mental disturbances may follow surgical operation (delirium, obsessions, hysterical excitement, morbid fears, illusions, hallucinations, amnesia, confusion, hypochondria, psychasthenia, melancholy, and genuine insanity). Insanity is no more frequent after abdominal

operations than after other operations if we exclude operations involving the ovaries. Removal of the testicles and ovaries are peculiarly provocative.

Postoperative insanity is most common in females and in adults. The predisposing elements connected with a surgical operation are: Apprehension, fear, pain, insomnia, and exhaustion before operation. The effects of the anesthetic, shock, and hemorrhage. Postoperative pain, insomnia, worry, and perhaps homesickness. There is always predisposition, hereditary or acquired. The patient may have been on the brink of an outbreak when the operation was performed. The insanity may be apparent as the patient awakes from the anesthetic sleep (Savage), and in these very rare cases the anesthetic is blamed. Most acute insanities come on in from three to five days after operation.

If a man has ever had an attack of insanity operation exposes him to distinct danger of another attack. No special mental condition characterizes postoperative insanity. There may be mania, melancholia, stupor, delusional insanity, or acute confusion. Acute confusional insanity is the most usual form. In some cases sepsis is present, in others it is not. We must not mistake insanity for febrile delirium, delirium tremens, delirium from opium deprivation, delirium from taking morphin or cocain, delirium of iodoform-poisoning, delirium of the senile, traumatic delirium, hysterical delirium, uremic coma, or bromism.

Many cases of postoperative insanity must be sent to a hospital for the insane. Only cases of brief duration can be properly cared for in a general hospital.

**Intracranial Tumors.**—An encephalic tumor may originate within the skull. It may have arisen from an external growth invading the cranial cavity, or may be metastatic. A tumor that arises within the cranium may take origin from the periosteum, from one of the membranes of the brain, from the vessels, from the neuroglia, or from the brain substance.

No region of the body is so liable to tumors as the brain. During the course of a number of years the autopsies of the Munich Pathological Institute are stated by Bollinger to have shown 1 tumor of the brain in every 85 autopsies. Hale White's experience is that such tumors are even more common than this, and he estimates them at 1 in every 59 autopsies.

In endeavoring to determine the causes of intracranial tumors we must accredit heredity with considerable influence in tuberculoma, and possibly with some force in sarcoma and carcinoma. Tumors of the brain are decidedly more common in males than in females, probably because of the greater male liability to injury, syphilis, and alcoholism.

The majority of cases of tumor of the brain occur between the ages of twenty-five and fifty. Children are particularly prone to suffer from glioma and from tuberculous growths. In aged persons a tumor of the brain very rarely develops. In 100 cases of brain-tumor collected by Hale White only 2 were aged seventy or over. In 100 cases collected by Mills and Lloyd only 1 was over seventy.

Injury may be responsible for the development of sarcoma, of fibroma, and possibly of other forms; in fact, a syphiloma may arise in a syphilitic person at the seat of an injury.

We use the terms "intracranial" or "encephalic tumor" not only to include true neoplasms, but also to designate growths of parasitic, syphilitic, or tuberculous origin. It is of importance to attempt to make a diagnosis as to the form of tumor that is present, and this may be possible on account of the fact that in many cases the form affects the symptoms. A useful classification of these growths has been made by Knapp, and is as follows: (1) The infective granulomata, including tuberculous growths, gummata, and actino-

mycotic areas; (2) connective-tissue growths; (3) epithelial growths; (4) aneurysms. The most common of all these tumors is undoubtedly that due to tubercle. In fact, Gowers estimates that, if we exclude syphiloma, tubercle is responsible for one-half of the cases, and glioma and sarcoma together for one-third.

*Tuberculous Tumors* (*Tuberculous Gummata; Tuberculomata*).—Tuberculous growths may be primary, but are usually secondary to tuberculosis in some other region of the body. Tuberculous tumors are the most common form met with. They are at least four times as common in children as in adults. They may be single, especially in adults, but are often multiple, especially in children; and multiple growths may be very widespread. According to Allan Starr, these growths are most common in the cerebral axis (especially in the basal ganglia), next in the cerebellum, next in the cerebral cortex, and are least common in the centrum ovale. A tuberculous tumor usually arises in the pia mater, particularly in an arterial distribution, but may begin in a ventricle, or even in the brain substance. Some of these growths are distinctly subcortical. The tubercle bacilli responsible for the condition are carried by the blood. Some of these growths are small aggregations of miliary tubercles in thickened pia. Others are large masses. A large tuberculous tumor is due to the coalescence of many foci. It undergoes caseation in the center, and is surrounded by a zone of softened or sclerotic brain substance. Tuberculous meningitis is present in two-thirds or three-fourths of the cases of tuberculoma.

*Gummatous Tumors* (*Syphilomata*).—As gumma seldom arises from inherited syphilis, the condition is very rare in children. There may be a single gumma, but, far more often, syphilitic growths are multiple. A gumma may be round or may be irregular in outline; in fact, the outline is frequently blurred and indistinct. Some of these growths are soft, and some, which contain a quantity of connective tissue, are hard. A syphiloma usually arises from the membranes, and, hence, is generally on the surface of the brain; and the membranes in the region of the growth usually show distinct inflammation. Soft gummata are most common at the base; hard gummata, in the cortex of the cerebrum or cerebellum.

*Actinomycosis*.—This is a very rare condition, in which the mass may remain solid like a tumor, but is far more apt to break down into an actinomycotic abscess.

*Sarcomata*.—Injury seems to play a considerable part in the production of intracranial sarcoma. Any variety of sarcoma may arise. It may be primary or secondary. As a rule, at least in the beginning, the growth is single; but it may be multiple or may become so. The majority of sarcomata arise from the membranes or from the periosteum, but some cases take origin from beneath the cortex. Early in their progress these growths may be encapsulated, but some of them, from the very start, are infiltrating; and even those that were at first encapsulated later infiltrate. The cortex and the cerebellum are the most common seats for sarcoma. *Endothelioma* is sometimes met with. What is called *angioma of the brain* is, in reality, *angiosarcoma*. A *psammoma* is usually sarcomatous.

*Gliomata*.—A glioma is a growth which is often so ill defined and so slightly differentiated in appearance from the brain substance that it may easily be overlooked in an exploratory operation. It arises much more frequently from the white than from the gray matter, and develops from the neuroglia of the cerebrum, of the cerebellum, of the pons, or of the medulla oblongata. A glioma may be soft or may be hard; and soft gliomata are probably, in reality, sarcomata (gliosarcomata). Hemorrhage is very apt to occur in these growths. They have a tendency to become cystic. They destroy tissue as they grow, hence pressure signs are late or absent.

*Fibromata.*—Intracranial fibroma is a rare growth. Tumors of the cerebellopontile angle are apt to be of this character. It is of firm consistence, is encapsulated, and may grow to a large size. Such growths can be readily enucleated. Injury seems occasionally to be responsible for their formation.

*Osteomata.*—Osteophytic growths not uncommonly take origin from the inner surface of the skull, but the osteomata arising in the dura or in the brain substance are rare. Such growths, however, occasionally occur.

*Cholesteatomata.*—These tumors are fibrous growths covered with endothelium and containing layers of cholesterin. They are particularly apt to arise in the pia mater, but may begin in either of the other membranes or in the brain substance. A cholesteatoma is commonly called a *pearl tumor.*

*Enchondromata* and *true neuromata* are rare, and *lipomata* are exceedingly uncommon.

*Adenomata.*—An adenoma occasionally springs from the conarium or the pituitary body.

*Carcinomata.*—Primary intracerebral carcinoma is rare, but does occur. Secondary carcinoma is more common, and may follow cancer of any part of the body, although it is most apt to follow cancerous growths about the face or neck. A primary growth may begin in the meninges or in the lining of the ventricle. Intracerebral carcinomata may be single or multiple. They are soft and non-encapsulated growths, infiltrating and very vascular.

*Cysts.*—Mills says that cysts arise about an old hemorrhage, are small retention-cysts of a vascular plexus, or are porencephalic. Dermoid cysts are extremely rare. Hydatid cysts are very rare in the United States. A cyst may result from the degeneration of a glioma or a sarcoma.

**Symptoms.**—They are divided into two sets: (1) *General*, due to increase of pressure, and (2) *local, localizing*, or *special*, arising because of the part of the brain involved.

In some cases general symptoms are absent, in some special symptoms are absent, in others even tumors of large size produce no recognizable symptoms, either general or special. A large infiltrating growth, a glioma, for instance, may produce no symptoms at all if situated in a silent region, and if it destroys brain substance as it grows, so that intracerebral pressure is not increased.

*General Symptoms.*—The chief general symptoms are headache, vomiting, and choked disc. All of these may be present, any one of them may be absent, any two of them may be absent, and in some cases all of them are absent. Other general symptoms that may or may not be present are vertigo, general convulsions, insomnia, mental failure, and somnolence or partial stupor.

*Headache.*—This is the symptom most commonly present. It occurs sooner or later in a very great majority of cases.

At first it may be noted only at certain times of the day and it is usually complained of most on rising in the morning. The headache of brain tumor when once established is intense in most cases, and as a general thing it is practically continuous, with episodes of increased violence. In rare cases it is paroxysmal. In some cases it is trivial, in some few cases it never arises at all. The headache of brain tumor usually interferes with sleep. It may be general, one sided, frontal, or occipital. The situation of the pain is without localizing value unless there is tenderness on percussion or pressure over the seat of pain, which is sometimes noted in growths of the meninges or cortex.

Headache is less common in children. In very young children the explanation of this is found in the open fontanels and the expansile cranium. In older children in the fact that gliomata are common in children and gliomata may not cause intracerebral pressure.

The dura is sensitive, as Cushing says, to "pull or pressure, not to an incision." The headache of brain tumor is due to stretching of the dura.

*Vomiting* is present at times in many of the cases. It may happen occasionally or it may never occur.

It is usually cerebral, that is, vomiting with a clean tongue without nausea, and without any relation to the taking of food. In some cases it is projectile, quantities of vomitus being suddenly projected from the mouth. It may, however, be associated with nausea, and in some cases there is nausea without vomiting. It is apt to be most severe on getting up, especially on rising in the morning. It is usually most severe when headache is most intense.

The worst attacks of vomiting occur in cases of cerebellar tumor.

The cause of vomiting is uncertain. Some believe it to be due to stimulation of the vagus center; others, to reflex stimulation of a vomiting center in the medulla. If associated with vertigo it may arise from the auditory centers.

*Choked Disk* (*Optic Neuritis, Papillitis, Descending Neuritis, Papilloedema*).—This is a most important symptom. When present it is, as Cushing says, one of the "most reliable" signs of tumor. It is present in 80 per cent. of all cases. It may be noted in a few weeks after a tumor begins to grow, in a few months, or longer. It may come on rapidly or gradually.

It is particularly common and severe in growths beneath the tentorium. It is decidedly less common in tumors of the motor cortex. It is nearly always bilateral, but it is common to find it more marked in one eye than in the other. That it is worse on one side *suggests*, but *only suggests*, that the tumor may be on the side on which the choked disk is worst. Monocular choked disk is very rare. When it exists it indicates that the growth, in all likelihood, is situated near the back of the orbit on the same side as the choking of the disk. Gowers recorded a case of unilateral choked disk due to a tumor compressing the left optic nerve ("Lancet," July 10, 1909).

Choked disk may exist for some time and attain a high grade without noticeably impairing vision, but ultimately it leads to retinal hemorrhage, white atrophy, and blindness. When atrophy begins, vision wanes. The presence of choked disk does not *prove* the existence of tumor. The cerebral edema of Bright's disease may cause a condition known as albuminuric retinitis, which practically is not to be distinguished from the choked disk caused by tumor. Cushing believes that the processes are identical in tumor and Bright's disease. Choked disk may occur in meningitis, brain abscess, cerebral syphilis, sinus-thrombosis, myelitis, infectious diseases (typhoid, influenza, diphtheria, and other conditions), toxemias from lead, arsenic, and alcohol, anemia, diabetes, or as an hereditary or family disease.

The cause of choked disk has been much disputed. Some believe that the condition is a neuritis due to a toxic condition of the cerebrospinal fluid. Others believe that it is not inflammatory, but is a papillo-edema due purely to mechanical pressure, and is an edema or dropsy. The latter theory is largely held by surgeons because they have become convinced by experience that relief of intracranial pressure relieves or cures choked disk. De Schweinitz and Holloway ("Therapeutic Gazette," July 15, 1909) make the following statement in regard to mechanical pressure causing choked disk: "That this is the *only* etiologic factor may with propriety be disputed; indeed, it seems certain that a combination of factors must be active in the production of this condition, but increased intracranial pressure is the one of which we have most certain knowledge."

Cushing, of Harvard, who has done such notable work on this subject, believes that "almost all, if not all, cases of choked disk are primarily of mechanical origin and do not justify the term 'neuritis'" ("Keen's Surgery," vol. iii). The presence of choked disk is disclosed by the ophthalmoscope.

*Convulsions.*—Generalized convulsions may occur, and children are especially liable to them. Many cases of supposed general convulsions have an unobserved local beginning which is a focal symptom.

*Vertigo.*—Mills says it is noted in one-third of all cases and is due to dural, ocular, or labyrinthine irritation.

*Insomnia* is often due to headache and is apt to be associated with restlessness, lack of emotional control, and irritability. It is most pronounced in cases of syphiloma and some forms of malignant disease, and is worse in adults than in children.

In rare cases there is *somnolence* or partial stupor rather than insomnia. In such cases slow speech is often noted.

*Mental Failure.*—There is often great lack of emotional control, characterized by irritability and outbreaks of anger. Failure of memory is common and change of character is the rule. There may be great slowness of thought and of mental response to stimuli, with defective power of orientation. Progressive mental deterioration may occur. There may be mental depression, apathy, or mental excitement. Tumors in certain regions may cause delusions, illusions, or hallucinations.

Hysteria and neurasthenia sometimes arise.

*Pulse and Respiration.*—Whereas the pulse is often slow, it is very variable, and I agree with Cushing that "pressure-symptoms which characterize acute lesions (namely, rise in blood-pressure; slow, vagus pulse, and Cheyne-Stokes respiration) are conspicuous by their absence" ("Keen's Surgery," vol. iii).

*Special Focal or Localizing Symptoms.*—These symptoms, when present, indicate the situation of the growth. If the tumor is in a silent region there will be no focal symptoms. General symptoms may exist without focal symptoms and focal symptoms may exist without general symptoms.

Among localizing symptoms we should mention various forms of aphasia, hemianopsia, paralysis, Jacksonian epilepsy, sensory disturbances (to touch, pain, or temperature), sensory aura, disturbances of taste and smell, impairment of muscular sense, alteration or impairment of reflexes, nystagmus, and incoördination.

*Exophthalmos* means direct pressure upon the cavernous sinus. A unilateral exophthalmos is nearly always upon the side of the tumor. In bilateral exophthalmos the protrusion is usually worst on the side of the lesion (Weisenberg, in "Jour. Amer. Med. Assoc.," vol. lv).

**Diagnosis.**—In many cases a diagnosis is made at a period so late that irreparable damage has already been inflicted. Early diagnosis is of the first importance. A careful and painstaking study of the patient and of his history will usually enable us to make a diagnosis before, and often long before, the attainment of a degree of pressure which causes papillo-edema, headache, and vomiting. In doubtful cases an exploratory operation should be performed. Exploration is always called for in advancing cerebral palsy and in focal epilepsy.

In *abscess* the symptoms usually develop much more acutely than in tumor. It is true that some cases of abscess last for months, but in them intervals of complete remission of symptoms occur, which is not the case in tumor. An abscess follows middle-ear disease or some other pyogenic process or perhaps a head injury. There may be fever and leukocytosis. Choked disk is far less common than in tumor.

*Chronic meningitis* from syphilis exhibits periods of increase and periods of subsidence of the symptoms, and usually palsy of one or more cranial nerves. The symptoms may pass away under the use of mercury and iodid of potassium. There may be clinical evidences of syphilis. There should be a positive Wassermann reaction.

*Chronic tuberculous meningitis* causes a headache which is apt to be general and more violent than that of tumor, and there is more commonly cutaneous hyperesthesia and hyperesthesia of the organs of sight and hearing. Optic neuritis may be absent. When present it is of less intensity than that observed in tumor. In some cases the ophthalmoscope discloses tubercles on the choroid. In meningitis there is a continued, irregular fever. The cerebrospinal fluid obtained by lumbar puncture usually exhibits an excess of lymphocytes. It may contain the bacilli. Carbohydrate is absent. It may be sterile. Of course, a tumor might exist with meningitis.

In *Bright's disease* symptoms strongly suggestive of tumor may arise (headache, vomiting, and choked disk) with, perhaps, but little change in the urine. The difficulty in diagnosis becomes evident when we recall that in cases

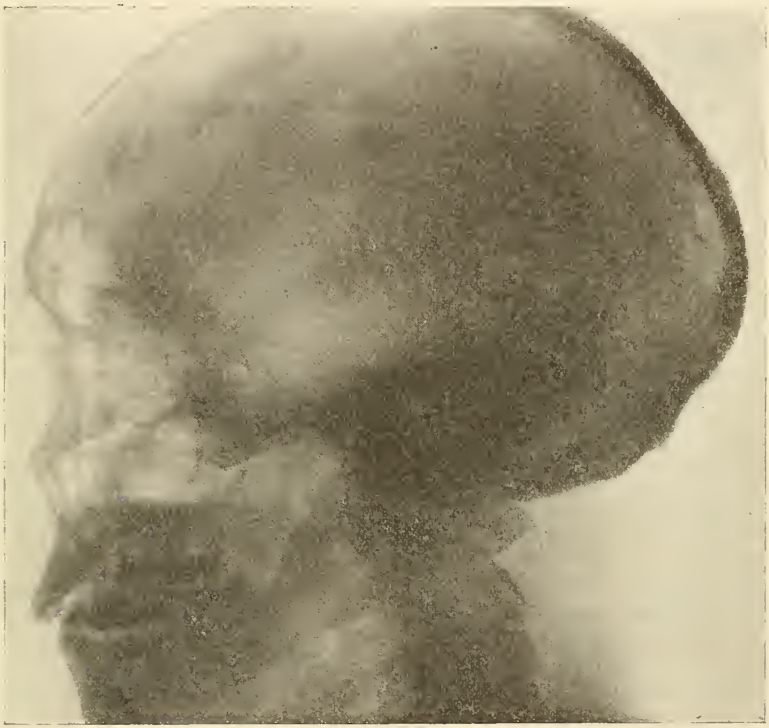


Fig. 524.—Professor Gibbon's case of brain tumor.

of brain tumor the urine may contain casts and albumin. A sudden onset and brief duration of the symptoms suggest uremia.

*Ependymitis* with ventricular dropsy may cause symptoms resembling tumor with hydrocephalus. Optic neuritis is not so common as in tumor. Bilateral spastic paralysis may arise.

*Severe anemia* is capable of producing symptoms which suggest tumor. It can even produce choked disk. A blood examination and the rapid improvement under proper treatment makes the diagnosis evident.

In some cases of brain tumor (especially frontal lobe tumors) there are striking evidences of hysteria and neurasthenia. Always think of this when inclined to make a diagnosis of hysteria or neurasthenia.

The *x*-rays may aid us in diagnosis and many brain tumors can be skiagraphed (Fig. 524). Lumbar puncture may aid in the diagnosis, but it must be

used with the greatest care, because in tumor the withdrawal of any considerable quantity of fluid may be followed by sudden death due to the brain stem sinking into the foramen magnum.

**Cause, Duration, and Termination.**—A brain tumor, unless caused by syphilis, is a certainly fatal lesion if not removed by operation.

Some brain tumors grow for years before they produce symptoms. Some grow with great rapidity. When pressure symptoms appear the patient's life will terminate within a few months unless an operation is performed. It is usually stated that the average duration of life is three years.

Sudden death may occur at any time.

Horsley states that in a very few cases brain tumors have disappeared after mere operative exposure, the tumor not having been removed. Cushing has had the same experience, and regards such an amazing disappearance as due to the cystic degeneration of a glioma.

**Localization of a Tumor.**—The situation of a tumor is determined not only by localizing symptoms, but also from their mode of onset and manner of combination. In some cases the symptoms are not characteristic, in others they are definite. The more marked the signs of compression, the less the value of localizing symptoms. The nature of the tumor, its depth, and whether it is single, and if other tumors exist is, if possible, determined. Localizing symptoms may be due to irritation or destruction of functionating power. Irritation causes spasm, and destruction induces paralysis. Convulsions which are local or which begin locally are known as *Jacksonian epilepsy*. A local convulsion points to an irritative lesion of, or immediately adjacent to, the center which presides over the muscular movements of the part convulsed. Local paralysis points to a destructive lesion of the center which presides over the movements of the paralyzed part. In some cases a center is damaged and the muscular movements it controls are paralyzed, but the adjacent brain-areas are irritated and the muscles they represent are attacked with spasms. In some cases an apparently paralyzed part becomes convulsed, the center not being completely destroyed and sudden hyperemia serving to awaken spasm. Always note the order of invasion of different regions and observe if spasm is followed by muscular weakness or anesthesia.

**Lesions in the Cortical Motor Area.**—A slow-growing tumor which irritates the cortex will cause tonic or clonic convulsions on the opposite side of the body. These convulsions have a local beginning (Jacksonian epilepsy). After a time paralysis may develop (monoplegias and perhaps, ultimately, hemiplegia). An irritative lesion of the lower third of this area causes spasm of the opposite side of the face, angle of mouth, or tongue; and this condition is often associated with tingling. The spasm may remain limited or may extend widely, and may even become general. An irritative lesion of the middle third of the cortical area causes spasm, which is limited to or begins in the fingers, thumb, wrist, or shoulder. An irritative lesion of the upper third of the cortical motor area causes spasm, which is limited to or begins in the toes, ankle, leg, or thigh. If such lesions exist, an aura is occasionally felt in the affected region before the spasm begins, and there is often numbness after the spasm. Destructive lesions of the motor area cause local paralysis, which may be preceded by local spasm of the same parts, and is often associated with local spasm of other parts. If paralysis comes on unpreceded by convulsions the lesion is *subcortical* rather than cortical, that is, it is in the white matter between the motor area and the internal capsule.

**Tumors of the anterosuperior portion of the prefrontal region** give no definite localizing symptoms, but produce general symptoms. Mental disorders may arise in tumors of any area of the brain, but in tumors of the prefrontal region they are most apt to occur. The intelligence is nearly always

impaired, and there is apt to be mental apathy, loss of memory, hysteria, irascibility, and pronounced change of character. As the tumor grows it may subsequently involve the motor region, which in all probability lies entirely in front of the fissure of Rolando.

In tumors of the prefrontal region there may be focal convulsions or local palsy on the opposite side, due either to a spread of irritation from a superficial tumor to the motor cortex or to involvement by a deep tumor of the commissural fibers which join the frontal lobe and the motor cortex.

**Tumors of the Antero-inferior Portion of the Prefrontal Region.**—In a right-handed man tumor of the second left frontal convolution causes *agraphia*, and of the third left frontal convolution, *motor aphasia*. In a left-handed person these localizations are on the right side of the brain.

**Tumors of the parietal lobe** may occupy a silent region of this lobe. The centers for general sensibility and for the muscular sense are back of the fissure of Rolando in the parietal lobes. Hence a tumor in this region may cause disturbance of muscular sense and general sensibility in the limbs without spasm or palsy (Durante). There may be word-blindness when the left angular gyrus is affected.

The extension forward of a parietal tumor will involve the motor zone and produce spasm or palsy. Extension backward will involve the occipital lobe and produce hemianopsia.

**Tumors of the occipital lobe** are apt to produce *lateral homonymous hemianopsia* (blindness of the nasal half of one retina and the temporal half of the other retina. If the right side of the brain contains the lesion the right side of each retina is blind, and vice versâ). Wernicke's pupillary sign is absent. By this sign we mean loss of pupillary light reflex when light is cast upon the blind part of the retina. This sign only occurs in lesion in or in front of the centers for pupillary reflex. Lesions of the cuneus and of the calcarine fissure are especially apt to produce hemianopsia. A lesion of the occipital cortex may produce it. Tumors of both occipital lobes cause blindness. In tumor of an occipital lobe color vision only may be affected (Cushing). Tumor of the optic radiation may produce hemianopsia (that is, growths of the chiasm, optic nerve, or pulvinar may, but it is not certain that a growth of the quadrigeminal bodies or of the external geniculate body can).

In a tumor of the occipital lobe (the left lobe in right-handed persons and vice versâ) there may be *mind-blindness*, that is, an inability to know what is seen. He may fail to understand written or printed words (*word-blindness*), the nature of things (*apraxia*), or signs (*asemia*).

**Tumors of the temporosphenoidal lobe** frequently produce no symptoms. In the temporal lobes the cortical centers for hearing are placed, and each center is connected with both auditory nerves, but the crossed auditory bundle is larger and more active than the direct (Francesco Durante, "Brit. Med. Jour.," Dec. 13, 1902). Tumors in the left lobe are particularly apt to cause deafness and may cause *word-deafness*. Tumors at the apex of the lobe may cause perversion or impairment of taste and smell. Tumors of the temporosphenoidal lobe may make pressure on the motor tract and cause paralysis of the opposite side.

**Tumors of the Corpus Callosum.**—These growths affect mentality as do tumors of the frontal lobes and frequently cause hemiplegia of each side.

**Tumors of the Crus.**—These tumors cause hemiplegia of the opposite side and paralysis of the third nerve on the side of the lesion. If the optic tract is pressed upon there will be hemianopsia. If the tegmentum is involved there will be hemianesthesia.

**Tumors In or About the Optic Thalamus.**—On the side of the lesion there are nystagmus and impairment in the movements of the eyeball. There

is muscular weakness and impairment of sensation in the limbs of the opposite side. There may be sudden and uncontrollable movements, astereognosis, mental dulness, emotional outbreaks, and impairment of emotional control of the facial muscles. A lesion of the posterior part of the thalamus causes hemianopsia and the pupillary reaction of Wernicke.

**Tumors** of any size in or about the **corpus striatum** cause hemiplegia of the opposite side by pressure upon the internal capsule. Pressure upon the *optic thalamus* produces homonymous hemianopsia and hemianesthesia. Growths near the basal ganglia produce intense choking of the disk and early pressure because of distention of the ventricles.

**Tumors of the Corpora Quadrigemina.**—These tumors produce cerebellar ataxia, palsy of the eye muscles, and sometimes, perhaps, hemianopsia. Hearing may be impaired in the ear opposite to the lesion. Tumors of the corpora quadrigemina are apt to involve the crura, and later the third nerve. Ocular symptoms are always present (loss of pupillary reflex and nystagmus). If the third nerve is involved there are paralysis of the motor oculi area on the side of the lesion (external strabismus, dilated pupil, and drop-lid) and hemiplegia of the opposite side of the body from pressure upon the crus. This condition is a form of *crossed paralysis*.

**Tumors of the Pons.**—Pontine lesions produce symptoms by pressure upon the particular nerves which come from this region, with or without the evidences of pressure upon the motor path. Forms of crossed paralysis may exist. Lesions in the lower half of the pons may affect the fifth, sixth, and seventh nerves on the side of the lesion and the limbs on the opposite side. The auditory nerve may be involved in the lesion. In crossed paralysis the face on the side of the limb paralyzed is usually not affected, but in extensive tumors it may be paralyzed. *Conjugate deviation* of the eyes may occur *away* from the *facial paralysis*. In tumors of the upper part of the pons the pupils may be first contracted because of irritation of the third nerve nuclei, and later dilated by destruction of these nuclei. Anesthesia as a result of pontine tumors is not nearly so common as motor paralysis, and convulsions are rare. There may be hemiplegia with crossed hemianesthesia. A tumor on the side of the pons which involves the peduncles causes ataxia and sudden, uncontrollable movements. Knee-jerk may be absent in a case of pontine tumor.

**Tumors of the Medulla.**—An extensive lesion inevitably causes death. Cranial nerves only may be involved, but crossed paralysis may take place. There may be hemiplegia with crossed paralysis of the hypoglossal nerve. Vomiting is common, retraction of the head is not unusual; respiratory and circulatory disturbances and dysphagia are usually noted; sometimes there is numbness and occasionally there are convulsions; usually there is incoordination because of pressure upon the cerebellum.

**Tumors of the Cerebellum.**—They often cause internal hydrocephalus. In general it may be said that tumors of the cerebellum cause headache, vomiting, vertigo, choked disk, and early blindness. *Tumors of the middle peduncle* cause sudden uncontrollable movements of the trunk, either toward the side of the tumor or away from it. Vertigo and nystagmus are common. Symptoms are frequently complicated by evidences of pontine disease proper.

*Tumors of the middle lobe of the cerebellum* cause a sense of lost equilibrium and obvious unsteadiness in attempting to walk or even to stand. The knee-jerks vary. The Babinski reflex is absent in an uncomplicated case. The patient has a tendency to fall backward; there are giddiness and vomiting, early and violent choked disk and occipital headache, nystagmus, inability to stand steady when the feet are together and the eyes shut, and perhaps temporary palsies of the eye muscles.

*Tumors of the cerebellar hemisphere* produce no localizing symptoms. The usual unsteadiness of gait is due to pressure upon the middle lobe (Nothnagel).

**Tumors of the Cerebellopontile Angle.**—Tumors of this region are usually attached to the acoustic nerve, sometimes to the fifth nerve. They produce general pressure-symptoms and may cause cerebellar or pontine symptoms by making direct pressure on those parts. If the tumor involves the acoustic nerve there will be tinnitus, objective vertigo (sometimes with, sometimes without, forced movements), a liability to sudden attacks of falling to the ground, failure of hearing or actual deafness, perhaps sudden blindness or sudden unconsciousness, and sometimes tonic extensor spasms. If the tumor involves the trigeminal nerve there will be violent neuralgia in the course of the nerve.

**Tumors of the Hypophysis Cerebri.**—The pituitary body may be subjected to pressure from, or become involved in, tumors of the region about it or tumors or cysts may spring from it directly. Tumors of or tumors making pressure upon the hypophysis may in some cases cause acromegaly, and in others impotence, genital infantilism, a tendency to fat deposit and stunted stature, and amenorrhea in women. There is intense headache, amblyopia due to primary atrophy (no preceding choked disk), appearing often as a bitemporal hemianopsia (Cushing, "Jour. Amer. Med. Assoc.," July 24, 1909). In some cases extreme drowsiness has been noted, and in some cases there has been binasal hemianopsia. When there is stimulation of the pituitary (hyperpituitarism) acromegaly develops; when there is lessened secretion (hypopituitarism) there are obesity and genital atrophy.

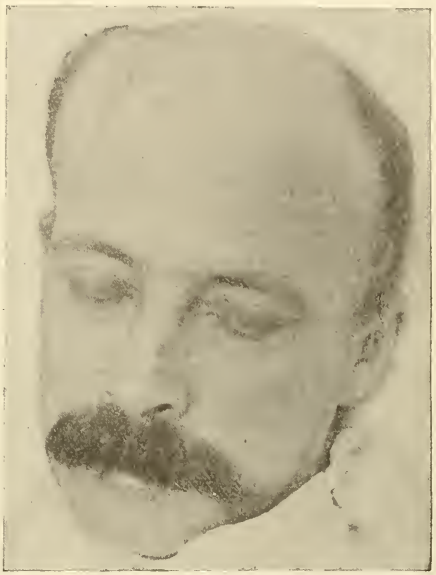


Fig. 525.—Case of cerebellar tumor. Bulging of flap after osteoplastic exploratory operation.

Any brain tumor which causes internal hydrocephalus may thus cause pressure on the hypophysis and lead to the development of hypophyseal symptoms. (For full information regarding the hypophysis see the classic monograph by Harvey Cushing, called "The Pituitary Body and Its Disorders").[1]

**Treatment.**—If any doubt exists as to the existence of brain syphilis, and if the Wassermann reaction is positive, give the patient a dose of salvarsan (intravenous) and a course of iodid of potassium. Give the iodid at first in small amounts, but rapidly increase it until heroic doses are taken (100 or more grains a day). Mercury should also be given hypodermatically or by inunction. If salvarsan, iodid of potassium, and mercury really relieve the symptoms, and if the improvement is not merely temporary, operation is unnecessary, although it may be demanded later in order to remove an irritant scar. If antisyphilitic treatment fails, the question of operation must be considered. The test of success is improvement in the choked disk. If this improves, the

[1] For full consideration of localizing symptoms see particularly the writings of Gowers, Mills, Allan Starr, Potts, Lloyd, Burr, Dana, Dercum, Osler, and Cushing, which have been freely used in the above section.

treatment is succeeding; if it does not, the treatment is a failure. It should not be persisted in over six weeks if there is no improvement in the eye-grounds. To delay operation further may mean blindness. We must always bear in mind that in certain cases of glioma the symptoms *temporarily* improve under antisyphilitic treatment. The term *operable case* does not of necessity mean a tumor which can be entirely removed by operation. Some tumors which can be only partially removed should be operated upon. An operable case is one in which an attempt may be made to remove the tumor and in which the tumor can be entirely removed or in which a part can be removed, the removal of this part promising relief. We are justified in being radical, because without operation a brain tumor is a certainly fatal malady. In many cases of undoubted tumor excision for cure is not attempted because of the absence of localizing symptoms or because of the inaccessible situation of the growth. In all cases operation is, first of all, exploratory. Tumors of the dura which have not infiltrated the brain, many cortical and some subcortical growths, are operable. Cerebral and cerebellar cysts may be opened and drained in hope that benefit will result. Tumors of the lateral lobe of the cerebellum and tumors of the cerebellopontile angle have been removed. Byrom Bramwell maintains that tumors at the base, tumors of the pons and medulla, of the corpus callosum, of the basal ganglia, and of the deeper parts of the centrum ovale are irremovable. Surgeons now regard some tumors of the cerebellopontine angle as operable, but agree with Bramwell's views as to growths in the other situations he mentions. Frazier has concluded that "if the tumor is found to be very vascular and of the infiltrating type, it is very questionable . . . as to whether any attempt whatsoever should be made to extirpate" ("University of Penna. Med. Bull.," April–May, 1906), and with this opinion I certainly agree. In tumors which are very extensive complete removal is usually out of the question. There is no use in removing secondary malignant tumors. It often happens that the brain itself (as in syphilis) is so extensively diseased, or that other organs (as in tuberculosis) are so involved, as to render attempts at removal of the tumor futile or actual removal useless. Mills thinks that 50 per cent. of cerebellar tumors can be attacked surgically ("New York and Phila. Med. Jour.," Feb. 11–18, 1905). He classifies operable tumors of the cerebellum as follows: 1. Tumors situated entirely or chiefly in the lateral lobe. 2. Tumors upon or even invading a part of the vermis or middle lobe. 3. Tumors of the cerebello-oblongatopontile angle. The most favorable tumors for removal are fibromata and encapsuled sarcomata. Gliomata and gliosarcomata have been removed, but are very apt to return. A syphiloma requires removal as truly as a real tumor. The cases are commonly unfavorable, as there is often widespread brain disease. The same is true of tuberculoma. Among inoperable tumors are most gliomata and all infiltrating sarcomata, metastatic tumors, and multiple tumors. Bramwell[1] tells us that he has studied 82 cases of intracranial tumor, and he considers that in only 5 of them could the tumor have been entirely removed. In 157 reported cases the tumor was either not found or not removed; in 104 reported cases the tumor was found, and in some of them it was removed (Ransohoff, in "Jour. Amer. Med. Assoc.," Oct. 11, 1902). The conclusion is that though some tumors of the brain may be successfully removed, extirpation is feasible in only a small minority of cases and is to be decided on only after careful study of all the indications and contra-indications offered by the case. When about to operate, apply an apparatus to the arm and take the blood-pressure just before the operation and at frequent intervals during it. Thus by noting a great fall in blood-pressure the surgeon gets early warning of dangerous shock, learns when to hasten, and if the operation should be temporarily abandoned and be completed at

[1] "Edinburgh Med. Jour.," June, 1894.

another time (**two-stage operation**). We may be driven to abandon operation after cutting the bone and dural flaps, and if we are forced to stop, we restore the bone and dura to position, and complete the operation after a day or two. I agree with Frazier that the lessening of hemorrhage by temporarily clamping the common carotids in the neck is not free from danger, and it is not proper to do more than apply Crile's clamp to the vessel on the side operated upon. In a brain tumor when the dura is first opened there is usually at once marked bulging of the brain, which is called *initial bulging;* after working for a time on a brain, even when there is no tumor, bulging occurs from traumatic edema, which is called *consecutive bulging.* That consecutive bulging may occur is a sound reason for operating rapidly (Frazier). The mortality from tumor operations is large, death being due to shock and hemorrhage. Haas collected 122 cases in which the tumor was removed; the mortality was 60 per cent. Operations completed at one séance give a larger mortality than two-stage operations. During the operation an erect posture causes the brain to recede and permits of extensive exploration under the dura (Ransohoff-Cushing). The same thing is accomplished by lumbar puncture (Cushing). The fibromata constitute the best cases for operation. In operating on a cerebral tumor make a large osteoplastic flap. If on opening the dura the tumor is not visible, and if the localizing symptoms were reasonably positive, the surgeon is justified in making an exploratory incision through the cortex to see if there is a subcortical growth. Operations for cerebellar tumors are peculiarly difficult because of the large blood sinuses, because of the limited space obtained to work through, because of the great bulging after the dura has been opened, because of the impossibility of reaching the anterior, mesial, or upper surfaces through the incision, because of the liability to injure the pons and medulla, and because of the difficulty of retracting the parts (Frazier, "New York and Philadelphia Med. Jour.," Feb. 11–18, 1905). In approaching tumors which are not within a cerebellar hemisphere by a one-sided exposure of the cerebellum, it may be best to remove a considerable portion of the hemisphere in order to obtain free access to the growth. I prefer Cushing's "cross-bow" incision and bilateral exposure (see Fig. 537). This enables the surgeon to dislocate the sound lobe outward and so obtain room to work upon the lobe containing the tumor. The diagnosis of cerebellar tumor is usually doubtful, hence practically all operations are at first exploratory and are then made palliative or radical as the case demands. Operation must be early because cerebellar growths quickly cause blindness. Sir Victor Horsley, McArthur, Hochenegg, von Eiselsberg, Cushing, Frazier, and others have operated for tumor of the hypophysis. Removal of a healthy pituitary body from dogs is sure to be fatal, as the gland is necessary to the life of the dog. Removal of a healthy pituitary in man would in all probability prove fatal. The entire anterior lobe should not be removed. If the tumor is associated with acromegaly, removal of the tumor may arrest the acromegaly. One route of approach to the hypophysis is from the side, just as we reach the Gasserian ganglion (Carton, Paul, Horsley). Another route is by way of osteoplastic frontal resection, the longitudinal sinus being ligated and the frontal lobes lifted (Krause, Hartley, Borchardt). Another method involves osteoplastic resection of the anterior wall of the frontal sinus and nose. This is called the transphenoidal route. The transphenoidal route has been variously modified. Cushing's plan of approach through a sublabial incision is described on page 834. Though thorough extirpation of a brain tumor is feasible in only a minority of cases, operation should often be performed for palliative purposes when the tumor cannot be located, when it is in a region from which it cannot be removed, or when its nature forbids removal. Grainger Stewart, Annandale, Horsley, Macewen, Cushing, and Keen have advocated

*palliative trephining* in certain cases. Simple trephining is of little value. In order to really relieve pressure the dura must be opened and left unsutured, so that hernia cerebri may follow. Cushing has had some cases of extraordinary improvement in cases of cerebral tumor after trephining in the right temporal region and removing a piece of the dura. The operation is called by Cushing a *decompression operation.* The brain bulges through the dural opening, but the dense temporal fascia stitched together over it prevents fungation. It is the temporosphenoidal lobe that bulges, and the right side is selected because word-deafness might ensue if the operation were done on the left side. I have seen several of Cushing's cases. One of them, a colored man, had been almost blind for some time, and was unconscious and had rapidly failing respiration when the operation was performed. He was so much benefited that he returned to work and has useful vision and no pain. I have had several very gratifying results in my own practice. A decompression may, in rare cases, cause a glioma to degenerate and pass away. Horsley saw such a case. In cases of cerebral tumor subtemporal decompression, and in cases of cerebellar tumor suboccipital decompression, may be performed. Sir Victor Horsley believes that the opening for decompression should be near the tumor and not always in the subtemporal region. He believes an opening near the tumor possesses the advantage of aiding us later if the tumor should become operable. He maintains that subtemporal decompression may later confuse the localizing symptoms should such symptoms develop.

Decompression may relieve choked disk and thus retard or prevent optic atrophy and blindness. Cushing demonstrates that intracerebral pressure is the chief element in choked disk.

This procedure is of value in diminishing excessive intracranial pressure, and thus relieving headache and choked disk, and decreasing the tendency to sudden death from inhibition of the heart or from respiratory failure. The usual method of decompression will not relieve the headache caused by tumor of the hypophysis. This headache is due to distention of the dural box in which the gland is placed, and can be relieved only by incision of the dural box (Harvey Cushing, "Jour. Amer. Med. Assoc.," July 24, 1909).

We conclude that in most cases of brain tumor operation should be performed for exploration; in some cases extirpation may be performed; in most cases extirpation is impossible, and the surgeon must be content with the palliative influence of Cushing's decompression operation. A tumor of the brain if not cured by antisyphilitic treatment is of necessity fatal if unoperated upon, and exploratory operation is not very dangerous.

In a case of brain tumor if operation is refused, if extirpation is impossible, or if decompression fails, it may be necessary to use the bromids for convulsions and morphin for headache. The headache is often benefited by purgatives, courses of potassium iodid, and the ice-bag to the head.

**Operative Treatment of Epilepsy.**—The shock of an accident or a cerebral concussion may establish epilepsy, especially in those predisposed by heredity or other causes. Traumatic epilepsy, Le Dentu[1] tells us, may be due to: (1) Bone-fragments from skull fracture; (2) outgrowths of bone due to tumor; (3) cicatrices of meninges resulting from laceration of membranes by bone-fragments; (4) chronic meningitis which ends in sclerosis of membranes; (5) cysts resulting from intracranial hemorrhage at the point of fracture; (6) arteriovenous aneurysm. We would add: (7) tumors of the brain; (8) sclerosis of the cortex. We refer here, in speaking of traumatic epilepsy, purely to the condition when it follows a head injury, and this is the common meaning of the term. Remember that epilepsy, as shown by Sachs, may follow a long-forgotten injury. Before undertaking a brain opera-

[1] "La Presse médicale," June 9, 1894.

tion for epilepsy it is a sound rule to remove all sources of definite peripheral irritation. I have seen apparent cure follow the removal of a tender cicatrix and follow circumcision of a patient laboring under phimosis. Briggs reported a case of epilepsy in which there was a distinct depression of a portion of the skull. There was also necrosis of the tibia, and after the cure of the necrosis the convulsions ceased. The removal of supposed peripheral irritation, however, is only occasionally beneficial. Are operations upon the skull and brain curative? Surgeons are much less enthusiastic than they were a few years ago. I believe operation can cure less than 5 per cent. of cases, but it is important to remember that in some cases in which operation seems to have failed medical treatment becomes much more efficient than it was before the operation. The high rate of cure (70 per cent.) once claimed for operations was due to failing to follow the patient sufficiently long. A patient should not be reported as cured until at least three years or, better, five years have passed without any evidence of the disease. Another source of error was a failure to understand that any traumatism may improve epilepsy *for a time.* "The administering of an anesthetic, the shock of an injury, the traumatism of an operation, just like a febrile seizure, may interrupt an epileptic habit and cause a patient to go for weeks or months without an attack" (the author, in "Medicine," Feb., 1904).

Operation must never be indiscriminately applied. In some cases it gives hope of relief, in others it is obvious that it would be utterly futile. In order to determine if a case is or is not suitable for operation it must be studied with great care. The history must be carefully obtained, particularly as to hereditary predisposition, the first convulsion, and its *supposed* cause. The question of injury, recent or old, should be thoroughly investigated, and it is a sound rule to have the head shaved and then examine for a scar and for a depression. Convulsive seizures must be studied by an expert, hence the patient should be in a hospital, constantly watched by a trained nurse, until one or two fits have occurred. The nurse watches the convulsion and describes it in writing, noting particularly if it had a local beginning. The general health must be investigated. An *x*-ray should be taken.

I am accustomed, for surgical purposes, to make the following classification of epilepsy. It is a modification of Sir Victor Horsley's classification (the author, in "Medicine," Feb., 1904):

1. Reflex epilepsy, the surgical treatment of which I shall not discuss in detail.

2. The common non-traumatic, idiopathic, or essential epilepsy, in which the attacks are general and are without a local onset.

3. Idiopathic epilepsy with a local onset of attacks (focal or Jacksonian epilepsy).

4. Traumatic epilepsy. This may be subdivided into two forms: (*a*) attacks without a local onset, and (*b*) attacks with a local onset (focal or Jacksonian epilepsy).

5. Jacksonian epilepsy due to gross brain disease (tumor, aneurysm, etc.).

6. Epilepsy following infantile cerebral palsy.

7. The posthemiplegic epilepsy of adults.

1. **Reflex Epilepsy.**—Remove the supposed cause of irritation. When epilepsy follows traumatism and a scar is found on the scalp, excise the scar. This is an imperative duty if the scar is tender or the seat of an aura.

2. **Essential or Idiopathic Epilepsy.**—Operation upon the brain is useless. If persistent headache exists, it may be proper to trephine and open the dura for exploration. Such an operation is done to relieve headache. Some claim remarkable results from bilateral excision of the cervical ganglia of the

sympathetic (see page 761). The operation is a theoretical one and of doubtful utility. It was founded upon a misconception as to the cause of epilepsy, and favorable reports are no more favorable than have been set forth regarding various other now abandoned procedures.

3. **Idiopathic Epilepsy with Local Onset of Attacks** (*Focal or Jacksonian Epilepsy*).—Many of these cases begin in young children who have had infantile palsy, the traces of the palsy having disappeared. In such cases the convulsions may begin on one side, and, in fact, may be nearly limited to one side. If, from the very beginning, the attacks began in one group of muscles or in one extremity, whether or not they spread to the rest of the body, and if the case is seen within two years of the first attack, the surgeon is justified in exposing the brain and excising the irritated portion of cortex. This operation, it is true, cures very few cases, but it benefits many for a considerable time and seems to make them more amenable to medical treatment. In the vast majority of cases fits recur, but rarely as severely as before. After fits have been going on for two years operation offers no prospect of cure, as the association fibers have surely degenerated. But, even in very old cases, if the attacks are frequently repeated and thus threaten life, the excited center should be removed to save life.

In cortical excision more of the cortex than the excited center is of necessity removed, because, in order to get the entire center, we must go wide of it. Paralysis of the parts controlled by the extirpated cortical area follows. The paralysis is seldom permanent except to the finer movements. The operation gives the best prognosis in young persons, and when done early in the case. The return of fits after apparent cure is thought to be due, at least in some cases, to the formation of *adhesions* between the brain and its membranes. Various unsatisfactory attempts have been made to prevent adhesion by the insertion of silver-foil, gold-foil, rubber tissue, egg-shell membrane, and Cargile membrane. In operating for cortical epilepsy a large osteoplastic flap is required. In the previous remarks we dealt with partial epilsepsy and with generalized epilepsy in which, from the first, the attacks had a local beginning. If cases of apparent idiopathic epilepsy develop Jacksonian attacks (attacks with a local beginning), it is useless to excise the cortex. The entire cortex is diseased, though one region is particularly unstable.

4. **Traumatic Epilepsy.**—Always remember that a traumatism to a person who becomes epileptic may have been only a coincidence; the condition may be essential epilepsy and the traumatism may have had nothing to do with it. Epilepsy ensuing upon traumatism may not begin until months or even several years after the injury. In the earliest attacks consciousness may or may not be lost. The causative injury may have been slight or severe. "An injury may cause a hemorrhage or a depressed fracture; may be followed by a scar upon the membranes; may occasionally lead to the development of an innocent or malignant tumor or a cyst, or may merely induce some trivial change in the subtle chemistry of the nerve-cells" (the author, in "Medicine," Feb., 1904). Injury may produce general epilepsy or Jacksonian epilepsy. If an identified traumatism exists, the surgeon should operate even after years. When the traumatism has not left definite evidence, the surgeon is justified in making an exploration any time up to the termination of the third year after the accident. The earlier the operation, the better the prognosis. The best prognosis of any form of epilepsy is given by Jacksonian epilepsy of traumatic origin.

"In focal epilepsy with evidences of skull injury or depression, trephining is imperative and somewhat promising. The dura should invariably be opened, even if it seems in good condition. A dural scar should be extirpated.

The brain should be examined by sight and by touch, and should be explored with the little finger and with the dural separator to well beyond the limits of the opening in the dura. If a tumor is found, it should be removed; if a scar upon the brain exists, it should be extirpated; if a cyst is discovered, it should be drained; and if there is any obviously damaged area in the brain tissue, it should be unhesitatingly cleared away. If nothing obvious is found on exploration, and if the attacks have been distinctly local in origin, it is justifiable to extirpate the motor center from which the discharge seems to originate.

"When Jacksonian epilsepy has followed an injury in the motor region, the chances of effecting a cure are much better than they are when the epilepsy has followed an injury in the sensory region. When it has followed an injury in the frontal region, operation affords very little hope of cure.

"When the condition is not focal but essential epilepsy, the surgeon will remove a scalp scar; and if there is any evidence of bone injury, he will trephine the bone, open the dura, and explore the brain. It is needless to say, however, that in such a case he will not extirpate any of the cortex.

"In cases of focal epilepsy I use the osteoplastic method of operating. In cases of generalized epilepsy I use the simple trephine and leave the button of bone out, as a means of effecting a prolonged modification in the intracerebral pressure" (the author, in "Medicine," Feb., 1904).

Bramwell maintains that when traumatism is followed by epilepsy and the epileptic discharge starts from a cortical center which is not beneath the scar, the surgeon should trephine first at the seat of injury, and if this fails, he should trephine over the excited center.

5. **Jacksonian Epilepsy Due to Gross Brain Disease.**—The treatment of this condition is the treatment of the brain disease.

6. **Epilepsy Following Infantile Cerebral Palsy.**—In this group of cases the palsy is manifest. It is justifiable to operate upon a child, but not later in life. The prospect of benefit is poor even in a child.

7. **The Posthemiplegic Epilepsy of Adults.**—Operation is useless.

Our conclusions are that these operations sometimes seem to cure epilepsy, but so, occasionally, does any operation. White[1] records 90 trephinings in which, though no cause was found for the epilepsy, great relief followed, and 2 cases were apparently cured; he mentions benefit or apparent cure following tracheotomy, ligation of the carotid artery, incision of the scalp, etc. The same effect may be obtained by a great shock, high fever, the administration of an anesthetic, or an accident. The fact seems to be that any operation, by means of nervous shock, may interrupt the epileptic habit; but in ordinary operations the fits tend after a time to recur and soon reach their old standard of frequency. In the special brain operations with removal of obvious lesions or extirpation of discharging centers the fits usually recur, but they will rarely reach the old standard of frequency, and will be more amenable to medical treatment.

In non-traumatic chronic epilepsy without localizing symptoms trephining is not justifiable unless persistent headache calls for it as a means of relief from intracranial pressure. Annandale advised us to consider experimental operation in such cases when the drug-treatment has failed and when the patient's condition seems hopeless. He says there is no chance of improvement without operation, and operation may possibly disclose a removable lesion.[2] After trephining for epilepsy five years should elapse without a convulsion before cure is resaonably assured; and if convulsions arise, they must at once be met by medical treatment. A man having once had a convulsion may at any time have others; hence he should always be watched. It is not

[1] "The Supposed Curative Effects of Operations *per se*," "Annals of Surgery," Aug. and Sept., 1891.

[2] "Edinburgh Med. Jour.," April, 1894.

unusual for a few convulsions to occur soon after an operation for epilepsy, and then to cease for a considerable time. These early fits result from habit (*habit fits*). Among the operative procedures suggested for the treatment of epilepsy may be mentioned circumcision, clitoridectomy, ocular tenotomy, ligation of the vertebral arteries, removal of the cervical ganglia of the sympathetic (see page 761) (Alexander, Jonnesco, Jaboulay), and the actual cautery to the head (Féré).

**Operative Treatment of Insanity.**—(See the author, in "Journal of Nervous and Mental Diseases," June, 1904.)

1. **Epileptic Insanity.**—The conditions which call for operation on a non-insane epileptic (see page 821) call for it on an insane epileptic. It is sometimes justifiable to operate if there has been a head injury, and operation may lessen the number and diminish the violence of the attacks. If focal seizures exist, we may proceed as for focal seizures in the sane. In status epilepticus we may operate to relieve pressure. It will be observed that operation is for the convulsions and not for the insanity.

2. **Paresis.**—I do not advocate operation in paresis. If we believe in traumatic paresis, we may be inclined to advise operation. Personally I do not believe that genuine paresis is ever cured; the lesions of the disease are widely disseminated; the pons, medulla, and even the cord may be diseased and the lesions cannot be removed.

3. **Non-traumatic Insanity and Paranoia.**—Operation cannot cure the insanity and is not to be advised.

4. **Hypochondriacal Delusions.**—Operation is useless. Some practice it with the idea of getting rid of a delusion by removing a part to which the attention is directed. Such attempts always fail, because it is the insanity which causes the delusion, not the delusion which causes the insanity.

5. **Operations for Traumatic Insanity.**—A psychosis constructed on the basis of a traumatic neurosis never calls for operation. The only cases in which operation is ever justifiable are those in which traumatism is the direct cause. Insanity may begin at once or soon after an injury, but is often unrecognized for weeks or even months. Nearly all of these cases are predisposed to insanity and the injury has been only an exciting cause. Traumatism is the direct cause in about 2 per cent. of cases of insanity.

"An antecedent injury may have directly induced the alienation; it may have had no bearing at all upon the latter; or it may have produced an insanity by fear and shock, and not by creating a direct brain lesion. Again, the head injury, by increasing the individual's susceptibility to alcohol and to the effects of the sun, may, if this person drinks alcohol or exposes himself to the rays of the sun, be indirectly responsible for lunacy.

"In insanity following an injury to the head there may be various supposed causative lesions: A fracture of the skull, with or without depression; the development of an exostosis; sclerosis or softening of the cortex; edema of the membranes or of the brain itself; cerebral hyperemia or congestion; thickening of the membranes; adhesion of the membranes to the skull, to each other, or to the brain; new growth; inflammation of the membranes; or minute, slowly developing, widespread nutritive changes. The injury may be assumed to be the cause of the insanity if the insane condition becomes manifest almost at once or soon after the accident; but if the symptoms do not appear until long after the accident the traumatism may be considered to be the directly exciting cause in some cases, and not in others. It may be blamed if, between the time of the accident and the appearance of the insanity, there has been a marked change in the patient's disposition, temperament, or character; if he has developed headache, insomnia, irritability, passionate outbreaks of temper, moodiness, or lapses of memory; if he has plunged into

immorality or excesses in alcohol; if he has displayed a tendency to neglect business or family obligations, and if he has shown increased susceptibility to alcohol and to the sun. Sometimes epilepsy may develop during this period, (Richardson, 'American Journal of Insanity,' July, 1903. The author's 'Address on Surgery,' delivered before the meeting of the Medical Society of the State of Pennsylvania, May 18, 1897). If there was none of these intermediate changes in the normal mode of thinking and way of acting, one cannot count the traumatism as causative. Many persons that have received severe head injuries have shown these changes, but have never gone insane. I have been studying this point for a number of years, and have decided that quite a few patients that have been trephined for fracture or for meningeal hemorrhage have subsequently shown pronounced and permanent changes in character and disposition. Of the number that show such changes, many never go insane, but some do. Such an insanity is distinctly traumatic in origin" (the author, in "Journal of Nervous and Mental Diseases," June, 1904). The prognosis is very unfavorable; some recover sanity after operation, many do not. Some recover sanity without operation. Sometimes operation cures by removing a lesion; sometimes by shock, etc. Some cures following operation did not result from the operation.

On what cases should we operate?

We should operate on cases "in which insanity has soon followed a head injury. If the site of the trauma is indicated by a scar, a depression of bone, local tenderness, fixed headache, or some localizing symptom—motor or sensory—operation should positively be undertaken. In a case in which the insanity has developed later, in which the intermediate period between the injury and the development of the insanity has shown the change from the normal mode of thinking and way of acting previously alluded to, and in which the site of trauma is indicated by any of the evidences mentioned above—operation should positively be performed. One should not operate upon a case simply because there is a dubious record of an antecedent fall or blow, which merely suggests the possibility of a traumatic origin for the insanity. In any case in which there are positive signs of increased pressure it may be considered proper to trephine as a palliative measure" (the author, Ibid.).

**Abdominal, Gynecological, and Genito-urinary Operations.**—If an insane person has a disease which is dangerous to life or which is productive of pain, discomfort, or ill health, he or she is entitled to be cured, if possible, by a surgical operation. The removal of pain and other depressing influences may result in great improvement in the general health and in notable mental improvement. The operation may thus indirectly exercise a beneficial influence on the insanity, but the influence is not direct and it is never justifiable to do such an operation as oöphorectomy upon an insane woman unless the condition of the ovaries would call for it in one not insane.

**Operations On the Skull and Brain.**—As a preliminary it is well to note that urotropin (hexamethylenamin) given by the mouth quickly appears in the cerebrospinal fluid, and that the fluid contains the maximum amount in from one-half hour to an hour after ingestion. The presence of this drug decidedly inhibits the growth of bacteria in the fluid. This is Crowe's discovery. We believe it wise to give this drug in all cases in which meningitis is threatened or exists (S. J. Crowe, in "Johns Hopkins Hospital Bulletin," April, 1909). It should be given for twenty-four hours preceding and for several days after an operation upon the brain or spinal cord. From 40 to 60 gr. a day are given by mouth. Cushing has subjected this drug to the severest test in his operations to reach the pituitary body. He gave 60 gr. a day of this drug "on the day preceding and for some days after the operation" ("The

Pituitary Body and Its Disorders," by Harvey Cushing). After 33 transsphenoidal operations there were 2 cases of meningitis. One of these cases (which was inaugurated by violent sneezing) was fatal.

**Trephining for a Fracture of the Skull.**—The patient should be anesthetized unless he is unconscious, and should be placed upon the back with the shoulders a little raised. A sand-pillow is placed under the neck, and his head is turned away from the side to be operated upon. The position of the surgeon is such that the patient's head is a little to his left. A large semilunar incision is made with the base down, which incision goes through the periosteum, and the flap is lifted. The bleeding vessels of the flap are caught by forceps. The fracture is sought for and found. The pin of the trephine is projected beyond the crown and is set upon sound bone, the crown overhanging the line or edge of the fracture. The surgeon tries to avoid the region of a sinus or large artery. A gutter is cut in the bone, the pin of the instrument is withdrawn, and the trephining is completed. In going through the diploë bleeding is copious. The inner table feels very dense. Stop from time to time, clean out the gutter in the bone with gauze, and try the bone with an elevator to see if it is loose. When the fragment is loose enough, pry it out. If the surgeon desires to replace the button, hand it to an assistant, who places it at once in a bowl of warm normal salt solution, kept warm by standing in a basin of water at 105° F., or in warm carbolized towels. The edges of the opening should be rounded by a rongeur, and the bone, if depressed, must be elevated. Sometimes it may be necessary to remove splinters and fragments of bone. After removing the fragments the edges of the opening should be smoothed by the use of the rongeur forceps. The dura should be examined to see if injury exists, and hemorrhage must be stopped. Bleeding from the dura is arrested by passing a ligature of silk or catgut threaded in a small curved needle under the vessel on each side of the wound, and tying the ligatures (*suture-ligatures*). Bleeding from the pia is arrested by direct ligation, by suture ligature, or by gauze packing. Bleeding from the diploë is arrested by the use of Horsley's wax. The wound is cleansed, the edges of the dura are sutured by catgut or fine silk; in some

Fig. 526.—Galt's conical trephine.

Fig. 527.

Fig. 528.

Fig. 529.

Figs. 527–529.—Hudson's burrs.

cases the button of bone is reintroduced; in other cases some chips are cut from the bone and scattered upon the dura, but in most cases no attempt is made to fill up the gap in the bone. The scalp is sutured by silkworm-gut, and horse-hair or gauze drainage is employed for a day or two. Sterilized gauze dressings are put on, a rubber-dam is laid over them, and a gauze bandage wet with bichlorid of mercury is applied.

Instead of the trephine some surgeons use the chisel or gouge and hammer to remove a portion of the bone. Other operators, believing that this procedure may cause concussion, employ the surgical engine.

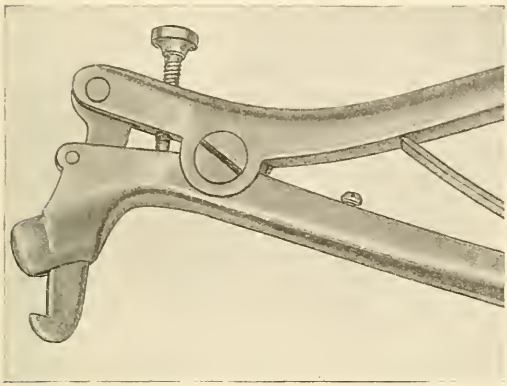


Fig. 530.—Hudson's modified DeVilbiss forceps.

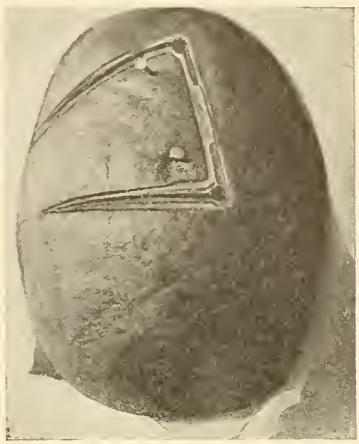


Fig. 531.—Combined osteoplastic operation. First step. Incision through superficial tissues and bone. Flap held in place on the bone by tacks.

I now seldom use the old trephine, preferring instead the instruments of Hudson (Figs. 527–530).

**Osteoplastic Resection of the Skull.**—Wolff suggested this operation, and in 1889 Wagner performed it. It is employed for the removal of tumors and the Gasserian ganglion, for focal epilepsy, and for exploration. It is the operation of choice when a large opening is needed, as when the operation is,

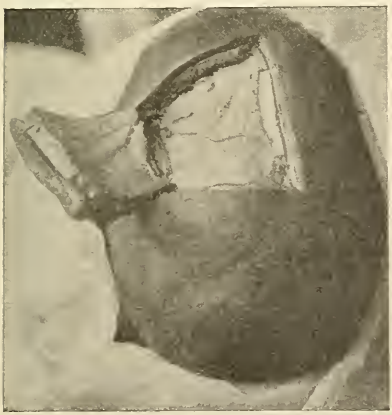


Fig. 532.—Combined osteoplastic operation. Second step. Bone flap turned down.

Fig. 533.—Combined osteoplastic operation. Third step. Showing exposure of brain by removal of dural flap.

first of all, for diagnosis. The incision shown in Fig. 531 is made through the scalp and periosteum, and the flap is tacked to the bone, ordinary long

tacks being used. Otherwise our manipulations may separate the flap from the bone (Fig. 531). A groove corresponding to this incision may be cut in the bone by special gouges or chisels. I do not use chisels. I am convinced that the blows of the mallet add to shock, may cause hemorrhage or add to existing hemorrhage, may extend a line of fracture or cause a fracture, may diffuse a purulent collection, or produce concussion of the brain. Some surgeons use the surgical engine. It is difficult to keep it sterile, it runs at too high a speed to be readily controlled, and it is troublesome to cut a bevel with it. The instrument is dangerous except when in very skilful and highly trained hands. Some surgeons make trephine openings and then cut from within outward by the Gigli wire saw (Obalinski). Cushing, of Harvard, does what is called the *combined* method. I prefer this to any other plan. It is rapid and free from all danger of wounding the dura. I make two or several openings with Hudson's burr. This excellent instrument divides bone with great rapidity, but does not divide the dura. In fact, one cannot divide the dura with it, for the burr binds as soon as it is through the bone. Figs. 527 to 529 show Hudson's burrs. The sides of this bone-flap are rapidly cut by Hudson's improvement of the DeVilbiss forceps (Fig. 530). The upper margin is cut on a bevel with the Gigli saw. Because of this bevel when the flap is restored to place the upper edge of the flap rests on a shelf of bone and does not press on the brain. By whatever method performed, three sides of the bone-flap are cut through, but the bone is left attached to the scalp. It is a good plan to save the scalp from detachment by temporarily nailing it in place (Fig. 531). The bone is then broken outward, the fracture taking place at the base of the bone-flap, the dura is opened a little distance from the edge (sufficient space being retained for sutures), and the exploration is made and the operation is performed (Figs. 532 and 533). When we are ready to suture the dura we note if the brain bulges greatly. If it does, manipulation will surely injure it, and we should cause the brain to recede before suturing by placing the patient nearly erect or by performing lumbar puncture. After suturing the dura the bone which is still adherent to the pericranium and scalp is restored to its proper place, and the scalp is sutured.

Besides restoring a flap of bone into position, or replacing a button of bone, or strewing the dura with bone-fragments, other methods of closing the opening have been practised—for instance, heteroplasty with a decalcified bone-plate, with a celluloid plate, or other foreign material.[1]

**Trephining the Frontal Sinus.**—This operation may be employed for inflammation of the lining membrane of the sinus or for empyema. Make a vertical incision in the middle of the forehead, starting $1\frac{1}{2}$ inches above the nasion and terminating at the root of the nose. The button of bone is removed and the opening is enlarged if necessary. The mucous membrane is incised, the opening into the nose is found and is dilated, and a drainage-tube is passed into the nose from the sinus, the upper end being left in the sinus. In some severe cases Jacobson advises us to curet the sinus, to disinfect it by the use of silver nitrate or chlorid of zinc, and to insufflate an "aseptic powder." In some cases resect the mucous membrane. I prefer an osteoplastic resection to trephining the frontal sinus.

**Trephining the Mastoid.**—(See Operation for Mastoid Suppuration, page 830.)

**Technic of Brain Operations.**—In focal epilepsy a faradic battery is required. Always shave the scalp and always antisepticize it. In localizations, mark out the fissure upon the scalp with an anilin pencil, with iodin, or with silver nitrate. Have the patient semirecumbent. Mark three points upon the bone with the

[1] See Bretano, in "Deutsche med. Woch.," May 17, 1894.

center-pin of the trephine before incising the scalp (both ends of the Rolandic fissure and the point at which the trephine is to be applied). Make a semilunar flap 3 inches in diameter, with the base below. Control bleeding in the flap by forceps pressure. If the operation is by trephining the 1½-inch trephine should be employed, but if a smaller trephine or the Hudson burr is used the opening must be enlarged with a rongeur. Before enlarging the opening separate the dura from the bone by a dural separator. In most cases an osteoplastic flap is preferable to trephining. It is always employed in explorations for tumor. As a rule, open the dura and examine the brain. The dura is lifted by mouse-toothed forceps and is opened by scissors along a line ¼ inch from the bone edge, a broad pedicle of dura being left uncut. Hemorrhage is arrested by pressure and hot water or by passing suture-ligatures of silk or catgut around any bleeding vessel by means of a curved needle. In some cases packing must be retained or forceps must be kept on. In packing, endeavor to use but one piece of gauze, so as to avoid leaving in a forgotten piece. Upon opening the dura cerebrospinal fluid flows out, the stream being increased at each expiration. Absence of pulsation of the brain points to abscess or tumor, and a livid color indicates subcortical growth. An old laceration is brownish. If the brain bulges through the opening, it means increased pressure (tumor, abscess, effusion into the ventricles, etc.). After opening the dura employ no antiseptics, especially when the surgeon intends using electricity to locate a center. Irrigate only by warm salt solution. In operating for tumor the dura is opened and in some cases the brain is incised. The tumor is turned out by the finger or, if this

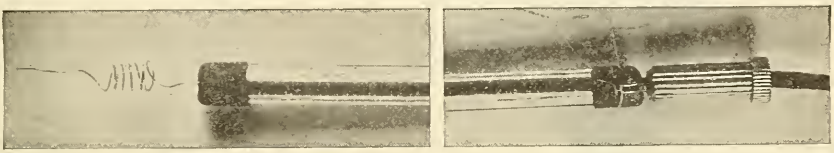


Fig. 534.—Showing terminal and connection of Cushing's electrode (¾ natural size). Instrument should be 16 inches or more in length.

is impossible, by the dry dissector, the scissors, the dull knife, or the sharp spoon. If the entire tumor cannot be removed, it is sometimes proper to take away as much as possible. The removal of a portion may retard the growth of the remainder, and the trephining, by lessening cerebral pressure, may relieve the symptoms and prolong life. After removing a tumor arrest distinct points of bleeding by ligatures or by suture-ligatures. Pack the tumor cavity with gauze and bring the end of the strand out of the wound. Stitch the dura with silk and suture the scalp with silkworm-gut. In electrifying the brain faradism is employed of a strength about sufficient to move the fibers of the exposed temporal muscle. The best electrode is that of Cushing (Fig. 534). It is a "glass unipolar electrode carrying a fine platinum wire core, coiled into a spiral at the end" (Cushing, in "Keen's Surgery," vol. iii). The other pole is attached to an extremity, "preferably on the homolateral side." During the electrical test the patient must not be deeply anesthetized. A careful observer watches the muscular movements. If, for instance, the surgeon wishes to remove the thumb center, he moves the electrode from point to point until he obtains thumb movements. The region is sliced away bit by bit until the center which is responsible for the conclusive movements is removed. It will be found impossible to remove only the thumb center. Adjacent centers are sure to be more or less damaged, and a certain amount of paralysis follows the operation. If we wish to *tap the ventricles*, Keen directs the trephine opening to be 1¼ inches behind the external

auditory meatus and the same distance above the base-line of Reid (Fig. 535, *a*). A grooved director or metal tube is passed into the brain in the direction of a point "$2\frac{1}{2}$ to 3 inches above the opposite meatus." The normal ventricle will be entered at a depth of 2 to $2\frac{1}{4}$ inches, but the dilated ventricle will be entered sooner. The moment of entry is marked by lessened resistance and a flow of cerebrospinal fluid. Drainage can be maintained by introducing a rubber tube. This operation has been employed in hydrocephalus. Kocher punctures the ventricle $2\frac{1}{2}$ cm. from the midline and 3 cm. in front of the fissure of Rolando. After an aseptic cerebral operation, as a rule, do not drain unless hemorrhage has been considerable. In many cases after trephining replace the bone, but not when the bone is diseased, is infected, is very compact, or if we desire to alter pressure.

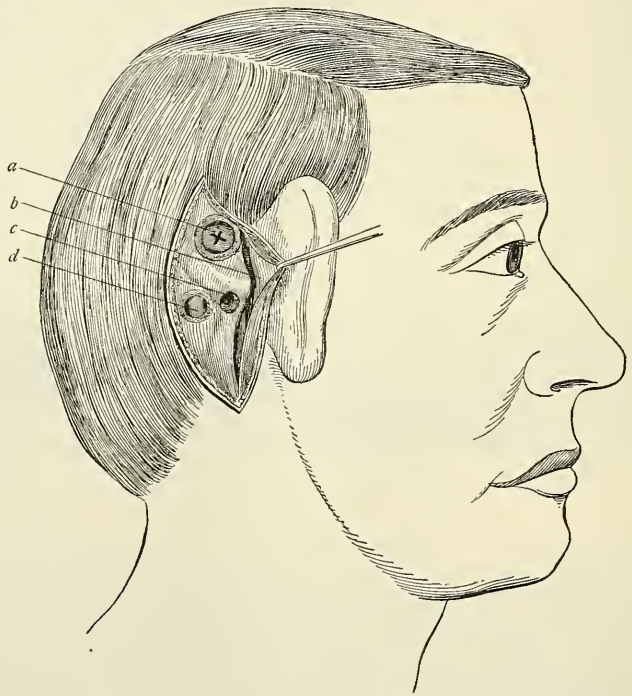



Fig. 535.—Opening the mastoid antrum and the lateral sinus; exposure of the temporosphenoidal lobe and puncture of the descending horn of the lateral ventricle: *a*, Temporosphenoidal lobe (descending cornu of lateral ventricle is 1 cm. deeper); *b*, inner surface of periosteum; *c*, mastoid antrum; *d*, lateral sinus (Kocher).

**Operation for Mastoid Suppuration.**—Place a sand-bag under the neck. An incision is made $\frac{1}{4}$ inch posterior to the auricle and down to the bone, and in the direction of the long axis of the mastoid. The bone is bared and examined, especially at a point in the line of the incision, which is on a level with the roof of the meatus (Fig. 535, *c*). The bone will usually be found softened. Gouge it away and thus open the mastoid antrum. The bone-opening is within the limits of *Macewen's suprameatal triangle*, a space bounded by the posterior root of the zygoma, the posterior bony wall of the meatus, and an imaginary line joining the two. If the mastoid is opened in this triangle the antrum is entered directly and there is no chance of wounding the lateral sinus. If, in the adult, pus is not found on opening the mastoid antrum, gouge downward and backward, but with great care, so as to avoid the

lateral sinus. If there be any possibility of the existence of pus in the groove of the sinus, the sinus should be unhesitatingly exposed. After evacuating the pus from the mastoid, gouge away bony septa, enlarge the opening between the mastoid and the middle ear with the gouge and remove the superior half of the posterior bony wall of the meatus (avoid the facial nerve on the floor of the meatus), turn the head toward the side operated upon, and irrigate the mastoid with salt solution, dust with iodoform, pack with iodoform gauze for a few days, and then introduce a silver drainage-tube. Treat the causative ear disease. Sheild and Macewen operate on inveterate cases of mastoid disease as follows: A thick flap is raised behind the auricle, the flap including the orifice of any sinus and being "left attached by its stalk." The auricle is "detached forward and the soft parts over the mastoid are turned backward by horizontal incision." The "lining membrane of the canal is separated from the bone." The mastoid is opened and dead bone and caseous matter are removed, overhanging edges are chiseled down, and the posterior bony wall of the external auditory meatus is gouged away. The skin-flap is pushed into the cavity and is held in place by pads of gauze. The margins of the flap may be sutured, but this is not necessary. Macewen calls this procedure "papering" the cavity with skin.[1]

If mastoid suppuration has established *abscess in the temporosphenoidal lobe*, trephine, 1¼ inches behind and 1¼ inches above the middle of the external meatus (Barker's point), and search for pus as directed on page 804. If *abscess of the cerebellum* exists, trephine below the line of the lateral sinus. "The position of the lateral sinus is indicated by a line running horizontally outward from the occipital protuberance to within about 1 inch of the external auditory meatus, and thence downward to the mastoid process" (Owen's "Manual of Anatomy"). If *infective sinus-thrombosis* exists, break into the lateral sinus (Fig. 535, *d*) from the mastoid opening and proceed as directed on page 807.

**Linear Craniotomy.**—Make a large flap. Trephine the skull a finger's breadth from the sagittal suture, and the same distance back of the coronal suture. Rongeur the bone away in a line parallel with the sagittal suture and a safe distance from the longitudinal sinus, up to a point in front of the lambdoidal suture. Remove the pericranium which covered the bone excised. Insert the dural separator or pass it along the margins. In some cases an additional portion of the bone is removed over the fissure of Rolando. Various suggestions have been made as to the direction and situation of bone-sections. Bleeding is arrested and the flap is closed without drainage.

**Removal of Gasserian Ganglion.**—(See page 766.)

**Operation for Infective Sinus-thrombosis.**—(See page 807.)

**The Decompression Operation** (*Decompressive Trephining*).—This operation is employed particularly in cases of inoperable brain tumor. It differs from palliative trephining in the fact that the dura is incised and an opening left to permit of bulging of the brain. The bulging relieves pressure. By Cushing's method we get a hernia of the brain, but not a fungus cerebri. I have followed Cushing's recommendation in tumors, and have used it in fractures of the base of the skull, and I believe it often saves vision and life (in the latter condition it is done on both sides).

Cushing and Bordley have performed it in cases of uremia and improvement has followed ("Amer. Jour. Med. Sciences," Oct., 1908). They suggest that the operation be used in certain cases of renal disease when medical treatment and lumbar puncture have failed to abate uremic symptoms, or when blindness is impending. It has been used in apoplexy.

The effect of the operation in cases of brain tumor is sometimes extraordinary. Its most prominent benefit is in abolishing choked disk. It must

[1] "Lancet," Feb. 8, 1896.

not be done directly over a tumor, because the bulging tumor might become the seat of hemorrhage. It may, however, be done near the tumor (see page 820).

It is, of course, useless in relieving blindness, for blindness means atrophy, but it is often very valuable in *preventing* blindness. When choked disk exists operation should be done early even if there is good vision. If in advanced cases any sight remains, it should be performed. Now and then there is an unfavorable result, for instance, the development of retinal hemorrhages or the loss of vision, which was good previous to operation. (See deSchweinitz and Holloway on "Operative Treatment of Papillo-edema Dependent Upon Increased Intracranial Tension," "Therapeutic Gazette," July 15, 1909.) The permanence of the relief to the choked disk is variable. It is not always permanent.

Cushing's subtemporal decompression is done upon the right side, as a rule, but in some cases on the left side. An objection to doing it on the left side is

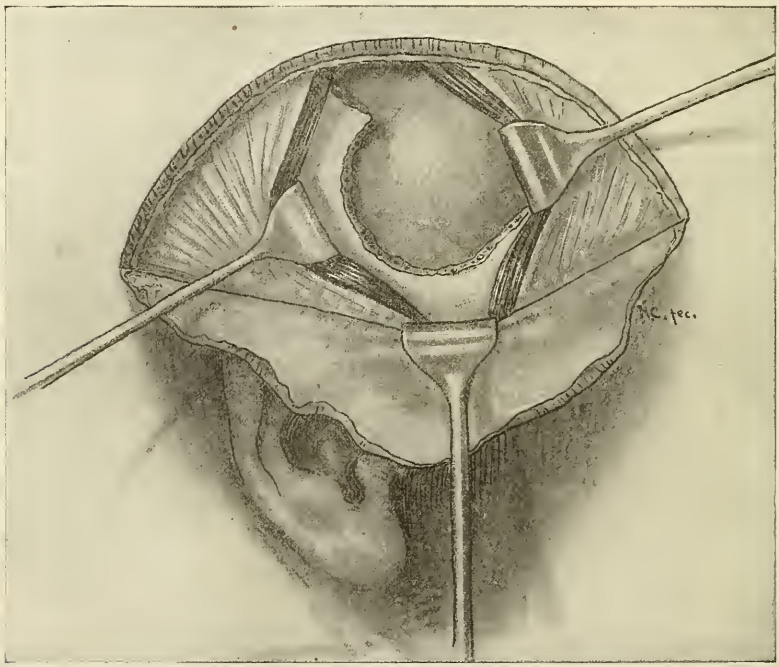


Fig. 536.—Sketch of the intermusculotemporal field of operation, showing exposure with subtemporal bone defect partly made (Cushing, in "Keen's Surgery").

that the bulging of the left temporosphenoidal lobe may cause word-deafness. A curved incision is made through the skin and subcutaneous tissue, the flap is turned down, the temporal fascia is incised in the direction of the muscle-fibers beneath it, the temporal muscle is split and not cut, the periosteum is separated from the bone, the soft parts are retracted, the bone is opened as the surgeon prefers, and the opening is enlarged by a rongeur (Fig. 536). The dura is opened and radiating incisions are made through it toward the edges of the bone gap. The wound is closed by four layers of fine silk sutures.

Figure 537 exhibits the exposure for suboccipital depression as done for subtentorial tumors. The same exposure is obtained in order to remove a cerebellar tumor.

**Drainage of the Cisterna Magna** (*Haynes's Operation*).—An incision is made in the middle line from the occipital protuberance to the posterior arch

of the atlas. The periosteum is stripped from a portion of the occipital bone at and above the foramen magnum. The bone is trephined and is cut away into the foramen magnum. The occipital sinus may not be present. If present as a double sinus, incise the dura between the two sinuses. If there is one sinus, divide the dura between two ligatures. Open the dura and arachnoid by a very small incision. As soon as excess of fluid has escaped enlarge the incision. By lifting and separating the cerebellar lobes the surgeon can determine if the foramen of Magendie is patent. Gutta-percha tissue is used as a drain. The muscles are sutured together by catgut. The skin is closed below the drain

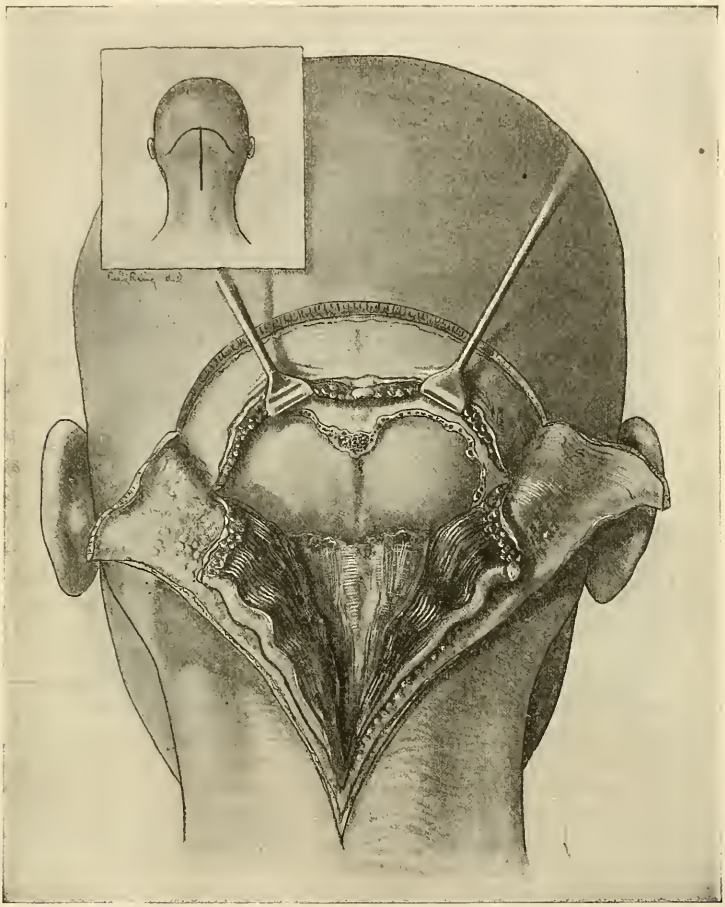


Fig. 537.—The suboccipital exposure, showing opening partly made and Cushing's "cross-bow" incision (Cushing, in "Keen's Surgery").

by silkworm-gut (abbreviated from Haynes's description in "The Laryngoscope," June, 1912).

**Methods of Reaching the Pituitary Body.**—The subtemporal route was suggested by Carton and Paul. Horsley has operated in this way. The bone is removed from the subtemporal region, the dura is opened, the brain is lifted, the dural box of the hypophysis is incised. It may be necessary to remove bone from each subtemporal region and raise the brain from both sides. Borchardt performed osteoplastic frontal resection, ligated the superior longitudinal sinus, and lifted the brain.

Giordano suggested a transsphenoidal operation, and Schloffer improved his method. Schloffer freed the nose so as to turn it toward the right side and then excised the turbinates, orbital wall, maxillary sinus, the middle septum, the left nasal process of the superior maxillary, and the ethmoid cells (Cushing, "The Pituitary Body and Its Disorders"). In the inferior route of Kanavel most of these structures are preserved and the incision is within the mouth.

McArthur ("Jour. Amer. Med. Assoc.," June 29, 1912) trephines the frontal eminence of one side 4 cm. above the supra-orbital notch and preserves the button in warm salt solution. By means of the DeVilbiss forceps and the chisel he cuts away a bone-fragment, which brings with it a considerable part of the roof of the orbit. This fragment, too, is placed in warm salt solution. The rest of the orbital roof is cut away by a rongeur. The dura covering the inferior surface of the frontal lobe is separated from the bone, while the lobe is raised and the orbital contents are held out of the way. Between the clinoid

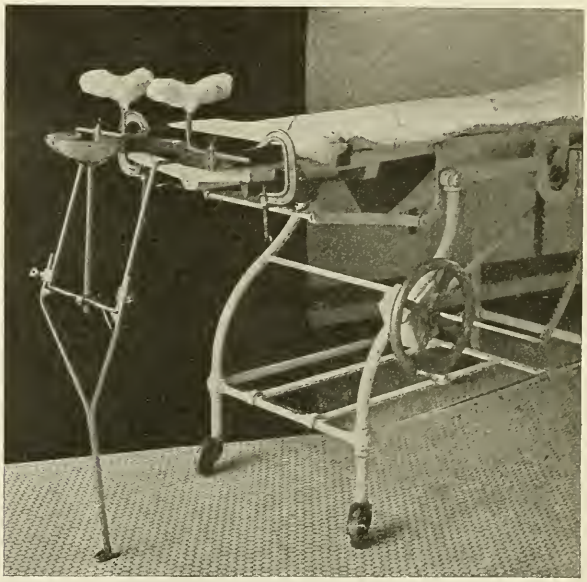


Fig. 538.—Horner B. Smith's head-rest for operations upon the cerebellum (modified by William J. Taylor).

processes the dura is divided by Krause's hook-shaped knife. An enlarged hypophysis will now be accessible. At the conclusion of the operation on the hypophysis the bone-fragments are replaced. Frazier has modified McArthur's operation ("Annals of Surgery," Feb., 1913).

**The Combined Method of Harvey Cushing** (From Cushing's "The Pituitary Body and Its Disorders").—This surgeon, whose experience is very large, operates as follows: Intratracheal anesthesia. Place the patient with the shoulders slightly raised so that the head drops back. Pack the posterior nares with a sea-sponge. Insert cotton wet with adrenalin in each nostril. Lift the upper lip. Make a transverse incision across the frenum, and carry it down to the anterior nasal spine. By blunt dissection raise the soft parts on each side from the inferior margin of the osseous nasal opening until the cartilaginous septum is exposed. From now on the operation is submucous. Separate the membrane on each side from the bony and cartilaginous septum. Introduce a retractor on each side between the separated mucous membrane and the septum. Separate the blades of the instrument. Remove "most of the vomer,

the lower edge of the median plate of the ethmoid, and a small strip of cartilage." It may be necessary to remove by means of a rongeur the anterior maxillary spine. With the retractors retained, a series of dilating plugs are introduced. These "flatten the turbinates." The largest dilator has a diameter of 1.8 cm. The retractors are removed and a bivalve speculum is inserted. An electric head-light must now be used. It may now be found necessary to remove "the prow of the vomer."

The body of the sphenoid is identified and "the anterior and lower walls of the sinuses are chipped away with long-handled nasal rongeurs." The lining of mucous membrane is removed, when the protrusion of the sella can be identified. The floor of the pituitary fossa is then clipped away. The dural case of the gland (or tumor) is divided by a hook-shaped knife.

Removal of the entire anterior lobe must never be done, as this lobe is necessary to life.

## XXV. SURGERY OF THE SPINE

**Congenital Deformities.—Myelocele or Rachischisis.**—This condition is due to deficiency in the formation of the vertebral arches, the cord being rudimentary, the medullary plates having failed to coalesce, the central canal not having formed, and the endothelium which should line it being exposed. If the entire cord is involved, the condition is called *amyelia* or *total rachischisis*. If a part of the cord is involved, the condition is called *partial rachischisis*. In partial rachischisis a portion of skin is absent in the midline. At this area is a circular, dark-red focus surrounded by a very thin and glistening membrane which becomes continuous with the skin. A dimple at the upper part and a dimple at the lower part of the dark area indicates the situation of the central canal above and below. Victims of rachischisis are usually stillborn or, at most, live but a few days.

**Spina Bifida.**—This is a deformity similar to the one just discussed, but in it the cord is much more developed. The first accurate description of it was given by Tulpius in 1685. It is a congenital sac of fluid due to vertebral deficiency, permitting protrusion of the contents of the spinal canal in the median line. In this condition the cutaneous epiblast is adherent to the spinal exiblast, because structures from the mesoblast have failed to grow between. The laminæ or spines of one vertebra or of several vertebræ or of many vertebræ may be deficient, most frequently in the lumbosacral region. In very rare cases there is division of the vertebral bodies and the projection is forward and to the side. A case in which there are ununited laminæ but no protrusion is called *spina bifida occulta*. Sometimes there are two protrusions in one person. In spina bifida the dura does not cover the sac because it is cleft as well as the laminæ. There are three distinct varieties of spina bifida: 1. *Meningocele*. In this condition the dura is cleft (Hildebrand), there is a protrusion of the arachnoid, fluid gathers in the arachnoid meshes and "distends this so as to form one continuous cavity which is traversed by nerve-roots" (Henle, in "A System of Practical Surgery," by von Bergmann, Bruns, and von Mikulicz. Translated and edited by Wm. T. Bull and Carlton P. Flint). The cord is not in the sac. 2. *Meningomyelocele* (the commonest form) is a protrusion of arachnoid, the sac containing cerebrospinal fluid, nerves, and cord-substance. The cord may spread upon the sac wall or it may pass through the sac and re-enter the canal. A cutaneous dimple or furrow indicates that the cord is attached and hence is within the sac. 3. *Syringomyelocele* is great distention of the central canal, the sac wall being formed of the thinned cord and the spinal membranes. A spina bifida varies in size from that of a walnut to that of

an infant's head; it grows rapidly during the early weeks of life; it is usually sessile, but may present, where it joins the body, a definite constriction or even a pedicle, the base of the sac is covered with healthy skin, and the fundus is covered only by thin epidermis or by the spinal membranes themselves. Pressure upon the tumor may diminish its size and increase the tension of the anterior fontanel, and possibly cause convulsions or stupor. The cyst is translucent and the margins of the bony aperture are distinct. Crying, coughing, or pressure upon the anterior fontanel makes the tumor more tense. Spina bifida is apt to be associated with club-foot, with hydrocephalus, and with rectal or vesical paralysis. Spina bifida usually causes death (90 per cent. of cases die during the first year of life). A few meningoceles and a very few meningomyeloceles undergo spontaneous cure by growth of the vertebral arches constricting the neck of the sac. The sac may remain distended with fluid or may shrink. Syringomyelocele is invariably fatal. The cause of death may be rupture of the sac or marasmus. The *x*-rays show the bony gap. *Spina bifida occulta* is a cleft in the vertebral column without any protrusion of the cord or the membranes. In this condition there is usually a profuse growth of hair in the skin over the bony gap and the hairy condition may be much more widespread. In some cases the hair is present at birth; in others it appears at puberty. Trophic changes and deformities may exist in the lower extremities.

**Treatment.**—Very small protrusions which grow slowly and are covered with sound skin may be treated by the use of a compress and bandage, by an elastic bandage, or by applications of contractile collodion. It was formerly regarded as proper to tap and drain the sac. Injection was used by many. The skin being cleansed, the child was placed on its side and a little chloroform was given. A fine trocar was plunged obliquely in at the side of the sac through sound skin, little or no fluid being drawn off, and 1 dram of Morton's fluid injected (iodin, 10 gr.; iodid of potassium, 30 gr.; glycerin, 1 oz.). The trocar was withdrawn and the puncture was sealed with a bit of gauze and iodoform collodion. The child was put to bed. If injection proved successful, the sac was found to shrink; if the injection failed, it was the custom to repeat it at intervals of from seven to ten days (Jacobson, White). Surgeons now prefer excision of the sac (see page 859). Whenever possible the incision should be through healthy skin. If the sac contains nerves they should be placed within the canal. Bayer treats it as he would a hernia. Robson in some cases excises the entire sac. Operations upon children much under the age of five have an enormous mortality. Operations are comparatively safe when the child reaches the age of five. Operations for spina bifida have been done successfully immediately after birth (Lovett, in "Amer. Jour. Orthop. Surg.," Oct., 1907). We should not operate if there is hydrocephalus or extensive paralysis, if the mass is very large and growing rapidly, or if there are other marked deformities. A ruptured sac should be operated on at once, otherwise death is practically certain.

**Sacrococcygeal Tumors.**—Dermoids external to the sacrum are occasionally seen in this region. Dermoids also arise between the rectum and sacrum. In the lower sacral or coccygeal region the cutaneous structures sometimes fail of complete coalescence and a *postanal dimple* or *sinus* is the result. Such a sinus is lined with skin and its wall contains numerous glands and often hairs. It may inflame or suppurate. If it blocks up at the outlet, a form of dermoid develops. Teratomata, lipomata, and hydatid cysts may develop in the sacrococcygeal region.

**Treatment.**—Dermoids require extirpation. If a postanal dimple causes no trouble, it is let alone; otherwise it should be dissected out. It may or may not be possible to remove teratomata. Lipomata and hydatids are extirpated.

**Anosacral Cysts.**—These cysts develop between the sacrum and rectum and originate from remnants of the postanal gut and neurenteric canal. Such cysts may be multilocular or unilocular. They can be detected by a finger in the rectum.

**Treatment.**—Some of these growths are removed after osteoplastic resection of a portion of the sacrum; others are removed by incising the rectal wall.

**Tumors of the Spinal Cord.**—Tumors may arise from the cellular tissue, fatty tissue, the nerve-roots, the membranes of the cord, or from the vertebra (*extramedullary tumors*). They may arise within the cord (cord tumors proper or *intramedullary tumors*).

**Extramedullary Tumors.**—Syphilomata, hydatid cysts, tuberculomata, and inflammatory masses or adhesions may compress the cord and produce symptoms indistinguishable from genuine tumor. Among extramedullary tumors are secondary carcinoma, sarcoma (primary or secondary), fibroma, myxoma, lipoma, chondroma, and neuroma. Dermoid sacral cysts may exist. Lipoma, fibroma, and certain cysts may be congenital. Injury of the back sometimes seems to bear a causal relation to extramedullary tumors.

The *symptoms* are due to pressure upon nerve-roots and the cord. The most prominent symptoms are pain in the back and evidences of nerve-root irritation.

The early or irritative symptoms are pain and stiffness of the back, usually very severe and interfering with sleep, shooting pains in the area of the implicated nerve-roots, and sensory abnormalities in the same area. There may be hyperesthesia of a limited area. The area of distribution from one or two roots is involved in pain, sensory disturbance, and slight motor impairment. In some cases the nerve-roots of one side only exhibit irritation and the symptoms are strictly unilateral. In other cases the symptoms are bilateral, but are most marked on one side. In some cases the symptoms are symmetrically bilateral and indicate pressure upon the cord rather than upon nerve-roots. Muscular spasms may occur. There may be lateral curvature of the back, the concavity of the curve being on the side of the tumor. Sooner or later paralytic symptoms come on (motor and sensory paralysis). They may be due to pressure upon and destruction of nerve-roots or to compression of the cord. When anesthesia exists there may be a zone of hyperesthesia above its upper limit. As motor palsy develops from root compression the pain usually abates. The muscles undergo atrophy.

A tumor may, by cord pressure, produce the symptoms of compression-myelitis, locomotor ataxia, or myelitis. Contractures or paraplegia may arise from tumor. The location of the growth can be inferred by a study of the territory of paralysis and the zone of sensory disturbance. The tumor is always situated somewhat above the upper limit of anesthesia. In many cases the diagnosis is impossible. Gradually increasing painful paraplegia, with pain in the back and with hyperesthesia or anesthesia after a time appearing and ascending from the feet toward the trunk, points to tumor as a cause. The paralysis is usually spastic, but may be flaccid, or it may be spastic at first and become flaccid. In spastic paraplegia the reflexes are increased. In flaccid paraplegia they are decreased. In spastic paraplegia there are ankle clonus, the Babinski sign (extension of the great toe or all the toes when the sole of the foot is irritated), Gordon's paradoxical reflex (extension of the great toe or all the toes when pressure is made upon the deep calf muscles), and Oppenheim's reflex (extension of the great toe or all the toes when the handle of the percussion hammer is drawn along the inner edge of the tibia so as to make pressure from above downward). Trophic lesions are apt to arise in the trajectory of nerve involvement. The sphincters are usually involved. Growths outside

the membranes produce particularly pain and spasm; growths within the membranes produce especially motor paralysis and anesthesia. Symptoms that are unilateral, were at first unilateral, or which are most marked on one side, are very significant.

**Intramedullary Tumors.**—These tumors develop in the substance of the cord. They are far less common than extramedullary growths and are more often benign. These growths are, as a rule, primary and solitary and do not produce symptoms of pressure until they attain the size of a hazelnut. The most common tumors are glioma, sarcoma, and tuberculoma. Syphiloma occasionally arises. Most tumors in cord-substance are small, but the glioma may involve practically the entire cord. A tumor is often for a time limited to one side of the cord, but later it presses upon and finally involves the opposite side. When the cord is pressed upon, degeneration occurs. In some cases a wrench or bruise of the back is supposed to be causal. Glioma may be congenital.

*Symptoms.*—They are at first, in most cases, very uncertain. In some cases, however, paralysis develops early.

There is often pain in the back, but it is not nearly so severe as in extramedullary tumor. The most prominent symptom is a slow-developing motor palsy. In some cases the palsy is at first unilateral, but later becomes bilateral. Irritative root symptoms are absent (spasm and darting pain). Anesthesia or hyperesthesia develop. The sphincters are involved and trophic disturbances arise. There may be spastic paraplegia or flaccid paraplegia. If the tumor is in or presses upon the anterior cornu there will be limited muscular atrophy. Tuberculoma produces the symptoms of transverse myelitis.

In glioma there are paresis and muscular atrophy. Although sensibility to pain, heat, and cold are lost, sensibility to touch is preserved (sensory dissociation).

**Treatment of Tumors of the Cord.**—If syphilis is suspected, give the patient a course of heroic doses of iodid of potassium, and administer mercury hypodermatically or by inunction. Intravenous injection of salvarsan is advisable. In a focal lesion not due to dissemination of a known malignant growth, perform the operation of laminectomy to permit of exploration and possibly of removal. The laminæ of at least three vertebræ should be removed and the tumor looked for distinctly above the upper level of the zone of anesthesia. It is not necessary for the patient to wear a spinal support after the performance of laminectomy. Extramedullary tumors are usually removable. Localized intramedullary tumors should be removed if they can be located and if removal can be accomplished without serious injury to the cord. After exposing the cord and discovering an intramedullary tumor, follow the advice of Elsberg and Bier ("Amer. Jour. Med. Sciences," Nov., 1911) and make a short incision in the posterior median column a little external to the posterior median fissure. The cut reaches the tumor, which bulges through it. Do not attempt removal now. Suture the skin and muscles and wait one week. On opening the wound the tumor will be found almost completely extruded from the cord. In order to remove it it is only necessary to divide a few strands of tissue. Extrusion by this method probably inflicts little injury upon nerve-fibers. McCosh truly said that operation for spinal-cord tumor was decidedly more hopeful than for brain tumor, because localization was much more accurate and removal could be effected with less permanent damage. Lloyd collected 51 operations: 10 per cent. died and 31 per cent. were actually cured or improved. Joseph Collins ("Med. Record," Dec. 6, 1902) collected 70 cases of spinal tumor, 30 of which were operated upon. In 12 the operation was a success, that is, the pain disappeared and motor power returned; in 8 the operation was partly successful, that is, the pain disappeared

and the motor power improved; in 10 the operation failed and death occurred within a few weeks. If the tumor is found to be irremovable, McCosh suggests division of several nerve-roots to relieve the pain.

**Acute osteomyelitis of the vertebræ** is a rare disease; it may be associated with osteomyelitis of other bones, may be secondary to some distant suppurative focus, or may occur alone. Infections of the viscera not unusually accompany it. In many cases there is a history of trauma. Any part of a vertebra may suffer from it. This condition may follow cold, overexertion, or traumatism, and is more common in the first two decades of life than in elderly people. The process may be superficial or it may involve the bone deeply and widely. Suppuration always occurs; sequestra generally form, and phlebitis is a dangerous complication. Any region of the spine may be attacked, but the lumbar region is particularly liable to invasion, next the dorsal, next the cervical. The sacral region is least often affected. The situation of the abscess varies with the situation of the disease. If the vertebral bodies are diseased the pus passes forward (retropharyngeal, mediastinal, psoas, or pelvic abscess). If the vertebral arches suffer the pus passes backward (lumbar or dorsal abscess). The membranes of the cord, the cord itself, the nerves, and the vertebral articulations are frequently involved in the process. Staphylococci, streptococci, or other pyogenic bacteria may be cultivated from the pus.

**Symptoms.**—The general symptoms are those of osteomyelitis. The local symptoms depend on the seat of disease. If the posterior portion of the column is diseased there is a hard swelling which, in the neck, is in the middle line; in the dorsal and lumbar regions, in the middle or to the side; and in the sacral region, invariably to one side.

Rigidity of the spine always exists. If the vertebral bodies are affected, rigidity is noted, the spine is tender, and special symptoms appear, their nature dependent on the region affected (retropharyngeal abscess, etc.). Occasionally symptoms of meningomyelitis are noted. The constitutional symptoms of sepsis are marked. The condition is sudden in onset, and purulent collections diffuse widely and rapidly. These points enable the surgeon to make a diagnosis between osteomyelitis and Pott's disease. In osteomyelitis angular deformity very rarely arises, because the patient is obliged to be recumbent and because hyperostosis is taking place. The mortality, according to Hahn, is 60 per cent. Death may be due to pachymeningitis, pneumonia, empyema, retropharyngeal abscess, invasion of the cord, or amyloid disease (H. S. Warren, "Boston Med. and Surg. Jour.," May 7, 1903).

**Treatment.**—The patient is kept recumbent. His constitutional treatment is such as will combat sepsis (food, stimulants, etc.). A puriform area must be incised and disinfected. If bone denuded of periosteum is found, it is touched with a solution of chlorid of zinc or with the actual cautery. If a sequestrum exists, it is removed. A drainage-tube is inserted and dressings are applied (Müller, Makins, Abbot, and Chipault).

**Typhoid Spine.**—It was pointed out by Gibney in 1889 that typhoid fever may leave as a legacy a painful, stiff, and weak back; 74 cases of the condition have been reported (F. W. White, in "Jour. Amer. Med. Assoc.," Feb. 13, 1908). The muscles of the back are found to be rigid and there is tenderness of one or more vertebræ. The pain may only be appreciated on motion, but in some cases there is aching even when the patient is at rest. The pain may be localized, may run into one or both thighs, or may be felt in the abdomen. The symptoms arise at an uncertain period after the fever, develop rapidly, and are occasionally associated with transient episodes of fever. Kyphosis or lateral curvature may develop. (See L. W. Ely, "Medical Record," Dec. 20, 1902.) Many of the patients are hysterical. The condition

is due to osteitis and periosteitis, or chronic osteomyelitis. The prognosis is excellent.

**Treatment.**—The use of a plaster or leather jacket; counterirritation by the hot iron; later, massage and electricity.

**Cervical Rib.**—This condition was first described by Hunauld in 1743. The anterior limb of the transverse process of the seventh cervical vertebra, which has an independent center of ossification, may develop into a separate bone of large size, known as a cervical rib. Such a rib may form on one side or on both. It may scarcely reach beyond the transverse process, it may project well beyond the transverse process and have a free end, or it may constitute a complete rib which fuses anteriorly with the sternum, the cartilage of the first rib, or with a cervical rib of the opposite side.

Most instances described were found in the dead body, although Tillmanns collected 26 cases among the living (Carl Beck, in "Jour. Amer. Med. Assoc.," June 17, 1905). Of late *x*-ray findings indicate that the condition is much

Fig. 539.—Cervical ribs.

more common than was formerly supposed. I have seen 4 cases. It may never produce any uneasiness, and hence may escape detection and seldom does produce trouble in youth. It may lead to damage of the subclavian artery (Keen's case developed aneurysm), or gangrene of the hand may result from bending or blocking of the vessel, or neuritis of the brachial plexus may arise from pressure. When sufficiently large to produce venous or vascular trouble, a cervical rib can be felt and the pulsating artery over it is very distinct and higher than natural in the neck. The *x*-rays confirm the diagnosis. The treatment, when the rib is causing trouble, is excision of the rib with its periosteum (see page 707). (See Kammerer, in "Annals of Surgery," Nov., 1901, on "The Diagnostic Difficulties.")

**Spinal Curvature.**—There are four chief forms of spinal curvature: (1) Lateral curvature (the scoliosis of the older surgeons); (2) posterior curvature (the excurvation, gibbosity, or kyphosis of the older surgeons); (3)

anterior curvature (the lordosis of the older surgeons), and (4) angular curvature (from spinal caries). The normal spine has four curves: the *cervical* curve, the convexity of which is forward; the *dorsal* curve, the convexity of which is backward; the *lumbar* curve, which is convex anteriorly, and the *pelvic* curve, which is concave anteriorly. The dorsal and the pelvic curves, which are primary, are due to the formation of the cavities of the chest and pelvis, and depend upon the shape of the bones (Treves). The cervical and lumbar curves, which are compensatory, depend upon the shape of the intervertebral disks, and only appear after birth when the erect position is assumed.

CONDENSED DIFFERENTIAL DIAGNOSIS TABLE OF SPINAL DISEASES AND CONDITIONS WITH WHICH THEY MAY BE CONFOUNDED.

| | Scoliosis. | Rachitic Spine. | Pott's Disease. | Hyperesthetic Spine. | Arthritis Deformans. | Torticollis, Congenital. | Hip Disease. |
|---|---|---|---|---|---|---|---|
| Age. | 8 to 16. | 4 to 6. | 4 to 6. | 16 to 20. | After 30. | Any age. | 4 to 6. |
| Onset. | Insidious. | Insidious. | Insidious. | Sudden. | Insidious. | From birth. | Insidious. |
| Pain. | In back. | None. | Referred to anterior abdomen. | Severe in spine. | In spine. | None. | In knee. |
| History. | None. | Rachitic. | Tuberculous. | Trauma. | None. | From birth. | Tuberculous. |
| Posture. | Free. | Excessively free. | Guarded spine. | Guarded. | Guarded spine. | Typical. | Guarded hip. |
| Muscular rigidity. | None. | Free. | In spine. | In spine. | In spine. | In one direction only. | In hip in all directions. |
| Temperature. | Normal. | Normal. | 1 degree rise. | Varies. | Normal. | Normal. | 1 degree rise. |
| Local tenderness. | None. | None. | In spine. | Painful all over. | All over spine. | None. | In hip. |
| Night cries. | Absent. | Absent. | Present. | Absent. | Absent. | None. | Present. |
| Tendency to abscess. | None. | None. | Probable. | None. | None. | None. | Probable. |
| *X*-ray. | Characteristic deformity. | Normal. | Focus in spine. | Normal. | Late bridges of bone. | Distortion of cervical spine. | Spine normal. |
| Hot-water test. | No tenderness. | None. | Localized in spine. | Sensitive all over spine. | No necrosed sensitiveness. | No tenderness. | No tenderness in spine. |
| General symptoms. | Constant. | Constant. | Intermittent. | Constant. | Constant. | Constant. | Intermittent. |

**Scoliosis** is a non-pathological distortion of the spine characterized by rotation and lateral bending, hence the name, rotary lateral curvature (Plate 9). It is either functional or organic.

Functional scoliosis is caused by any prolonged alteration in the relationship normally existing between the axis of the shoulders and the axis of the hips. The etiological factors depend upon the maintenance of faulty postures in occupations, especially during period of growth. School-life is especially influential in producing the condition because school furniture is adapted to the average requirements of a given class, and therefore a very small proportion of pupils are able to find desks and benches that are suitable. Properly furnished modern schools are supplied with desks and seats that are adjustable to each occupant. Gould ("The Ocular Factors in the Etiology of Spinal Curvature," H. Augustus Wilson, "New York Medical Journal," July 12, 1906) has directed attention to the errors of refraction that produce head tilting and thereby induce scoliosis. In adults, scoliosis is observable in blacksmiths, fencing masters, and waiters, in whom excessive use of the right arm produces an asymmetrical posture of the body.

When functional scoliosis is not corrected or occurs in rachitic or indolent children, it results in organic or permanent changes in the contour of the bones composing the spinal column. Organic scoliosis may be congenital, caused by prenatal deficiencies or augmentations of the spinal column.

Postmortem examinations reveal a confirmation of Wolfe's law that prolonged alteration of the normal functions always results in changes of

anatomical structures. In extreme cases the bones of the spinal column become extensively altered in shape, the ribs are altered in contour, and the thoracic and abdominal viscera are forced into unnatural positions and assume abnormal shapes, their functions often being materially altered.

*Diagnosis.*—The patient is usually taken to the physician because of apparent elevation of one shoulder, or because one hip is thought to be larger than its mate, or one mamma higher than the other. Dressmakers and corset makers are generally the first ones to direct attention to the faulty posture (as scoliosis occurs in 8 girls to 1 boy). A patient suspected of having scoliosis should be nude at the time of inspection, as clothing hampers the normal action and tends to conceal the movements of the body. The accompanying diagnostic table (see page 841) is a condensed comparative statement of the important features in diseases and conditions that may resemble scoliosis in some respects. It is only by carefully studying the symptom-complex that a definite decision can be reached. It is frequently observed that patients with mild types of functional scoliosis can sit or stand erect for a few minutes and thereby deceive even a critical observer. The habitual posture, and also the rapidity with which the patient returns to the distorted position after temporary voluntary correction, demand careful attention. Young children, when chided by parents or teachers, often temporarily assume an approach to a normal posture without actual correction. The ease with which their surrounding joints yield in compensatory action is often overlooked. A patient with contracture of the pectoral muscles will elevate the shoulders into an apparent correction of stoop or round shoulders instead of throwing the shoulders well back, a posture that is to them impossible.

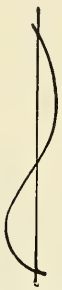


Fig. 540.—Lateral dorsal curvature to the right, and compensatory lumbar curve to the left.

Goldthwait has directed attention to forward curves of the scapula that are often present in patients who are stoop-shouldered and has devised an operation for correction.

In the same way, a patient who has preternatural contracture of the hamstring tendons, either unilateral or bilateral, will often conceal that condition by bending the knee or knees enough to permit the pelvis and trunk to bend forward.

*Treatment* consists especially in removing the cause. If the eyes produce head-tilting, proper refraction will be necessary. Adenoids should be removed. The clothing should be regulated to avoid constriction and the shoulder straps should fit close to the neck and not be allowed to slip on to the shoulder-joint.

Contractures of the pectoral muscles should be stretched by corrective manipulations. Hamstring contracture should be removed by corrective manipulation. Hoke ("A Study of a Case of Lateral Curvature of the Spine: A Report On an Operation for the Deformity," "Amer. Jour. Orthop. Surg.," vol. i, November, 1903, p. 169) has devised an operation of rib resection for cosmetic purposes. Every effort should be made to prevent the *occurrence* of scoliosis. The successful treatment of scoliosis depends on preventing its progress. Each individual patient requires careful study to determine the special characteristics that may be present.

Remedial measures should be employed that meet the peculiar individual requirements of each case.

School gymnastics are generally more harmful than beneficial in cases of scoliosis. No one but a physician should prescribe the gymnastic work. The soft bones may be still further distorted by injudicious exercises.

Rotary lateral curvature of the spine.

The first requirement in the application of applied physical culture is to secure the hearty coöperation of the patient. Without such coöperation progress cannot be expected.

No gymnastic apparatus of any kind is required when the patient can be instructed in the proper methods of autoresistance. There are over four thousand movements of the body that may be employed in remedial physical culture. From this vast assortment those may be selected that are suitable to the peculiar conditions of the patient. At first the least tiresome forms are to be employed, and gradually and progressively others are resorted to until the patient presents a strong robust development. Usually about a year is required for the purpose, as the progress must be essentially educational. Training in developing muscle action goes hand in hand with instruction in walking, in sitting, and in all the postures assumed by the human body in the various occupations of the patient.

In organic scoliosis, in which the distortion is more or less of a permanent character, much can be accomplished in preventing the progress of the condition as well as in aiding correction by removing any rigidity that may be present. By increasing the flexibility we facilitate muscular development in much the same manner as in functional scoliosis.

When rigidity is present it must be considered in the same light as rigidity of any other joints. Its presence prevents muscular development. Manipulative measures are similar in effect to those employed in fibrous ankylosis of any joint, and are peculiar to the parts involved. In the majority of cases the force for manipulative correction must be applied through the interposition of the ribs, and the great danger of producing fractures of these structures should be realized.

If, however, there is any tendency to increase of the deformity, a suitable brace or removable jacket of plaster or celluloid should be applied to maintain correction until the musculature has been strengthened and trained to perform its full functions.

Organic or structural scoliosis has been shown by Abbott, of Portland, to be capable of correction by several types of fixed jackets (E. G. Abbott, "New York Med. Jour.," 1912, and A. M. Forbes, Ibid., July 6, 1912). The keynote of the treatment in each method is flexion of the spine, as in that position the vertebræ are unlocked. Partial correction is secured by means of bandages pulling in various directions and, after proper padding, a plaster jacket is applied. Further progress to overcorrection is continued by means of pads of felt slipped under the jacket to increase the pressure in certain directions. After overcorrection has been obtained, a celluloid jacket is worn to maintain this and exercises are assiduously employed to restore muscle function. These methods of treatment constitute the greatest advances in this work in many years, but to secure the best results they must be followed out most carefully by a skilled physician. Special forms of apparatus are essential in the application of these jackets. The combination of the gymnastic and mechanical treatment in severe functional and in organic scoliosis proves the most satisfactory in that it not only restores muscle balance, but also corrects deformity, and secures as great correction of the deformity as possible before ankylosis occurs. Any appliance used in these cases should be made to order to fit the peculiarities of the patient and should never be the shop-kind that are so extensively advertised. Much valuable time is often lost while unmechanical and unsuitable apparatus is being used, during which time the bony changes may become permanent and beyond repair.

**Anteroposterior curvature** (not from spinal caries or from hip-joint disease) is an increase of the normal anteroposterior curves. Increase of the dorsal curve is *posterior curvature*, *kyphosis*, or *excurvation* (Fig. 541, A); in-

crease of the lumbar curve is *anterior curvature*, *lordosis*, or *saddle-back* (Fig. 541, B). Both lordosis and kyphosis are apt to be present. Scoliosis has nearly always some anteroposterior curvature associated with it. Lordosis is apt to be compensatory, to prevent the center of gravity going too far forward. Lordosis is found in pregnant women and in very fat men. In an old man kyphosis arises because of flattening out of the vertebral disks from pressure. Rheumatic gout may cause anteroposterior curvature. Anteroposterior curvature is often due to paralysis of the erector spinæ mass (from infantile paralysis). Pseudohypertrophic paralysis causes lordosis.

Fig. 541.—Kyphosis (A) and lordosis (B).

*Symptoms and Treatment.*—The *symptoms* of anteroposterior curvature are as follows: the thorax is flattened or pigeon breasted; the shoulder-blades are widely separated and the scapular angles project; the abdomen is protuberant; the patient complains of backache and soon tires. A recent kyphosis disappears when the patient lies upon his stomach. The facts that the erector spinæ muscles are soft and that pain is absent on concussion transmitted to the back separate kyphosis from caries. Lordosis is unmistakable. When the spine is movable, employ the same plan of *treatment* as in lateral curvature, suiting the gymnastics to the deformity. In painful kyphosis with partial ankylosis endeavor to make the ankylosis complete in order to prevent pain, obtaining this result by applying a plaster jacket which laces up and letting the patient wear it for several years.

**Angular curvature** (*spinal caries; spondylitis; Pott's disease*) is usually due to tuberculous caries of the vertebral bodies, and occurs particularly in children who are the victims of tuberculosis, but it may arise at any age. Any portion of the spinal column may be attacked. The dorsolumbar region is most prone to suffer. The chief *cause* is tuberculosis, but syphilis and secondary cancer of the vertebræ are occasional causes, and acute osteomyelitis is a very rare cause (see page 839). Blows or sprains appear to have a causal influence in some cases (see Trauma in Tuberculosis, page 223). Angular curvature may develop after an exanthematous fever.

The cancellous tissue of the anterior portion of the vertebral body becomes primarily carious, or the inflammation begins in an intervertebral disk. (The changes of tuberculous osteitis have previously been set forth—see pages 249, 494, and 495.) The body of the vertebra and the vertebral disk are destroyed, and the process extends to adjacent vertebræ. The weight which rests upon the spinal column causes softened bone to crumble, compresses the diseased vertebræ and disks, and produces angular deformity (the anterior part of the column formed by the vertebral bodies is shortened, the posterior part is not, and hence the spines project). In some cases the disease is spontaneously arrested by organization of inflammatory products, and ankylosis (fibrous or bony) in deformity is Nature's cure. In most cases, however, the disease spreads and caseous pus is formed, which, according to the point of formation and the route it takes, causes lumbar abscess, dorsal abscess, psoas abscess, or postpharyngeal abscess (see pages 241 and 242). In some cases the spinal cord is compressed, but in most cases it is not, and even when it is compressed paraplegia is rare and is usually temporary. Compression of the cord may be caused by the displaced vertebræ, by inflammatory material or caseous matter between the bone and dura mater, but is most often due to pachymeningitis. Caries of the cervical region constitutes a more dangerous disease than caries of either the dorsal or the lumbar region (*dangerous* pressure occurs more easily). Death may be caused by exhaustion, sepsis, hemorrhage,

amyloid disease, pneumonia, peritonitis, pleuritis, tuberculous dissemination, pressure upon the cord, or inflammation of the cord or its membranes.

*Symptoms.*—The sufferer from Pott's disease, if a child, grows tired easily. The disposition alters. The victim becomes moody and irritable, and complains of vague pains in many places, is disposed to lean, rest, or lie down, and walks with the back rigid, which produces a peculiar gait. A painful spot may be found by pressing upon the spines. Faradism to the back causes pain. Spasm of the erector spinæ mass is detected (Hilton, Golding-Bird). It is not proper to seek to develop pain by jarring the back or by pressing the head downward. The posture of the child and the muscular rigidity prove the existence of inflammation, and to seek to develop pain by the methods referred to may do harm, and at best can only call attention to what is already known. Pain in the back, which is increased by motion, by pressure, and by vertebral jars, may be absent until late in the case. Distinct pain and tenderness in the back often mean abscess formation. Neuralgic pains pass into distant parts (sciatica, intercostal neuralgia) and are often linked with muscular spasm. A chronic bilateral pain in the trunk or extremities is suggestive of Pott's disease. "Chronic bilateral bellyaches in children are almost diagnostic" (Jordan Lloyd). The pain of dorsal caries can be relieved by lifting the shoulders; the pain of cervical caries, by traction on the head. Cramp in the legs occurs in dorsal and in lumbar caries. The presence of the knuckle due to bending the spine at an acute angle is a very important sign of the disease. In many cases angular deformity appears late; in some cases it does not appear at all. An angular deformity is detected sooner in those regions where the normal curves are posterior than where the normal curves are anterior. The deformity appears early in the dorsal region, but late in the cervical and lumbar regions. In many cases lateral deformity occurs. Rigidity is an early sign of great importance. It is always present. Rigidity is manifest very early in cervical caries, tolerably early in lumbar caries, late in dorsal caries. Lloyd gives the following practical rules to enable us to detect rigidity.[1] In the cervical region: seat the patient in a chair and tell him to nod the head affirmatively. Stiffness in nodding points to occipito-atloid disease. Tell him to look far to the right and then far to the left. Stiffness of these motions suggests atlo-axoid disease. Tell him to place his shoulders against the back of the chair and carry his eyes back along the ceiling. Stiffness in this movement indicates disease below the second cervical vertebra. It is practically useless to examine the dorsal region of an adult for rigidity, but such an examination can be made in a child. Place the patient prone on an adult's lap, mark the tip of each spinous process with an anilin pencil, then make the child stand up straight on the floor, and observe if any of the pencil marks fail to come nearer together. If it is seen that two or more marks do not approach each other, there is rigidity which prevents approximation. To test for rigidity in the lumbar region lay the naked patient prone upon a couch. Grasp the patient's ankles and raise the pelvis from the couch. If the lumbar spine is flexible, the pelvis can be lifted without raising the chest from the bed, and the maneuver deepens the hollow of the loin. If the lumbar spine is stiff, the maneuver lifts the trunk and produces no alteration in the vertical outline of the lumbar spines. If a child with Pott's disease is asked to pick up something from the ground, because of rigidity or pain on movement he will not bend the back, but will bend the knees or get upon the kness. Paralysis may exist, and it is due to pachymeningitis more often than to pressure from bone. Cervical caries causes dyspnea and torticollis, the head requiring support with the hands. Dysphagia indicates abscess. In adults the first signs of

[1] "Birmingham Med. Review," April, 1897.

Pott's disease to attract attention are headache, backache, neuralgia, girdle-pain, cramp, or even paralysis. In abscess due to caries of the dorsolumbar vertebræ the pus usually enters the psoas muscle and passes out of the pelvis below the junction of the middle and outer thirds of Poupart's ligament. It may point here or may pass to the inner aspect of the thigh and point a little below the spot where a femoral hernia is met with if it exists. In a psoas abscess a mass is always felt in the iliac fossa above Poupart's ligament; in a hernia no such mass exists (J. T. Rugh). In sacral caries there is no deformity and frequently no pain. The diagnosis becomes apparent when bilateral abscess is detected in the buttocks or groins (Jordan Lloyd). If an abscess due to spinal caries opens spontaneously, healing will not occur, mixed infection takes place, and death often follows.

*Treatment of Caries of the Spine.*—When recent caries of the spine is active and affects a child; when it is accompanied by pain and fever; and when paralysis threatens, insist upon perfect rest. Place the child supine on a hard mattress, and, if possible, take it, while in a rolling bed, out of doors daily. Leeches, blisters, or the hot iron over the area of pain may do good.

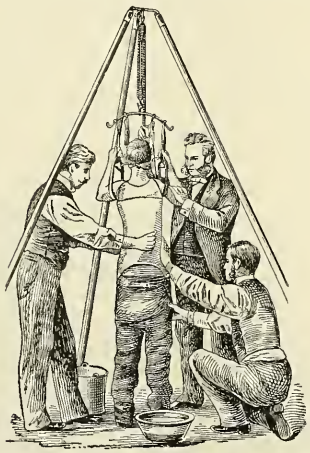


Fig. 542.—Plaster-of-Paris jacket (Sayre).

Fig. 543.—Plaster-of-Paris jacket and jury-mast applied (Sayre).

When the activity of the process abates, apply a fixation apparatus. In diseases at or near the vertebro-occipital articulation, as long as dyspnea persists, keep the patient supine with a small hard pillow under the nape of the neck (Hilton) and a sand-bag on each side of the head and neck. After several months mechanical support can be given by Furneaux Jordan's method. Jordan applies his support as follows: The patient lies on a flat, hard table, his arms are raised above his head, and traction is made upon the head by means of a pulley and a weight. Cotton pads are placed over the ears, the back of the neck and the clavicles, and are held in place by a broad flannel bandage applied as a figure-of-8 on the head, neck, and chest. The flannel bandage is overlaid with plaster-of-Paris bandages.[1] In disease of the cervical region below the axis, or of the dorsal region above the seventh vertebra, use Sayre's jury-mast (Fig. 543), or some other form of head support. Instead of the jury-mast a steel upright may be used to hold the head rigid. Sayre's appliance relieves the spine from the weight of the head and acts admirably. In most cases of dorsal and lumbar caries a steel, leather, or plaster jacket as a fixation apparatus must be employed. The best of all fixation apparatus

[1] See "Children's Deformities," by Walter Pye.

is Sayre's plaster-of-Paris jacket applied while the patient is suspended (Fig. 542) or, better, while the column is in hyperextension. The Sayre apparatus applied in this manner is used for the treatment of caries of the lumbar region and the lower half of the dorsal region, or a plaster jacket may be applied while the patient is lying prone in a hammock or stretched between two tables. Greater corrective pressure over the deformity is secured by this method than by suspension. When all subjective signs cease, substitute for the plaster-of-Paris jacket a felt or sole-leather jacket which laces down the front. Caries of the upper half of the dorsal region is often treated by a Sayre's jury-mast (Fig. 543); but if the jury-mast fails, it may be necessary to place the patient horizontally in "an open cuirass, fitted to the back from occiput to sacrum, and combined with pulley extension to the head and pelvis."[1]

During the course of caries of the spine have the patient eat fat-forming and nutritious food, insist on a plentiful supply of fresh air day and night, and administer tonics. Full antituberculous treatment is imperative (see page 230). Sea-air is very beneficial. When all active disease ceases and only angular curvature remains, use an apparatus to combine extension with mechanical support, the plaster jacket being generally employed.

**Albee's Bone Transplantation Method of Treatment.**—The ordinary ambulatory treatment of Pott's disease is seldom satisfactory. It is particularly unsatisfactory in the upper dorsal region. Ridlon and Jones emphasize this fact and declare that when the disease is situated in that region only prolonged recumbency prevents increase of the deformity. Albee reached the conclusion that the treatment should be one which secures bony union and hence perfect immobility. Perfect bony fixation means cure.

Albee was accustomed to see perfect cure in hip tuberculosis after ankylosing the joint, and in knee tuberculosis after erasion. He has secured a like result in vertebral caries by grafting into the spinous processes a large piece of bone cut from the patient's tibia. This graft usually becomes fixed to the spinous processes. It at least unites with the surrounding ligamentous structures and gives unyielding support which produces cure (see page 860). It is a highly useful method (Albee, in "New York Med. Jour.," March 9, 1912; in "Post-Graduate," Nov., 1912).

**Spinal abscesses** are treated as indicated on pages 245 and 246.

**Paralysis in Pott's Disease.**—Partial or complete motor and sensory paralysis may develop in the course of vertebral caries. It may be due to the pressure of tuberculous material or to pachymeningitis with thickening of the membrane. In only 2 per cent. of cases of paralysis is the paralysis due to the pressure of angled bone (Willard). The paralysis may come on gradually. There are weakness in walking or actual inability to walk, exaggerated reflexes, muscular rigidity, and impaired sensation in the legs, and loss of control of the bladder and rectum. Caries in the high dorsal region is more apt to result in paralysis than in any other region because of the small size of the canal. Pressure in the cervical region is highly dangerous.

*Treatment.*—We must remember that angulation is the rare cause, tuberculous masses the common cause. Treatment for paralysis due to tuberculous masses is the full open-air treatment of tuberculosis, with rest, fixation, and progressive straightening of the spine. The patient is kept in bed (see Treatment of Tuberculosis, page 230) on a Bradford frame and with his head overextended. If after one year the condition is not notably improved, do laminectomy and clear away tuberculous masses. If angulation is the cause of the paralysis, consider gradual correction, laminectomy, and Albee's bone transplantation. In several of the cases reported by Albee recovery from paralysis occurred soon after bone-grafting.

[1] Jordan Lloyd, in "Birmingham Med. Review," April, 1897.

**Gradual Correction of Angular Deformity.**—Pressure is made upon the hump with the hand, and while the hand is thus held the weight of the body is allowed to bear upon it above and below. Something is perhaps gained and then plaster of Paris is applied, somewhat later a little more gain is obtained, and so on. This method is safer and more satisfactory than forcible correction.

**Forcible correction of angular deformity** was advocated by Chipault and Calot in cases of Pott's disease without abscess. It was only used in angular deformity of the middle and lower part of the dorsal region and was not advised in the cervical, upper dorsal, or lumbar regions. The operation is not safe, and a number of deaths have been reported. Gabaert[1] pointed out certain disasters which may follow forcible correction; they are: death during anesthesia; rupture of an abscess; subsequent paralysis of the legs and bladder; disseminated tuberculosis, and shock, with convulsions and death. I do not believe in forcible correction and I do believe that the alleged dangers are real dangers, that the operation is unsafe, and that it should never be done.

**Laminectomy** is warmly advocated by some surgeons for paraplegia from spinal caries. This operation is rarely necessary, but in some few cases it is imperatively demanded. Many cases recover from paraplegia without operation. Operation for paraplegia has a very heavy mortality (25 per cent.), and many are not benefited at all by it. If degeneration of tracts in the cord has occurred, operation cannot help the paralysis. Nevertheless, in some cases laminectomy has certainly cured palsy and saved life. Menne has collected 132 cases of laminectomy. Of these 56 per cent. were cured or permanently improved and 18 per cent. were temporarily improved.

Laminectomy should not be undertaken until treatment by rest, fixation, and extension has been applied for at least one year. Laminectomy may become necessary in cervical caries to prevent asphyxia. The operation enables the surgeon to remove masses of inflammatory material which make pressure on the cord, and also to free the cord from pressure due to angulation. The dura should not be opened unless there is evidently trouble beneath it, in which case it is incised and any tuberculous area removed, the dura being subsequently sutured. Ménard removes the transverse processes of the diseased vertebræ and the heads and necks of the associated ribs in order to give the surgeon access to diseased vertebral bodies.

**Spondylitis Deformans** (*Bechterew's Disease*).—This is the name usually applied to osteo-arthritis of the spine (see page 642). In this disease osteophytic formation takes place at the vertebral borders, and the vertebræ become ankylosed. The vertebral bodies, as a rule, are most affected by the disease, but any portion of a vertebra may be attacked, and often the heads of the ribs are anchored to the spine by bone.

The disease may begin in infancy, childhood, youth, adult life, or old age.

**Symptoms.**—There are decided and persistent pain and tenderness of the spine, and occasionally evidence of pressure on the nerve-roots. Early in the case deformity is apt to occur, because at this period there is inflammatory softening.[2] The deformity is not angular, but is usually a total kyphosis, the column being bent forward from above and made into a single curve. Lateral curvature may occur. In many advanced cases and in some comparatively recent cases the spine becomes rigid and ankylosed, and when it does, there may be evidences of irritation of the posterior nerve-roots. In this condition there is rigidity of part or of the entire spine, other joints escaping. If the entire spine is involved, there is rigid cervicodorsal kyphosis, a condition which causes the neck to stick forward and the head to appear as if forcibly driven down between the shoulders. If the entire spine is in-

[1] "Ann. de la Soc. Belge," July 15, 1898.
[2] J. Jackson Clarke's book on "Orthopedic Surgery."

volved, the lumbar spine is rigid and the normal lumbar curve disappears. As a consequence the patient stands in an unnatural attitude, the hips and knees being partly flexed, and the legs and feet being in a condition of external rotation. In Bechterew's disease there are compression of the posterior nerve-roots, severe pain, muscular atrophy, and ascending degeneration of the cord. What Marie calls *spondylitis rhizomelique* is said by Osler to be a form of arthritis deformans. There is rigidity of the spine, shoulders, and hips, but no nervous lesions, as in Bechterew's disease.

**Treatment.**—Cure is impossible, but amelioration can be obtained.

The local and constitutional treatment is as for osteo-arthritis in any region (see page 642). If spinal curvature begins, a mechanical support must be applied or Albee's bone-grafting operation performed. Rugh operated on such a case with gratifying results ("Internat. Clinics," Vol. I, Twenty-third Series).

**Injuries of spinal ligaments and muscles,** which may complicate more serious injuries or may exist alone, are caused by wrenches, twists, and violent muscular efforts (as in lifting). Railway accidents may be responsible for these sprains and strains. The injury is called *railway spine* when it is caused by a railway accident.

**Symptoms.**—Injuries of the back, even without cord injury, are frequently linked with very deceptive nervous symptoms. Symptoms are often severe, but are usually temporary. In some few cases the symptoms are persistent. Secondary disease of the cord is extremely rare. Any region may be affected, but the lumbar is most usually injured, and the entire spine may suffer. The three marked symptoms are pain, tenderness, and stiffness of the back. At the time of injury and for a while after there is often marked shock, and hysterical excitement is occasionally observed. The cardinal symptoms may arise very soon, but may not become severe for a day or two. The pain is not acute when at rest, but becomes acute on movement.[1] The pain is felt in the back and sometimes darts into the extremities. The muscles of the back are rigid, the spasm being due to pain. The patient is very careful not to twist or bend the spine, because to do so increases pain. In a one-sided injury the rigidity is unilateral, and this symptom cannot be simulated. Often, but by no means always, the region of the back is swollen and the skin is discolored. The tenderness is not of the skin, but of the muscles. Firm pressure on a spot of real tenderness causes rapid pulse (*Mannkopf's sign*). The vertebral spines are regular and are not mobile. There is no distant paralysis or hyperesthesia unless the cord is damaged (though in some rare cases the bladder and the rectum are paralyzed when no cord lesion can be detected, and hyperesthesia may exist over the spines). Moullin tells us that the extremities feel weak because they are deprived of proper support on account of the immobility of the muscles of the back. For the same reason the action of the abdominal muscles is interfered with, and the power of micturition and of defecation is impaired (there are constipation and difficulty in emptying the bladder).

The **treatment** of recent injuries comprises rest, the application of an ice-bag, and leeching over the painful area. After a day or two hot fomentations, tincture of iodin, compression by adhesive strips, and inunctions of ichthyol and lanolin are used; and, later still, massage, douches, and frictions with a stimulating liniment are employed. Phenacetin helps to relieve pain, though in some cases opium is temporarily necessary.

**Traumatic neurasthenia** is apt to arise after the *immediate* effects of the accident subside. In this condition the patient grows tired easily and complains of pains and aches in the back and loins, interfering with or preventing

[1] Moullin on "Sprains."

work; paresthesia and numbness exist in the extremities; in many cases sexual intercourse is impossible because of premature ejaculation or of incapacity for erection. There are dyspepsia, eye-strain, insomnia, loss of memory, rapid and irregular pulse, cardiac palpitation, and mental depression or confusion. The reflexes are usually exaggerated, but they can be exhausted more easily than can the exaggerated reflexes of organic cord disease (because of irritable weakness). Some rigidity and tenderness exist in the back, and the skin over this region is often hyperesthetic. Attacks of retention of urine may occur. Hypochondriasis is not unusual.

*Treatment of Traumatic Neurasthenia.*—Employ rest, tonics, massage, douches, and frictions to the back. Secure sleep, and endeavor to bring about a gain in weight. If sexual incapacity or seminal emissions worry the patient, dilate the urethra with steel sounds.

**Traumatic hysteria** develops only in those predisposed by a neuropathic hereditary tendency; traumatic neurasthenia may arise in any one. In the first named disease the accident is only the *exciting* cause; in the second disorder it is *the* cause. Many cases of so-called "railway spine" are really examples of traumatic hysteria. Traumatic hysteria and neurasthenia may be associated. Neurasthenia is a condition of exhaustion associated with a number of chronic disorders; it forms a foundation on which hysteria is apt to build its structure. The structure of hysteria is made up of morbid impressionability, hyperesthesia of centers, lowered self-control, and sensitiveness of the peripheral nervous system. The accident plays a double part in producing traumatic hysteria—first, by its effect on the mind (psychical traumatism); second, by its effect on the body, which anchors the attention to one point. An area of pain or stiffness often serves as an autosuggestion which undergoes morbid magnification when viewed through the distorting medium of hysteria. Erichsen taught that the symptoms of what he named "railway spine" arose from inflammation of the cord and its membranes, a view now abandoned. A blow given to a hysterical person causes a feeling of numbness, and thus negative sensation from local shock may establish the idea of paralysis, or traumatism, acting as a suggestion, may inhibit motor representations and destroy the normal ideas of motion and feeling (Charcot and Pitre). Terror always causes a feeling of loss of power in the legs, and the terror of the accident may thus develop the idea of paraplegia. The site of a traumatism may localize symptoms; for instance, a blow upon the eye may cause amaurosis or blepharospasm. It is important to remember Charcot's saying that a hysteria long latent and unrecognized may be awakened into obvious activity by a blow or an accident. Pitre shows the same to be true of epilepsy. A not unusual lesion is hysterical traumatic monoplegia, not coming on at once after the accident, but usually some days afterward, and presenting flaccid muscles, the electrical reactions and reflexes remaining normal, but the muscular sense being lost (Pitre). The muscles usually waste. The skin of the paralyzed limb is anesthetic or analgesic. There may be anesthesia limited to a limb, hemianesthesia, or general anesthesia.[1] Hysterical paralysis is usually associated with the permanent *stigmata of hysteria*—concentric contraction of the visual field, pharyngeal anesthesia, convulsive seizures, and hysterogenic zones (Clarke and Pitre). The permanent stigmata may be latent. Hysterical phenomena lack regularity of evolution, and they may be produced, altered, or abolished by mental influences or by physical forces which produce no effect on organic disease. In most hysterical conditions the general health is not profoundly impaired.[2] In making a diagnosis of hysteria we must be most careful to

[1] J. Mitchell Clark, in "Brain."

[2] Read the works of Thorburn and Pitre.

exclude both malignant disease and imposture, because hysteria is a sort of diagnostic waste-basket into which we cast most things which we fail to understand. Babinski proposes that we should call phenomena hysterical only when they can be produced or can be cured by suggestion ("Brit. Med. Jour.," Jan. 23, 1909, p. 234). Brain tumor may cause hysterical symptoms.

*Treatment* is by moral means chiefly. Gain the confidence of the patient. Suggestion is of great value. In many cases separation from family and friends is necessary and isolation is desirable. The Weir Mitchell rest-cure is often the best plan of treatment, and all its details should be carried our faithfully.

**Malingering.**—Persons often pretend to suffer from maladies of the spinal cord or column as a result of accident when no diseases of those parts exist. Some get well upon the rendering of a favorable verdict by a jury (**litigation backs**). In any case always examine carefully, so as to be able to exclude malingering. Note the patient's behavior and motions when his attention is diverted from his disease. *Meningomyelitis* can be excluded if there be no spasm, paralysis, hyperesthesia, paresthesia, or anesthesia at a distance (A. Pearce Gould). If pain has lasted for months; if pressure downward upon the head or shoulders does not increase pain; if the vertebræ are movable and there is no angular displacement, exclude *caries*. Gould states that when there are wasted muscles, when moderate spine movement is painless, but effort in bringing the body erect causes pain in the erector spinæ region, the trouble is a *strain of the erector spinæ muscle*. If the muscle is not wasted and the pain is in bending forward rather than in straightening up, the *vertebral ligaments* are the seat of trouble. Unilateral spasm cannot be simulated. The administration of ether may dispose of a pretended paralysis, the patient moving the suspected extremity while drunk from the anesthetic.

**Concussion of the Spinal Cord.**—This term has no definite pathological meaning. It is probable that there is such a condition, but it is usually associated with laceration of capillaries and of cord substance.

The **symptoms** are shock, intense pallor, nausea, often vomiting, and sometimes syncope. With this condition special symptoms may be linked—as temporary paralysis, a girdle sensation, numbness and loss of power in the limbs, hiccup, torticollis, coarse tremors, pains in the back and limbs, areas of anesthesia and analgesia—depending on the portion of cord lacerated.

The **treatment** in concussion of the spinal cord is the same as that for sprains. Traumatic neurasthenia and hysteria or organic cord disease may follow this injury.

**Contusion of the spinal cord** may arise from a blow or a sprain, but it is usually due to extreme flexion of the spine. It causes hemorrhage into the gray matter of the cord (*hematomyelia*). The symptoms are motor and sensory palsy and diminished reflexes. Some cases recover, but others end in myelitis.

**Wounds of the spinal cord** are rare and are very dangerous. A knife is sometimes thrust in between the occiput and atlas. Wounds above the origin of the phrenic nerves cause almost instant death. Gunshot-wounds are the most usual form, the cord being damaged by the bullet and by bone-fragments.

In the American Civil War gunshot injuries of the cord were very rare ($\frac{1}{4}$ of 1 per cent. of all wounds), but at the present day in war they represent more than $\frac{1}{2}$ of 1 per cent. of all wounds, the increase in frequency being due to the increased penetrating power of the modern military bullet (Surgeon-General Robert M. O'Reilly, in "Keen's Surgery," vol. iv). The mortality is about 60 per cent.

A revolver bullet or a small-caliber bullet fired at long range may produce vertebral fracture without cord injury, and any bullet may fracture a process

of a vertebra without cord injury. If the laminæ are fractured the cord is almost sure to be injured. The cord may be concussed, lacerated, or cut across by bone or bullet, or compressed by bone or blood. The bullet may lodge or may perforate.

**Treatment.**—In a suspected wound of the cord perform exploratory laminectomy, arrest hemorrhage, and if the cord is divided, suture it. If a bullet is lodged, remove it.

**Compression of the spinal cord** may be due to blood or to inflammatory exudate, as well as to displaced bone (see page 844). *Compression from blood* may be due to *extramedullary* hemorrhage or to *intramedullary* hemorrhage. *Extramedullary* hemorrhage causes sudden pain in the back, the pain radiating from compressed nerve-roots; hyperesthesia and paresthesia in the area of the radiated pain; spasm of muscles supplied by the compressed nerves, sometimes of muscles whose nervous supply is below the lesion; tremors; convulsions; retention of urine; paralytic symptoms following the signs of irritation, but no absolute paralysis (Mills). A girdle sensation is usual. *Intramedullary* hemorrhage causes pain, a girdle sensation, abolition of reflexes, and paralysis. Spasms, rigidity, and paralysis come on early. Bedsores may form, and retention of urine and incontinence of feces may be observed. Paralysis from hemorrhage is rapidly progressive from below upward (*crawling paralysis*). Compression from extramedullary hemorrhage may be recovered from without operation, and in some cases recovery is rapid.

**Treatment.**—If paralysis from spinal bleeding extends rapidly and life is endangered through the probable involvement of a vital center, perform a laminectomy, remove the clot, and arrest hemorrhage. It is wise always to open the dura and inspect the cord. Extramedullary hemorrhage may be arrested by sutures or by packing. Intramedullary hemorrhage may be arrested by suture-ligatures or by packing. If an extramedullary clot is extensive it is proper to make a second laminectomy near the lower end of the spinal column in order to permit the surgeon to wash it out thoroughly. The dura must be sutured and drainage is to be employed. If there is paraplegia, complete anesthesia of the paralyzed parts, and entire abolition of the deep reflexes, operation is probably useless, but it is justifiable to try it because of a possibility that the cord is not completely divided. In some cases with persistent paraplegia the operation should be undertaken. If operation is not undertaken, have the patient lie upon his side, apply a spinal ice-bag, and give morphin hypodermatically. If hemorrhage continues in the cord and if the patient be plethoric, perform venesection. To promote absorption of the clot and exudate give a combination of carbonate and acetate of ammonium, order pilocarpin, and employ spinal galvanism and hot douches. Iodid of potassium may be given.

**Fractures and dislocations of the spine** are very rare. The spinal regions most liable to injury are the atlo-axial, the cervicodorsal, and the dorsolumbar (Treves). A vertebra may be fractured alone, but dislocation without fracture, except in the upper cervical region, very rarely occurs. These two lesions, dislocation and fracture, are so often associated that the term *fracture-dislocation* is used by many surgeons to include them both. The causes of fracture and dislocation are direct force (seldom) and indirect violence (commonly). In fracture by direct force the laminæ and spinous processes are most apt to suffer. In most cases the fragments are not greatly displaced. A fracture by indirect force may result from a fall on the shoulders, from a weight falling on the shoulders, or from a fall on the buttocks. Forced flexion or overextension is the commonest cause. In fractures from indirect force the cord generally suffers. In some cases the displacement of the vertebræ lacerates the cord, the vertebræ return into place, and no deformity is

detectable. Fracture-dislocation from direct force may occur at any part of the column, and in this accident the posterior vertebral segments are driven together, and the cord, as a rule, escapes injury. Fracture-dislocations from indirect force most commonly happen in the dorsolumbar region, but are met with in the cervical and dorsal regions. In the cervical region reduction can usually be secured, but in the lumbar region reduction is impossible.

**Symptoms.**—In fracture-dislocation great displacement is unusual, but some is almost always recognizable (irregularity of the spines or angular deformity). There are pain (which is increased by motion), tenderness, ecchymosis, and motor and sensory paralysis. Priapism, cystitis, and retention of urine often occur. Horsley has pointed out that in many cases paralysis passes away only to recur subsequently, the recurrence being due to

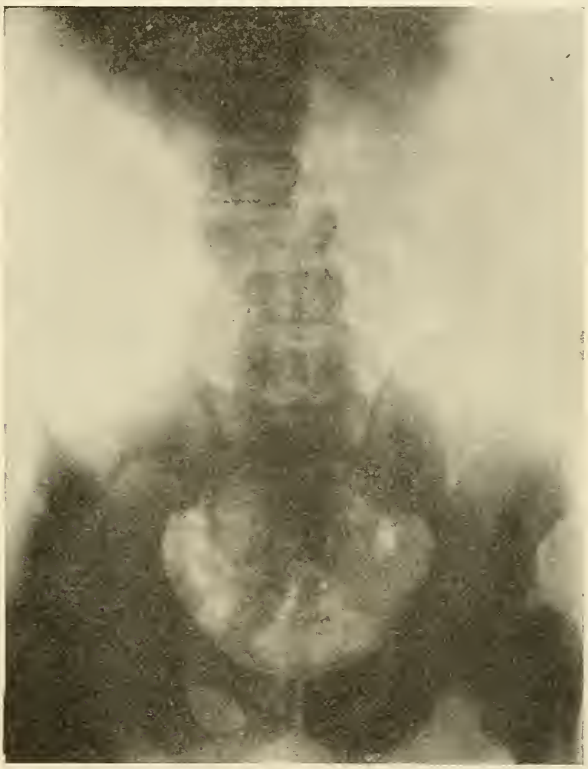


Fig. 544.—Fracture of third lumbar vertebra.

edema of the cord. In some cases of spinal injury there is *temporary paralysis* due to *shock*. *Persistent paralysis* may be due to laceration of the cord, division of the cord, or compression of the cord by bone, blood-clot (Fig. 545), or products of inflammation. The extent of paralysis depends on the seat of the cord injury. We must always try and decide if the spinal cord is completely divided or hopelessly crushed (Fig. 546). When the symptoms are not immediate in onset; when all the muscles below the seat of injury are not completely paralyzed; when there is some retention of sensation; when reflexes are present and muscular rigidity exists, we may be sure that the cord is not completely divided. When the cord is completely divided the symptoms are immediate, there are absolute flaccid motor paralysis and complete sensory paralysis (loss of appreciation of pain, touch, and temperature). The

line of anesthesia is definite and suddenly terminates (Walton). The bladder and rectum are paralyzed and there may be priapism. All the reflexes, superficial and deep, except, perhaps, the plantar have disappeared. There is pain, there are no muscular spasms, there is vasomotor paralysis with sweating of the paralyzed parts, and the symptoms persist and do not vary (J. J. Thomas, in "Boston City Hospital Med. and Surg. Reports"). There is usually tympanites (Walton). If this latter symptom-group is due to shock, it will usually be temporary, but occasionally, even when so caused, it persists some considerable time. It is also probable that concussion of the cord may in some cases simulate complete

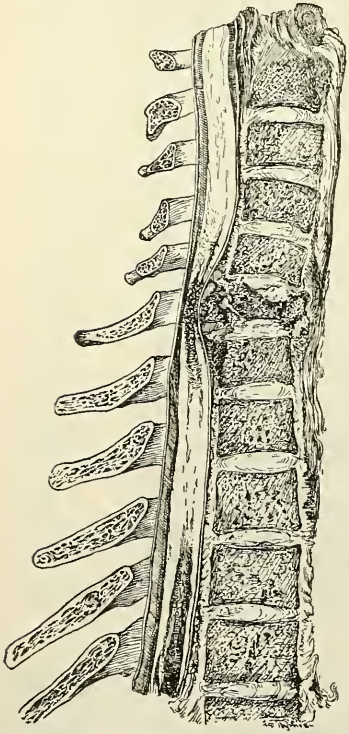


Fig. 545.—Fracture of the cervical spine, cord compressed by bone and blood. Hemorrhage into the cord at the seat of the lesion and below the lesion (Warren Museum). (From Scudder's "Treatment of Fractures." Drawn by Byrnes.)

Fig. 546.—Spine sawed. Fracture of the spinous processes of the seventh cervical and first and second dorsal vertebræ. Fracture of the bodies of the fifth, sixth, and seventh cervical vertebræ with displacement *backward* of the upper fragment. Total crush of the cord. The section passes a little to one side of the cord, which is seen in place, and the staining of the cord by hemorrhage into its substance shows plainly through the membranes even in photograph. The spinous processes of the second and third dorsal vertebræ were found fractured at the operation, and were removed (Thomas).

division. As Walton says, no symptoms *prove* a hopeless crush of the cord: it is the persistence of the symptoms which does prove it ("Jour. Nervous and Mental Diseases," Jan., 1902); I would add, the *unchanging persistence* of the symptoms proves it.

A. J. McCosh ("Jour. Amer. Med. Assoc.," Aug. 31 and Sept. 7, 1901) points out that definite pressure is indicated by marked symptoms and absence of reflexes. When there is not definite pressure the symptoms are irregular; there is incomplete palsy, or muscles of the same group show different degrees of paralysis; anesthesia is partial; signs of irritation are not distinct, and there are patches of hyperesthesia and zones of paresthesia. If in doubt at the end of twelve hours, perform an exploratory operation.

Fig. 547.—Fracture of the odontoid process of the second vertebra and dislocation between the first and second vertebra a number of years after injury. (From *X*-ray Dep't. of Jefferson Hospital.)

The **prognosis** depends on the amount of damage done to the cord. Fracture-dislocations in the cervical region produce obvious deformity, stiffness of the neck and irregularity of the spines, and a displaced vertebra may occasionally be detected by a finger in the pharynx. Crepitus can rarely be detected unless a spinous process is fractured. The Röntgen rays aid diagnosis immensely. The seat of cord injury may be determined by a study of the palsy and other symptoms.

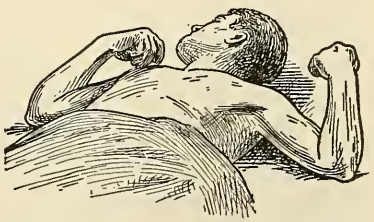


Fig. 548.—Lesion of spine between fifth and sixth cervical vertebræ. Note position of arms, due to paralysis of subscapularis. Brachialis anticus, supinator longus, and deltoid muscles intact. Elbow flexed, shoulders abducted and rotated outward (after Thorburn).

Fracture-dislocation of the atlas or axis usually causes instant death. When the displacement is only trivial, the patient may actually recover, but will probably die of secondary cord disease. Dr. N. J. Blackwood of the U. S. Navy records a case of fracture of the atlas and axis and forward dislocation of the occiput on the spinal column, life having been maintained for thirty-four hours and forty minutes by artificial respiration, during which time laminectomy was performed on the third cervical vertebra ("Annals of Surgery," May, 1908). Lofton has recorded a case of recovery after dislocation of the anterior arch of the atlas on to the odontoid process ("New York Med. Jour.," April 18, 1908). Fig. 547 exhibits a case which recovered after fracture of the odontoid and dislocation between the first and second vertebræ. In

injury of the third cervical vertebra the phrenic nerve is involved, the diaphragm is paralyzed, and death soon occurs. In fracture-dislocation of the fifth cervical vertebra the subscapularis muscles are paralyzed, but the biceps, brachialis anticus, supinator longus, and deltoid muscles escape, and the patient assumes a characteristic attitude (Fig. 548). In Jones's case of fracture of the fifth cervical vertebra no operation was performed, but the patient partly recovered and became able to walk, but with a spastic gait ("Lancet," Nov. 28, 1903). If the sixth cervical vertebra is dislocated there is palsy of the muscles of the hand. In injuries below the sixth cervical vertebra no muscle of the arm, forearm, or hand is paralyzed at first, although after some days paralysis may develop. Damage to the cord above the sixth cervical vertebra produces anesthesia of the body below the injury and of the entire upper extremity except the shoulder. In injury just above the upper level of the seventh cervical there are body anesthesia and anesthesia of the outer surfaces of the arms and ulnar margins of the forearms and hands. In any cervical injury there are body anesthesia and diaphragmatic respiration, and in cases without paralysis of the arms there is sure to be pain. Injuries of the dorsal spine can be accurately located. There is paralysis of motion and sensation up to, or almost up to, the seat of injury. The arms are not paralyzed. Very great pain in the legs occurs if the lumbar enlargement is involved. In injury of the twelfth dorsal or upper lumbar vertebræ there are paralysis of the bladder and rectum, incomplete anesthesia, and partial motor paralysis of the limbs.

**Treatment of Fracture-dislocations.**—When dislocation of the body of the vertebra obviously exists the surgeon may attempt reduction by extension and rotation. The maneuver is very dangerous in the cervical region, and, as deaths have happened, some eminent surgeons advise against reduction when the injury affects that region. Walton's plan for a unilateral cervical dislocation is as follows: Give the patient ether and hold him erect and sitting on a chair. The surgeon stands behind the patient and holds the head with both hands. The first motion is a slight degree of rotation to carry the dislocated process forward and "unlock" it. The head is then rocked toward the sound side and somewhat backward and finally the process is replaced by rotation. No force is used (Clopton, in "Interstate Med. Jour.," Jan., 1908. Quoted in "General Surgery," by John B. Murphy, 1909). After reducing a fracture-dislocation of the cervical region, place the patient in bed, elevate the head of the bed a few inches, and immobilize the neck and head. In fracture-dislocation of the dorsal or lumbar region the traditional plan is to straighten the spine, gently if possible, and to put the patient upon his back upon a water-bed or upon air-cushions. Empty the bladder every six hours with a soft catheter, which is kept strictly aseptic. Take every precaution to prevent bed-sores. Some surgeons advocate reduction of the deformity by extension and counterextension, and the application of a firmly fitting but removable jacket with the suspension collar (as used in Pott's disease). If this plan is employed, the head of the bed is raised and the collar is fastened to it. Every day extension is made gently—from the shoulders in dorsolumbar fracture and from the chin and occiput in cervical fractures. Extension may be maintained permanently until cure. Surgeons have come rather slowly to a belief in laminectomy. One deterrent factor has been the high mortality: Lloyd collected the records of 159 operations and found that 59 patients died almost at once and 39 died later. In Lloyd's collection of 185 cases there were but 24 recoveries and 40 improvements. In 82 immediate operations only 5 recovered. In 103 late operations there were 19 recoveries (John B. Murphy, in "Surg., Gynecol., and Obstet.," April, 1907). Some employ purely expectant treatment in vertebral fractures. My own

feeling is that when simply a spinous process or some other part is fractured, and there are no cord symptoms, we may treat the patient expectantly, following Burrell's advice, and fixing the patient in bed on a Bradford frame and having him carefully nursed and watched. Reduction by extension and counterextension is dangerous and unjustifiable if there is marked kyphosis and if cord symptoms exist. I agree with Burrell that it should only be done if operation is refused, or if there are no cord symptoms and no marked kyphosis ("Annals of Surgery," Oct., 1905). If it is attempted it must be done slowly and as gently as possible because it may cause grave or even irreparable damage to the cord. I fear to delay, and, with Burrell, Lloyd, Walton, and others, operate when the patient recovers from shock, if there seems to be even a gleam of hope that operation may help him. To wait when pressure exists means that during every hour of delay the pressure is damaging the cord. Another reason for operating is that we cannot know the condition of the cord without direct inspection. The operation to be performed is laminectomy. As before stated, this is to be done even if we suspect division or hopeless crush of the cord. In some cases, it is true, we may commit the error of operating when there is only concussion, but such a mistake is less grave than to fail to operate when there is bone-pressure or hemorrhage. An objection filed by the neurologist against laminectomy is that portions of cord above and below the level of the fracture may be damaged (see Fig. 545), but, as Lloyd says, this fact does not forbid operation, but renders it necessary to make a wider exploration than has been the custom. In many cases after prompt laminectomy we get some considerable improvement, and this improvement may be sufficient to enable a man to earn a living. It is true that statistics would indicate that late operations have been more successful than early ones, but these figures must be analyzed in the light of the knowledge that many of the fatalities after early operation would have occurred if no operation had been done, and some improvements after late operation would have occurred to as great or a greater degree after early operation. The prognosis of any operation, early or late, is never gratifying, and Thorburn feels no confidence in obtaining improvement except in injuries of the laminæ, hemorrhage, or injuries of the cauda equina, as he says laminectomy in the cervical region is followed by death, and laminectomy in the dorsal region, though not commonly fatal, is seldom followed by recovery of function. Our statistics of early laminectomy will show fewer deaths and fewer useless operations if we do not operate till shock abates. As Lloyd ("Phila. Med. Jour.," Feb. 5, 1902) says: "It is therefore evident that if we operate immediately after the injury we will have failures that should not be charged against the operation itself, and, if possible, we should wait before operating until the question can be settled whether the patient will overcome the shock or will succumb directly to the effects of the injury." All surgeons operate for compound fracture, for hemorrhage, and for cases with marked bone pressure. If early operation were not performed and if pachymeningitis arises, operation is called for.

My own convictions are that if symptoms are significant we should explore as soon as shock has passed away, even if we think it probable that the cord has been divided; and if it is found divided, it should be sutured. If in any case we are in doubt twelve hours after the injury as to whether or not pressure exists, we should explore. If soon after the accident we think pressure by bone exists, we should operate. If the case is improving, we should not operate even if there are pressure signs, unless there is a chance that pressure is due to bone, in which case we should operate. As McCosh says, pressure by blood or inflammatory exudate may pass away; pressure by bone cannot. Even long after an injury laminectomy may be productive of some benefit.

The rather radical views set forth above regarding the advisability of

operating even if the symptoms point to complete division of the cord arose largely from a knowledge of the well-known case operated upon by Stewart for total division of the cord. In a case of gunshot-wound of the dorsal spine treated at the Pennsylvania Hospital by Francis T. Stewart, and reported by Francis T. Stewart and Richard H. Harte ("Phila. Med. Jour.," June 7, 1902), an exploratory incision made three hours after injury showed that the spinal cord was completely divided. There was a fracture of the laminæ of the seventh dorsal vertebra. The spines and laminæ of the seventh and eighth dorsal vertebræ were removed. The bullet-hole was recognizable in the membranes, and the bullet and some bone-fragments were removed. When the dura was opened, the ends of the completely divided dorsal cord were found to be $\frac{3}{4}$ inch apart. Stewart freshened these ends and brought them together with two sutures of chromicized catgut. In this case a considerable degree of restoration of function took place. At the time of the operation, three hours after the injury, there were complete paralysis and absence of reflexes below the seat of injury; but sixteen months later the patient was able voluntarily to flex the toes, flex and extend the legs, flex and extend the thighs, and, while sitting, lift an extended leg from the floor. The movements of the lower extremity became more forcible when reinforced by contracting the muscles of the upper extremity while making them. The patient could stand with one hand resting on the back of a chair, and could get herself from her bed to her chair by sliding. The bowels were under perfect control, and there was no incontinence of urine when she was awake, although there was occasionally some when she was asleep. There were occasional cramp-like pains in the lower limbs. The sense of touch, temperature, pain, and position were perfect all over the previously paralyzed parts. Below the knee the localization of sensation was not so accurate. There was a slight amount of muscular rigidity; and on each side, an ankle and patella clonus, which was easily exhausted. When the sole of the foot was tickled, the big toe flexed, the thigh abducted, and there was slight contraction of the anterior tibial, the hamstring, and the tensor vaginæ femoris muscles. There were no reactions of degeneration and no trophic changes. There had never been any bed-sores. George Ryerson Fowler ("Annals of Surgery," Oct., 1905) operated on a gunshot-wound of the dorsal spine eleven days after the injury. He removed the laminæ of the tenth, eleventh, and twelfth dorsal vertebræ and found the cord divided, the bullet lying between the severed ends. A piece of dura $\frac{1}{8}$ inch wide was intact. The bullet and blood-clot were removed. The cord was sutured by three sutures of chromicized gut, which included the dura, and more sutures were taken through the dura only. The ends of the cord were easily approximated. The patient recovered from the operation. Twenty-six months later voluntary motion was found to be practically lost in the area below the injury, although when supported by the hands he could stand and when in a frame could move a little by a swinging movement. He is able to tell when his bowels or bladder are about to move, and, if furnished promptly with a utensil, does not soil himself. When asleep, he passes urine involuntarily. Both legs exhibit spastic rigidity, but there are no reactions of degeneration. Patella reflex on each side exaggerated. Ankle clonus is found on one side, but not on other. There is complete anesthesia of the affected area, except in a region 5 inches in length on the outer side of the right thigh. Touch is appreciated, but not correctly localized. In connection with the foregoing important cases we would note that Dr. Estes, of Bethlehem, has also operated upon a case of complete division of the spinal cord, in which suturing was apparently followed by some restoration of function.

In the light of these positive reports we must ask ourselves if we have not been wrong in the view that the spinal cord cannot regenerate. If there

is even a chance that we have been wrong, we must reverse our former conservative treatment and follow a radical plan. The 3 cases strongly suggest the possibility of some regeneration, but do not prove it. The cord may have appeared to be completely divided and yet minute undivided bundles may have escaped recognition. Again, as Fowler suggests, there may be a nerve anastomosis through uninjured portion of the dura or between adjacent nerve-trunks which arise above and below the lesion. At my request Dr. Samuel Lloyd, of New York, kindly wrote me a personal communication setting forth his views on this important subject. They are as follows: "The question of the regeneration of the spinal cord after traumatism of the spine deserves careful consideration in all cases that are operated upon. Up to the present time, however, although a number of operators have reported improvement following suture of the spinal cord in these cases, a careful analysis does not substantiate the fact that that improvement is due to an actual regeneration. It is a recognized fact on the part of all who have had experience with the surgery of the spinal cord that in almost every instance a certain amount of improvement is noted during the first few months. This is probably due to the fact that at the time of the injury minute hemorrhages occur into the adjoining segments, and that pressure is also increased in those portions of the cord by the inflammatory exudate and edema. Within a short time after the injury these conditions improve, and there seems to be an improvement in function; but in every case of spinal suture yet reported the amount of improvement may be explained by these facts. In no instance has there been a complete recovery of function, but in every one there has remained more or less permanent disability. This, however, should not discourage attempts at spinal suture, and in every case operated upon the dura should be opened and the condition of the cord examined. In those cases where a complete destruction has occurred and where the extent of it is not over $\frac{3}{4}$ inch, it may be possible to cut out the lacerated portions and coaptate the surfaces by a series of sutures placed in the dura. In all these cases the patient should be put up in a plaster retaining bandage in extreme extension, even the head being thrown back so as to relax as much as possible the tension on the line of suture. The operator should be very sure, however, that there are no undestroyed fibers traversing the lacerated area, for the destruction of these in case regeneration did not occur would increase the amount of paralysis." With the views of Lloyd I am in entire agreement, and now I always follow this plan, bearing in mind that it is often impossible to tell whether the spinal cord is completely divided or seriously damaged without examining it, and it can be examined only by exploratory operation; therefore, if the serious symptoms already indicated exist after shock has passed away, exploratory operation should be performed; if pressure exists, it should be removed; and if the spinal cord is found to be completely divided, it should be sutured. It is well to remember that Abbe's experiments have shown that there may be great difficulty in bringing the divided ends of the cord into apposition. In order to effect this it may be necessary to resect a vertebra.

**Operations on the Spine.—Operation for Spina Bifida.**—A. W. Mayo Robson[1] maintains that operation is not demanded when the sac is of small size and is well protected by sound integument; that operation is improper when a large portion of the column is fissured, or when paraplegia or hydrocephalus exists; that operation is advisable only in meningocele, in cases in which the integument is thin and translucent, in cases in which the cord is flattened out or the nerves are fused. Robson has closed the osseous defect by transplanting periosteum.

[1] "Annals of Surgery," vol. xxii, No. 1.

Surround the sac by elliptical incisions. Find the neck of the sac, and if it contains no visible nerves, ligate it and cut off the protrusion. Push the stump into the canal. Freshen the bone-margins and spring a piece of celluloid beneath them to close the gap (Park). Suture over the stump with small sutures of catgut.[1]

**Treves's Operation for Vertebral Caries.**—(See page 695.)

**Laminectomy.**—The patient lies prone and a sand-pillow is placed under the lower ribs. Make a vertical incision over and down to the vertebral spines, the middle of the incision corresponding to the seat of injury or disease. The sides of the spinous processes and the laminæ are cleared. The periosteum is incised in the angle between the laminæ and spines, and is lifted away from the arches. It is my custom to bore through a lamina on each side of a spinous process by means of Hudson's burrs. When this has been done the spinous process and lamina are easily bitten through and removed. The usual method of operating is as follows: The spinous processes are cut off close to their bases by means of bone-cutting forceps, the laminæ are removed on each side with the same instrument or the rongeur, and the dura is exposed. In some cases of fracture fragments will be found on exposing the vertebra, or a blood-clot will be seen between the dura and the bone; in other cases the dura must be opened by scissors vertically in the middle line while it is grasped by mouse-toothed forceps. After reaching and removing the compressing cause, or after failing to find or remove it, it is best not to close the dura completely, because, if we do so, cord pressure may result from hemorrhage. The dural wound is left open or is partly closed. I used to insert a drain of rubber tissue, but have given it up. Horsley shows that it is not necessary, and if we refrain from draining we lessen the tendency to headache, temporary pyrexia, and rapid pulse, which frequently follow laminectomy. The superficial parts are stitched with silkworm-gut and dressings are applied.

**Albee's Method of Bone-grafting for Pott's Disease of the Spine.**—In 1891 Hadra, of Galveston, advocated the treatment of Pott's disease of the spine by wiring the spinous processes of the diseased vertebræ to adjacent vertebræ for the purpose of securing fixation. Chipault, in 1895, and Calot, in 1896, did this after forcible correction of angular deformity. Lange buried steel wires on each side of the spine and anchored each one at each end by silver wire (J. T. Rugh, in "Internat. Clinics," Vol. I, Twenty-third Series). The object of these operations was to do away with the necessity for an external supporting jacket or brace. The objection to them was the introduction of a foreign material, and the fact that strain caused that material to cut through the tissues and permit relaxation.

In 1911 both Hibbs and Albee, working independently, reported methods for producing fusion of the arches of the vertebræ.

Hibbs ("New York Med. Jour.," May 27, 1911, and "Annals of Surgery," May, 1912) takes strips of the periosteum from the spines and laminæ, transposes the spinous processes, and sutures the periosteum and the supraspinous ligament over these processes. The periosteum is depended upon to produce new bone, which fuses the parts together. This operation, if successful, lessens kyphosis and produces fusion of spines and laminæ. Albee, doubting the reliability of the periosteum as a bone producer, practises bone-grafting. The patient is placed prone. An incision is made to expose the spines of the diseased vertebræ and also one or two vertebræ above and one or two below. Some make a straight incision. Rugh makes a curved incision at one side of the spine and turns back a flap. Each spine is split vertically by a chisel and each split portion is broken and pushed over to the same side. The interspinous liga-

[1] A full consideration of the various plans of operating will be found in an article by Marcy, in "Annals of Surgery," March, 1895.

ments are cut so as to correspond to the split in the spines. We thus obtain a wedge-shaped incision through bone and ligament.

The length of the cut is measured on a probe. The wound is packed temporarily. The leg is flexed upon the thigh. An incision is made over the anterior surface of the tibia. A wedge-shaped graft of bone is cut by means of a surgical engine, a Gigli saw, or a chisel and mallet. The graft is covered with periosteum on one side and contains some medullary tissue on the other. It is $\frac{1}{3}$ inch thick at its base. This graft is fitted to the unbroken sides of the spinous processes. If the kyphosis is marked, numerous cross-cuts are made with a saw into the thin edge. Thus the graft will be made flexible. No attempt is made to forcibly correct the deformity. Here and there the periosteum is incised to favor the emergence of osteoblasts. The interspinous ligaments are sutured over the graft by sutures of kangaroo tendon. The incision is closed. The patient is put on a Bradford frame or plaster of Paris is applied. The patient remains recumbent for from six to twelve weeks, then he is allowed to sit up and soon after to walk, of course wearing a brace or plaster.

The brace or jacket is removed in five or six months. The results of this operation are excellent. The fate of the bone-graft is a matter of dispute. Some hold that it really lives. Others hold that it is simply a scaffold for new bone. One thing is certain—it strongly stimulates the production of new bone from the raw surfaces of the split spinous processes. New bone comes, and new bone, however formed, constitutes the permanent splint (Albee, in the "Post-Graduate," Nov., 1912, and in "N. Y. Med. Jour.," March 9, 1912; J. Torrance Rugh, in "Monthly Cyclopedia and Medical Bulletin," Feb., 1913, and in "International Clinics," Vol. I, Twenty-third Series).

**Puncture of the spinal meninges,** or **lumbar puncture,** was devised by Quincke, and has been carefully tested by many surgeons. It is the operation for withdrawing cerebrospinal fluid from the subarachnoid space of the cord. It is employed as a means of diminishing cerebral pressure in hydrocephalus, cerebral tumor, uremia, and tuberculous meningitis, but in these cases it has proved of little or only of temporary therapeutic value. It may be of some service in cerebrospinal meningitis. The condition of a patient with a fracture of the base of the skull is sometimes temporarily improved by the operation. Pain is often temporarily relieved. In the performance of a brain operation the brain may bulge so that the dura cannot be sutured. Lumbar puncture makes suturing possible. Puncture is the preliminary step of spinal anesthesia. In some cases the examination of the fluid has been of great diagnostic value. The fluid is not only subjected to a naked-eye study, it is also studied microscopically and bacteriologically. If the fluid from the puncture gives no positive finding, the operation should be repeated (Lorgo). When a diagnostic tap is made we must know the appearance, nature, and pressure of the fluid normally.

Normally the fluid is clear, transparent, alkaline, and under a pressure of from 40 to 60 mm. of mercury (Dana) its specific gravity is from 1.006 to 1.008, and from 5 to 10 c.c. will flow out at a tap. It contains a very few endothelial cells and leukocytes. In cases of increased tension it flows out more forcibly, rapidly, and profusely (brain tumor, hydrocephalus, meningitis, and some infectious conditions). When there is meningeal inflammation the specific gravity is increased. In apoplexy and other hemorrhages beneath the cerebral arachnoid, in fracture of the base of the skull, and in hemorrhage beneath the arachnoid of the cord, the fluid contains blood. Laceration of the brain tissue without subarachnoid or ventricular hemorrhage does not make the fluid bloody. The fluid is turbid in purulent meningitis of the cord or brain, may contain many polymorphonuclear leukocytes, and also bacteria. Lumbar puncture is of great diagnostic use in the cerebral hemorrhage of the newborn and in some fractures of the base of the skull.

The *chemical* study of the fluid is sometimes of value.

In intracranial tumor, purulent meningitis, subarachnoid hemorrhage of the brain or cord, and apoplexy albumin is increased. The normal fluid contains a carbohydrate substance resembling glucose. This is absent in meningitis and is increased in saccharine diabetes.

In uremia the chlorids are diminished.

*Cytodiagnosis* (a microscopical study of the cells of the fluid) may furnish useful information; numerous polymorphonuclear leukocytes are found in meningitis. Lymphocytosis suggests a tuberculous lesion rather than an acute meningeal inflammation. Lymphocytosis occurs also in syphilis of the brain and cord, locomotor ataxia, paresis, and uremia.

*Bacteriologic* study by cover-glass preparations or cultures may give important information. In over 75 per cent. of cases of tuberculous meningitis of the brain membranes the fluid contains bacilli. Stadelmann has reported 37 cases in which tubercle bacilli were found in the fluid.[1] In tuberculous meningitis the fluid may or may not contain tubercle bacilli. In cerebrospinal meningitis the cerebrospinal fluid contains the meningococcus. In this disease diagnostic puncture is unnecessary if the nasal mucus contains the Diplococcus intracellularis. The operation of lumbar puncture is simple, and if done with proper precautions is harmless. The back should be carefully sterilized and thorough asepsis must be preserved in every detail. The patient may lie on the right side with the left knee well drawn up, may lie prone, with a pillow under the belly, or may sit in a chair, with the body bent forward. The site of the intended puncture may be frozen with ethyl chlorid, but no general anesthetic is required. A Pravaz syringe is employed. The needle, which should be 3 inches in length, is guarded by the surgeon's index-finger and the point is inserted $\frac{1}{2}$ inch to the right of the median line and between the third and fourth lumbar vertebræ. It is pointed upward and a little inward under a spinous process. It enters the canal in the middle line. In a child the needle enters the canal at a depth of from 2 to 3 cm.; in an adult, at a depth of from 4 to 6 cm. The fluid is permitted to fall drop after drop into a sterile test-tube. In some cases only a few drops of fluid can be obtained; in other cases many cubic centimeters may be removed. It is not wise to draw for diagnostic purposes over 5 c.c. from a child and 10 c.c. from an adult. If we evacuate too much cerebrospinal fluid, the ventricles are emptied and compression of the cerebellum may arise. The flow should be spontaneous and suction ought not to be used. Sometimes nausea, vertigo, and severe headache follow the operation, and sudden deaths have been reported. For a number of hours after tapping the patient should remain recumbent.

**The Mingazzini-Foerster Operation in Tabes.**—Mingazzini suggested intradural division of the posterior sacrolumbar nerve-roots for unbearable pain in the lower extremities due to tabes.

Foerster follows a like method in treating gastric crises. He divides the posterior dorsal roots from the sixth to the ninth. Some of these operations have been notably successful. Others have failed. In at least one case the condition was aggravated (Doerr, in the "Wien. med. Woch.," No. 45, 1911). For the pains and crises of tabes some surgeons advise extradural division of the posterior roots (Guleke). The posterior roots may be divided for intractable neuralgia and for athetosis. The roots selected depend upon the seat of pain or the region of athetosis.

**Intradural Root Anastomosis in Cases of Vesical Paralysis.**—Kilvington ("Brit. Med. Jour.," 1907, vol. i) suggested intraspinal anastomosis of nerve-roots. He found by experiments on dogs that the last lumbar root, when joined to the roots of the second and third sacral nerves, gives contraction to a palsied

[1] "Berliner klinische Wochenschrift," July 8, 1895.

bladder. Bird, at the suggestion of Kilvington, did the operation on a human being, but it was a failure. Frazier, in a case of Mills's, anastomosed the root of the last lumbar to the roots of the third and fourth sacral nerves. There was decided improvement (Frazier and Mills, "Jour. Am. Med. Assoc.," Dec. 21, 1912).

**Horsley's Operation for Chronic Spinal Meningitis.**—Sir Victor Horsley ("Brit. Med. Jour.," Feb. 27, 1909) states that during the past ten years he has operated on a number of cases for what he calls chronic spinal meningitis. Such cases are commonly confused with tumor, are much more frequent than tumors, and are often cured or greatly improved by operation. The first published case of this sort was reported by Spiller, Musser, and Martin ("Univ. of Penna. Med. Bulletin," March, 1903). Martin performed laminectomy, found a "circumscribed meningitis," and cured the patient. In these cases a fluid accumulation is found and this fluid is stagnating and under pressure. The cord passes into a condition of sclerogliosis. The symptoms are pain, advancing loss of power in the legs, perhaps slight kyphosis, and eventually progressive and fatal paraplegia (compression paraplegia).

The pain in these cases involves an extensive area, not as in extramedullary tumor a small area supplied by one or two nerve-roots, and there may be hyperesthesia over an entire extremity, which does not occur in extramedullary tumor (Horsley, Loc. cit.). Horsley has never seen absolute abolition of tactile sense. The operation consists in laminectomy, opening the theca, washing it out with mercurial solution (1 : 500 followed by 1 : 2000), and closing without drainage. Bailey and Elsberg ("Jour. Am. Med. Assoc.," March 9, 1912) suggest the term *spinal decompression* for laminectomy and opening of the dura when no lesion is located or found. They believe that in such cases as Horsley describes the laminectomy and opening of the dura do the good. A number of other intradural conditions may perhaps be benefited by the operation.

## XXVI. SURGERY OF THE RESPIRATORY ORGANS

**Asphyxia.**—In drowning, strangulation, suffocation, and hanging the mode of death is by the same process, that is, by asphyxia or apnea. "Asphyxia," though the commonly used designation, is an unfortunate term, and apnea is the more correct one. By asphyxia we mean the non-oxygenation or the incomplete oxygenation of the blood, and yet even in this condition death is not immediate, for the heart may continue to beat for some little time after all breathing has ceased. A man may be apparently dead from asphyxia and yet be capable of resuscitation. In general, it may be said that asphyxia produces lividity, a struggle like a convulsion to obtain breath, and finally, in many cases, genuine convulsions. In the very beginning of non-oxygenation the senses may be remarkably acute, but consciousness is soon lost. The veins stand out and the pulse becomes weaker and weaker. The individual may bleed from the nose, from the rectum and other mucous membranes, and there may be involuntary passage of urine. For a short time the chest heaves, making respiratory attempts, and after a short time the heart stops beating.

Asphyxia is a common mode of death. It is the mode of death occasioned by a foreign substance in the air-passages, by paralysis or by tetanic fixation of the respiratory muscles, by great pressure upon the chest or abdomen, by acute traumatic pneumothorax, by a clot in the pulmonary artery which cuts off the blood-supply of the lungs, by hanging, throttling, drowning, absence of sufficient oxygen from the gases breathed, presence of quantities of irrespirable gases (CO, $CO_2$), or the presence of irritant gases (Cl, $SO_2$) which cause spasm of the glottis.

The terms *smothering*, *stifling*, and *suffocation* mean prevention of entry of air into the lungs in sufficient quantity to properly aërate the blood.

**Treatment in General.**—Of course, each form requires some particular method of care. In general, it may be said that the surgeon should at once endeavor to determine the cause of the asphyxia and should particularly ascertain if it came on gradually or suddenly. As a rule, a gradual asphyxia is due to some intrathoracic lesion. If the asphyxia were sudden the surgeon must examine the neck and chest externally to see if there is any sign of injury. Then the mouth is opened, the tongue pulled forward, and the glottis felt by the finger to see that no foreign material is blocking the air-passages. The patient will do best in a free draft of fresh air, and it may be possible to excite respiratory activity by dashing hot water and then cold water on the face and chest and making a number of these alternate applications. The application of electricity to the phrenic nerve may do good (the anode at the root of the neck and the cathode over the epigastric region). If there is cardiac dilatation, bleeding is urgently indicated. When the patient is breathing, inhalations of oxygen do good. If there is laryngeal obstruction, tracheotomy or intubation should be done. In most cases of threatened asphyxiation artificial respiration is necessary. There are several different methods.

**Artificial respiration** is resorted to in case of suspension of breathing from any cause; among the more frequent causes are the inhalation of smoke or poisonous gases, drowning, profound anesthesia, opium-poisoning, and electric shock.

There are several methods of giving artificial respiration. One method may have an advantage over another in a certain type of case, as will hereafter be described.

Before resorting to any method the clothing about the neck, chest, and abdomen must be free and loose; the mouth unobstructed by foreign bodies, as false teeth, etc., the throat clear of mucus, etc.

If edema of the glottis, malignancy of the tongue, or an immovable foreign body should obstruct the air-passages, tracheotomy should be performed before attempting artificial respiration.

When giving artificial respiration the operator should not desist because respirations are not established in a few minutes, but, on the contrary, should persist (even using relays of men if necessary) for an hour or more. Many cases of drowning, opium-poisoning, asphyxia from smoke, etc., have been resuscitated after an almost incredible period of time, during which time artificial respiration was carried on.

During the manipulations of artificial respiration it should be remembered that the patient must be cared for as in shock: the body kept warm, frictions applied by the hands or rough towels, and massage over the heart be practised. Enemas of coffee by rectum and stimulating hypodermatic injections are important. The author prefers an enema of 5 oz. of hot coffee with 1 oz. of brandy and a hypodermatic injection of atropin sulph., gr. $\frac{1}{100}$, or strychnin, gr. $\frac{1}{20}$. One of the oldest methods of artificial respiration and the one most applicable in children is *mouth-to-mouth inflation*. In this method the operator holds the victim's tongue forward with a suture or with a piece of string tied around the tongue; with the other hand he closes the nostrils, and then, after taking a deep inspiration, blows directly into the patient's mouth. The air is then expelled from the patient's lungs by direct pressure on the walls of the thorax. This procedure is repeated 16 times a minute. Instead of the direct mouth-to-mouth inflation a soft-rubber catheter may be passed through the mouth into the trachea and the patient's lungs be expanded by the operator blowing through the catheter.

Artificial respiration must not be stopped when the patient has taken one or

two breaths, but should be continued until the respiratory movements are regular and normal. The patient must be carefully watched for fear of secondary apnea.

*Sylvester's method* is a popular one, and is probably the best in many cases in severe electric shock, but if there is any fluid in the lungs or air-passages (as in cases of drowning) another method may be preferred.

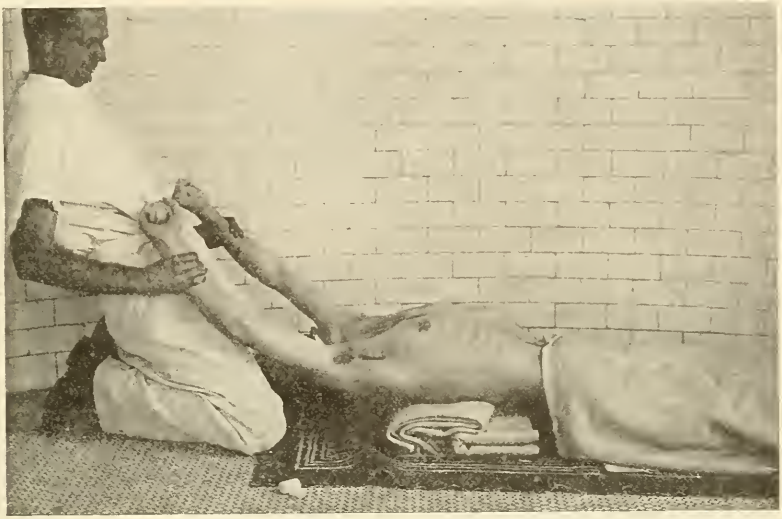


Fig. 549.—Sylvester's method. Inspiration.

To make artificial respiration by this method the patient is placed on his back with a folded coat or blanket under his shoulders. The tongue is pulled forward and held by forceps, a suture, or, in an emergency, may be tied with a

Fig. 550.—Sylvester's method. Expiration.

string, or even a necktie can be used. The operator, kneeling at the head of the patient, grasps the forearms just below the elbows and circumducts the arms outward and upward, meanwhile making traction until the arms are perpendicular to the body (Fig. 549). By this movement the chest is expanded and in-

spiration is caused. The arms are now brought slowly to the sides of the chest and firm pressure is made for two or three seconds, thus forcing the air from the lungs and causing expiration (Fig. 550). This procedure should be repeated about 15 or 16 times a minute.

This method is best suited to those overcome by gas, smoke, or apnea other than that due to drowning.

*Howard's Method.*—This method is now used by the United States Life Saving service in cases of drowning. The procedure as laid down by Dr. Howard is as follows:

"Rule 1: To expel water from the stomach and lungs, strip the patient to the waist, and, if the jaws are clinched, separate them and keep them apart by placing between the teeth a cork or a small piece of wood. Place the patient face downward, the pit of the stomach being raised above the level of the mouth by a roll of clothing placed beneath it. Throw your weight forcibly two or three times upon the patient's back over the roll of clothing so as to press all fluids in the stomach out of the mouth."

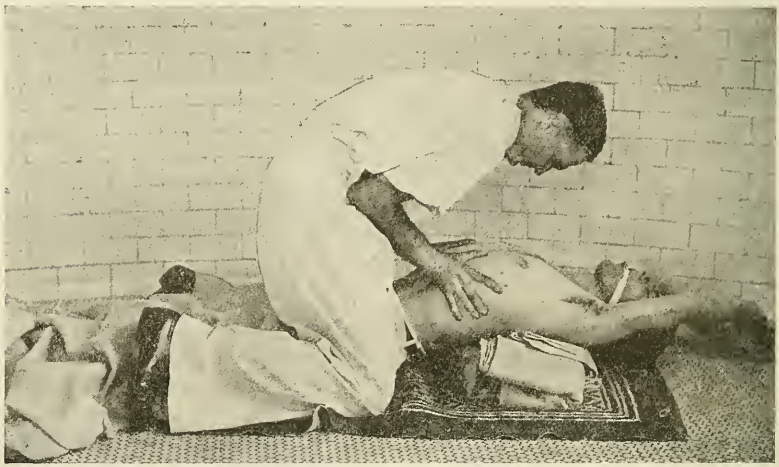


Fig. 551.—Howard's method of artificial respiration.

"Rule 2: To perform artificial respiration quickly turn the patient upon his back, placing the roll of clothing beneath it so as to make the breast bone the highest point of the body. Kneel beside or astride of the patient's hips. Grasp the front part of the chest on either side of the pit of the stomach, resting the fingers along the spaces between the short ribs (Fig. 551). Brace your elbows against your sides, and steadily grasping and pressing forward and upward, throw your whole weight upon the chest, gradually increasing the pressure while you count 'one, two, three.' Then suddenly let go with a final push, which springs you back to your first position. Rest erect upon your knees while you count 'one, two'; then make pressure as before, repeating the entire motions at first about 4 or 5 times a minute, gradually increasing them to about 10 or 12 times. Use the same regularity as in blowing bellows and as seen in natural breathing, which you are imitating. If another person is present let him, with one hand, by means of a dry piece of gauze, hold the tip of the tongue out of one corner of the mouth, and with the other hand grasp both wrists and pin them to the ground above the patient's head."

*Schäfer's Method or the Prone Method.*—In this method, instead of lying on his back as in the Howard method, the patient lies on his stomach, his face being turned to one side. The arms are placed above the head. A roll of

blankets or clothing are placed under the chest. The operator now kneels astride of the patient and grasps the thorax with both hands, the fingers running parallel with the ribs. Brace your elbows against your sides and press firmly inward and upward, throwing your whole weight against the chest. Release your pressure after two or three seconds, count "one, two," and again make pressure as before. This sequence should be repeated about 15 times a minute.

The advantage of this method is that fluid in the air-passages will gravitate out through the mouth and the tongue falls forward without being held.

*Marshall Hall's method* is more easily applied on the operating table than any other method of artificial respiration. On the other hand, it is not as efficient and is only justifiable when the patient's normal respirations are resumed after three or four applications of pressure. The patient lies on his back, and the operator, with a hand on either side of the thorax near the costal region, makes pressure upward and inward. This pressure is continued for two or three seconds, then suspended for the same length of time, and then pressure again made. This procedure is repeated about 15 times a minute.

*Laborde's method* is not as efficacious as the foregoing, but has its uses in cases in which, because of injury to the chest, shoulders, or arms, Sylvester's or Schäfer's method or their modifications cannot be employed.

Laborde's method rests on the assumption that "systematic and rhythmic traction" upon the tongue produces respiratory reflexes and causes contractions of the diaphragm, hence establishing respirations.

The tongue is grasped by tongue-forceps or a piece of gauze held between the forefinger and thumb, and is pulled well out of the mouth with considerable traction. It is held for two or three seconds and then relaxed. This procedure is repeated about 15 times a minute.

When a certain amount of resistance is felt, it is a sign that respiratory function is being restored. Noisy respiration first occurs, termed inspiratory hiccup.

When the condition of the chest will allow, that is, if there are no fractured ribs, empyema, etc., Laborde's method and Marshall Hall's method may be combined.

It can be readily realized that Laborde's method cannot be used when there is disease or injury of the tongue.

**Intratracheal Insufflation.**—This consists in forcing air by external pressure through a tube which passes through the mouth and larynx into the trachea. The air, under the influence of the same force which drove it in, emerges between the tube and tracheal wall. "The air-stream has to be interrupted several times a minute for only about two seconds at a time" (S. J. Meltzer, in "Keen's Surgery," vol. vi). (See Insufflation Anesthesia, p. 1199.)

**Artificial Respiration by the Pulmotor.**—The pulmotor is a most ingenious apparatus. It is for two purposes: (1) To give artificial respiration; (2) to administer oxygen.

Both the administration of oxygen and the production of artificial respiration are accomplished by means of oxygen which is under pressure. This fact makes the instrument especially valuable in cases of asphyxiation from illuminating gas or other poisonous vapors. The apparatus should be a part of the equipment of every modern hospital (for work on the ambulance, in the accident ward, and in the operating room), every gas and electric company, every mine, should be accessible at resorts where bathers congregate in any large number, and should be on the ground at every city fire.

The apparatus is contained in a narrow wooden case which can be carried by one person. The weight of the case with the apparatus is less than 50

pounds. A diagram of the apparatus (Fig. 552) is here given which shows its mechanism: C is a cylinder containing $11\frac{1}{2}$ cubic feet of oxygen, which will keep the apparatus in operation and create artificial respiration for forty minutes; V is a valve which opens and closes the oxygen tank and which is the sole governor of the apparatus.

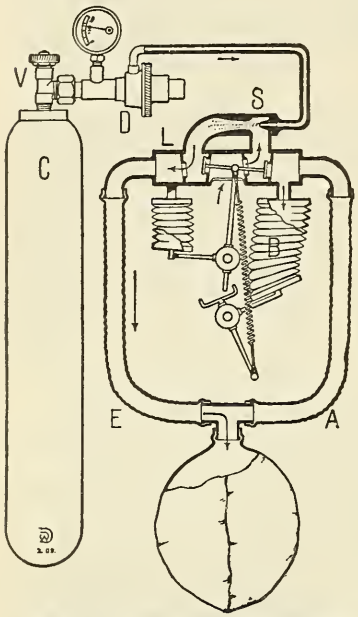



Fig. 552.—Diagrammatic illustration showing the action of the pulmotor. (See text for explanation of letters.)

The mask is fitted on the patient's head as in Fig. 553. The oxygen is then turned on by the valve V. The oxygen passes through the reducing valve D to the injector S, which has the property of drawing in a large volume of air with a certain force of suction, and propelling that air forward with equal force through the flexible tube in front of the injector. This suction and delivery injector therefore serves as a motor, automatically filling the lungs by pressure and emptying them by suction. The suction is accomplished by the leather accordion bellows B, which effects without cessation the automatic reversal of the apparatus from suction to delivery and vice versâ. During inflation the same pressure obtains in the bellows as in the lungs, but as soon as the latter are filled the bellows becomes inflated, and in moving forward throws over the valve in the reversing chamber L, which becomes reversed into position for suction. This operation is now reversed, and as soon as the lungs have been emptied the bellows contracts and automatically reverses the valve into position for inflation.

Fig. 553.—Application of mask of pulmotor, the tongue being held forward by forceps, and oxygen prevented from entering esophagus by pressure with right hand.

The apparatus adapts itself to any pulmonary capacity. The rhythm will be slow when the lungs are capacious and faster when they are of less capacity.

It causes all the movements of respiration without any assistance being required from the hands other than to keep the windpipe of the patient open and the gullet closed in order that air will not be forced into the stomach. This latter maneuver is accomplished by placing the hand on the windpipe, as in Fig. 553, and making pressure sufficient to close the esophagus. The tongue must, of course, be held forward, as in Fig. 553.

The apparatus is devised so that no residual air from the system can find its way into the lungs again. In this way no air contaminated with poisonous or asphyxiating gas is breathed.

After respiration has been established, the lever, which is seen in Fig. 552, is thrown from pulmotor to inhalation, and oxygen is then given as shown in Fig. 554.

In addition to this regular sized pulmotor, there is an infant pulmotor which is especially adapted for use in maternity hospitals and by obstetricians. Both

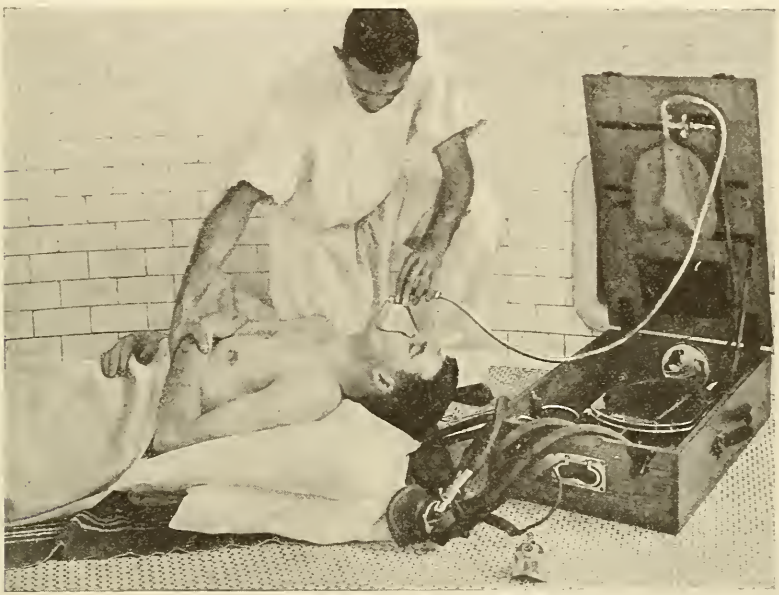


Fig. 554.—Administration of oxygen after respirations have been established.

the adult and the infant apparatus have a lever with which the operator can regulate expiration and inspiration at will, instead of using the automatic bellows.

If the mask fits, the pulmotor works admirably. If it does not fit, it fails. The mask fits some faces and not others. The great need of the instrument is a certainly adjustable mask.

**Drowning** is asphyxia brought about by having the mouth and nose submerged in fluid, air being thus excluded from the lungs. It is not only water that may be responsible for drowning. An individual may be drowned in mud or in a cesspool.

*Phenomena of Drowning.*—As asphyxia begins the victim struggles frightfully and clutches at anything that seems to be within reach. He usually sinks and rises an uncertain number of times. The old rule of three times has no particular force. It may be that the victim sinks and rises oftener, it may be that he does so less frequently. He may not rise at all. During this awful struggle air and water are inhaled, and, as a rule, some of the water is cast

out by vomiting and violent cough. During drowning water is certain to be drawn into the bronchial tubes and air-cells. While this struggle is going on the blood becomes less and less oxygenated, exhaustion deepens; finally the patient sinks to rise no more, some air is forced from the lungs causing bubbles on the surface of the stream, the ears ring, the muscles are convulsed, the mind wanders, merciful insensibility arrives, and death follows. During the mental wandering the memory may take clear and extensive or irregular and incoherent journeys through the events of years or a lifetime, but this does not always happen.

The duration of the death struggle is uncertain: it may be very brief, it may last a considerable time. The weaker a person is, the sooner it is over. It is probable that death occurs in two minutes after the final sinking. In spite of some specially trained divers being able to remain in water as long or even longer than this, "we may conclude, from all data, that fatal asphyxia is probable at the end of two full minutes' submersion of the head" (Draper's "Legal Medicine").

*Treatment.*—Because it is probable that a person is dead after two minutes' continuous immersion is not a sufficient reason for refusing to attempt resuscitation when it is alleged that the body has been immersed for longer than that period.

As Draper says, "the evidence as to time may be incorrect, and there are probably exceptional people who would not drown in two minutes. Resuscitation has been successful after five minutes and 1 case is recorded of recovery after twenty-five minutes" (Draper's "Legal Medicine"). The *ordinary emergency treatment* should be conducted out of doors. The mouth is emptied by turning the body on the face, with the head on a lower level than the body, and holding it so for a few seconds. While this is being done the mouth is opened and the tongue is drawn out. The body is turned upon the back, the head and shoulders being slightly elevated. The clothing is rapidly removed, and, while artificial respiration is being made (see page 866), hot bottles and hot blankets are being placed around the body and legs, and the mouth and nostrils are kept free of froth. If the individual makes an effort to breathe, help each respiration along. Give hypodermatic injections of brandy and strychnin.

Artificial respiration is continued until the patient breathes naturally and all cyanosis has passed away. As soon as he can swallow he should be given hot coffee and brandy. If minute follows minute and the patient does not attempt to breathe, we must still continue our efforts. Artificial respiration should not be abandoned for a full hour. Even after he begins to breathe he must be carefully watched for some time, as secondary respiratory failure is not uncommon.

On a battleship and upon the beach of a large seaside resort, where every preparation has been made to treat such accidents, more can be done than is described above. Artificial respiration can be made by the pulmotor or by the insufflation apparatus of Meltzer and Auer (see page 867). Adrenalin may be injected centripetally, as advised by Crile (see page 468). Hot enemata may be given and galvanism may be applied to the phrenic nerve.

After resuscitation there is danger of exhaustion and of bronchopneumonia.

**Hanging.**—A physician is occasionally called to a case of attempted suicide by hanging. In these cases there is no fracture and no dislocation of cervical vertebræ, as may occur in legal execution by the drop. The victim is dead or almost dead because of obstruction to the pulmonary air-way, the pressure upon the great vessels of the neck, and perhaps also upon the pneumogastric nerves.

*Treatment.*—The subject, after being cut down, is treated much like a case of drowning. Resuscitation is always difficult, no matter how soon the victim is cut down. It is impossible if asphyxiation is far advanced.

The neck and chest are bared, cold water may be applied to the head or face, as it sometimes will incite respiration. Artificial respiration is begun at once, external heat is applied, a hot enema containing brandy and hypodermatic injections of brandy are given. When there is distinct lividity, bleed from a vein of the leg (to do so from a vein of the arm might interfere with artificial respiration). Galvanism of the phrenic nerve is advisable. Even when breathing begins there is for some time the gravest danger of relapse and death.

**Throttling.**—When the assailant grasps the opponent's throat with the hands and squeezes the wind-pipe or larynx and forces it against the vertebræ the victim is said to be throttled. The ordinary fighter makes the attack from in front. The garroter seizes the throat from behind. Death from throttling is death from asphyxia plus an influence from the pressure upon the great vessels.

*Treatment.*—As for hanging.

**Smoke Asphyxia.**[1]—One of the first duties required of an intern in a hospital of a large city is ambulance service, and in performing such duty he is not infrequently called upon to attend fires. In fact, any physician may be called upon in such an emergency, and while on the fire ground may meet cases of asphyxiation by smoke. The clinical aspect and the treatment of such cases is not taught in the classroom. Literature on the subject is scanty. It is for these reasons that the author has deemed it expedient to insert a few words regarding it.

There are several factors which govern the character of smoke cases. In the first place, there are many different kinds of smoke. All smoke is hard to bear, but some kinds are worse than others. Some smoke is merely irrespirable, while other smoke is not only irrespirable, but is highly poisonous. Smoke from lumber, varnish, furniture, paper, rags, and wet hay is difficult to tolerate, whereas smoke from pitch, tar, and oils is not so pungent. Smoke impregnated with the fumes of ammonia, sulphur dioxid, chlorin, or pepper and other spices is frightfully irritant. Smoke containing nitric acid is highly irritant, and is apt to produce edema of the glottis and lungs. The hotter the smoke, the more irrespirable it is.

Again, the individual idiosyncrasy or susceptibility of the person plays an important part. It is remarkable how firemen can accustom themselves to remaining in a smoke-ladened atmosphere. The older members of a fire department can tolerate smoke much longer than those recently appointed. Firemen learn that the best air is near the floor or near the nozzle of the hose. There is a current of air along the floor, as the smoke naturally tends to rise, and the water carries through the hose a certain amount of air.

For clinical purposes, smoke asphyxia may be divided into three stages: the first stage is that in which the victim is conscious; the second is when consciousness is lost, but respirations are still present; the third stage, when respirations have ceased.

The first symptoms which occur are a choking sensation, severe throbbing in the head, dizziness, nausea, and muscular weakness. If at this time one is able to reach a window or able to leave the building and get fresh air he will probably recover quickly. His eyes are red and watery. He coughs and tries to vomit, and although his face may be hot and sweaty, yet his hands will be cold and clammy. His pulse is slow but bounding. Headache is intense and the eyes burn violently.

A patient in this condition should be rapidly removed to a spot where the air is free from smoke. He should be laid on a blanket. All constricting clothing should be loosened and he ought to be fanned. If he gags or attempts to vomit, but cannot do so, he may be given a drink of an effervescing salt, such

[1] The author, in "Therapeutic Gazette," March, 1903.

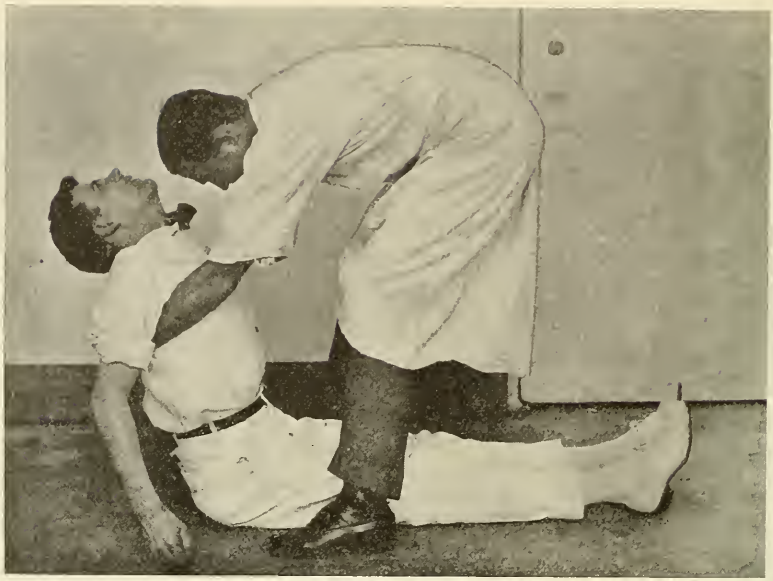


Fig. 555.—Fire carry. Step one.

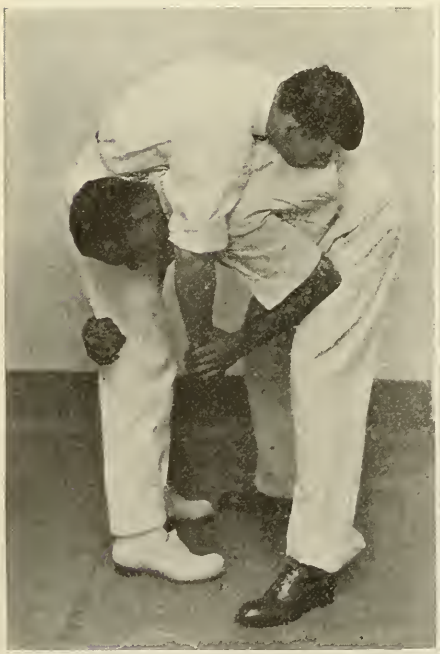


Fig. 556.—Fire carry. Step two.

Fig. 557.—Fire carry. Step three.

as a Seidlitz powder or a dessertspoonful of effervescent sodium phosphate in a tumbler of water. Firemen have great faith in weiss beer. It usually makes them belch and vomit and thus relieves them of much of the mucus and the

gases in their lungs and in their stomach. In this manner it acts as does an emetic dose of ipecac in the first stage of bronchitis. A drink which is often given, but which is most injurious, is whisky. The fireman who is a whisky drinker stands smoke poorly, and to take whisky after having been overcome with smoke only adds to the headache and nausea. Usually after vomiting has been induced and after he has had fresh air the fireman is able to return again to his work. If he does not rapidly recuperate or should he be overcome a second time, he should not be allowed to return to the smoky atmosphere. If a man who has been overcome by smoke has a chill he must be sent to a hospital. His usefulness is at an end for that fire. When a man becomes unconscious his comrades carry him to the street. Pictures of a very quick and satisfactory fire carry are given in Figs. 555–557. It is unusual for a man to leave a smoky building conscious and to lose consciousness after reaching the fresh air unless the smoke was impregnated with illuminating gas, which condition is discussed on page 874.

When an unconscious man is carried from a smoky building he must be taken to a spot where the air is free from smoke, his clothing must be loosened, and his body kept warm with blankets. His hands and face should be rubbed with a coarse towel, and oxygen (which should be carried on all ambulances and on all patrol wagons) should be administered. If the circulation or respiration is weak, he should be given a hypodermatic injection of strychnin or atropin, and when he is able to swallow he can be given a stimulant by mouth (as Hoffmann's anodyne or aromatic spirits of ammonia), or if he is trying to vomit he may be given weiss beer, effervescent sodium phosphate, or Seidlitz powder. He should not be given anything by mouth until he is entirely conscious and able to swallow. I mention this emphatically because I have often seen attempts made to pour whisky or some other stimulant down the throat of an unconscious fireman. The danger of such a procedure is obvious. As soon as practical he should be removed to the nearest hospital. The man should be taken to the *nearest* hospital unless the receiving ward of that hospital is overcrowded. He should then be taken to the next nearest hospital.

The third stage of asphyxiation by smoke is that in which respirations have been suspended. The lips are cyanotic, the skin is cold and clammy, the pupils are fixed, and usually dilated. The conjunctival reflex may be gone. The mouth may be open or, on the other hand, the teeth may be tightly clenched. The pulse is weak and fluttering, even imperceptible. There is frequently bleeding from the nose and mouth and involuntray evacuations of urine and feces may occur. A man in this state should never be placed in an ambulance to be taken to the hospital until respirations have been re-established. I have seen stupid and assertive policemen on many occasions insist on putting such cases in ambulances. He should be quickly laid on his back, all mucus cleared from his throat and mouth, all constricting clothing loosened, torn, or cut away. Artificial respiration is started at once. The pulmotor described on page 867 is most useful under such circumstances. If Sylvester's method is used, oxygen may be administered with a tube through the nostrils. The body must be covered with blankets and the man must be stimulated with strychnin, atropin, camphor, etc. When respirations have been established he may then be removed to the nearest hospital. As a rule, the man should be kept in a hospital at least twelve to twenty-four hours and may then be allowed to go home if there is no complication or sequel. In severe smoke cases there is a tympanitic percussion note extending well above each collar-bone and due, I believe, to blocking of bronchial tubes by spasm. It lasts often for several hours and then fades away.

In cases of edema of the glottis from the inhalation of irritant vapor, tracheotomy should be performed and artificial respiration given, oxygen pass-

ing in through the tracheotomy tube. It is imperative that the first-aid kits of all ambulances should contain a tracheotomy set. I have been compelled to do a tracheotomy with a penknife upon a fireman lying on the pavement.

A chill or a series of chills not infrequently follows smoke asphyxiation, and a man who has or has had a chill must always be sent to a hospital. If the smoke has been impregnated with ammonia, a piece of gauze or a handkerchief should be saturated with vinegar and held over the face so that the vapor may be inhaled. On the other hand, a few whiffs of diluted ammonia should be given after the inhalation of fumes from acids. The eyes should be carefully treated. Ice-compresses are most grateful. Pieces of lint or gauze are placed on ice and are transferred, when cold, to the eyes. They are changed at frequent intervals. The eyes must be washed with a saturated solution of boric acid at frequent intervals. Even after the acute symptoms have subsided it may be necessary to wear blue glasses for several days and to use an astringent eye-wash twice a day.

To relieve headache an ice-bag can be applied to the head and bromid should be administered by mouth. Bromid also relieves the severe nervousness which often ensues. Occasionally bronchitis or even bronchopneumonia may occur. Even after mild smoke cases there is usually a cough for several days, the sputum being streaked with black, carbonaceous material.

Inhalation of the vapor of nitric acid is apt to cause edema of the lungs and glottis and death. The acute edema may not come on for some hours after the inhalation. The *treatment* of edema of the lungs consists in venesection, hypodermatic injection of camphorated oil, the administration of alcohol and digitalis, and counterirritation of the chest. The treatment of edema of the glottis is set forth on page 873.

**Illuminating Gas-poisoning.**—Poisoning by illuminating gas is becoming more common. The statistics for the city of Philadelphia during 1912 showed that 152 cases died from illuminating gas-poisoning. Of these 77 were suicides and 75 were accidental. The number of cases has increased since the substitution of the so-called water-gas for coal-gas. The coal-gas formerly used was not nearly so dangerous. Coal-gas contains a small amount (about 7 per cent.) of carbon monoxid. In comparatively recent years, however, in order to reduce the cost and simplify the manufacture the new water-gas has been used. This is developed by forcing steam through hot coals or coke. To this water-gas hydrocarbons are added (methane, ethane, etc.). The amount of carbon monoxid in water-gas is about 38 per cent.

Jones ("Amer. Jour. Med. Sci.," 1909, vol. cxxxvii) gives as further reasons for the marked increase in poisoning by illuminating gas: first, concentration of population in cities; second, increased susceptibility to emotional states and insanity.

Suicide from illuminating gas is growing in frequency, due to the means being at hand, the known painless nature of the death, and also to the fact that the sale of toxic drugs to laymen is now forbidden.

Carbon monoxid has a marked affinity for hemoglobin. It completely destroys the oxygen-carrying power of the red blood-cells and thus deprives the tissues of oxygen. It enters the system solely through the lungs. An atmosphere becomes dangerous when it contains .05 per cent. of carbon monoxid (Gruber, cited by Edsall, in Osler's "System of Medicine"; Haldane, "An Investigation of Mine Air," 1895). It is not proved whether carbon monoxid has any direct action of its own, or whether it acts solely by robbing the blood of its oxygen. Edsall ("Amer. Jour. Med. Sci.," 1907, and Osler's "System of Medicine") says it is highly probable that both conditions occur. McCombs ("Amer. Jour. Med. Sci.," 1912, vol. cxliv) says that carbon monoxid has a direct toxic action on a human being.

Suicide cases make up approximately one-half of all the fatalities from illuminating gas in Philadelphia and in Massachusetts (McCombs, "Amer. Jour. Med. Sci.," 1912, vol. cxliv). Cases of accidental poisoning are due either to a leak from a gas-pipe or failure to close a gas-jet. Pettenkofer (cited by Edsall, in Osler's "System of Medicine") has shown that the leak may not be in the building itself, but the gas, having escaped from a broken main, may travel through the ground for some distance and finally enter a house. This is especially liable to occur in the winter when the heating of the building causes active motion of the atmosphere, thus drawing gas into the house by aspiration. Numbers of the employees of the large gas companies are overcome while working in the ditches in the city, yet among all such cases that have occurred in the city of Philadelphia there has been but one fatality. This is due to the careful instruction to the men by the physicians connected with the gas company. The men are not only instructed, but they also carry a first-aid kit and are competent to give immediate treatment.

Illuminating gas-poisoning has been divided into the acute and chronic forms. There is little evidence, however, to support the theory of chronic gas-poisoning. McCombs (Loc. cit.) has examined the blood of men who are constantly in contact with carbon monoxid, some of whom have been frequently overcome by it. He has found the average blood-count shows polycythemia. He has not noted any cases of muscular weakness, irregularity of the heart, bradycardia, lack of concentration, poor memory, cardiac dilatation, splenic enlargement, or pleural effusion. The acute cases are, of course, well known.

The symptoms of an acute case are ushered in by a sensation of vertigo or dizziness, headache, and muscular weakness. They are often accompanied or even preceded by a throbbing sensation in the head and throbbing of the vessels of the neck. McCombs (Ibid.) states that at first the pulse is slow, from stimulation of the pneumogastric. I have probably never seen a case in so early a stage, as every case I have examined has had a rapid and weak pulse.

During the early stage there is occasionally cerebral excitement. The pupils are dilated and the respirations are increased, both in depth and in frequency. There is no irritation of the mucous membranes. The victim has an odor of gas emanating from him. Unconsciousness now ensues. In some cases, however (as in the case of firemen working in smoke impregnated with illuminating gas), the irritant and suffocative qualities of smoke may overshadow all early symptoms of illuminating gas, and the victim may drop as suddenly as though he had been shot.

The foregoing symptoms have been called by McCombs the first stage of gas-poisoning. He then states that the second stage begins with syncope and ends with apnea. In the second stage, therefore, the patient is unconscious, but respirations are still in progress.

The respirations may be rapid and stertorous or may be of the Cheyne-Stokes type. The face is cyanosed. There is often frothing at the mouth. The froth may contain blood. The blood-pressure falls, but the temperature is usually elevated. There may be general tetaniform convulsions. The pulse is rapid and weak. The temperature varies from 99° to 103° F. Occasionally the temperature may rise rapidly, reaching 105° to 110° F., these cases usually terminating fatally. A great rise of temperature is usually coincident with the development of edema of the lungs. The high temperature is not due to the edema, but is probably due to the overwhelming toxemia. The toxemia is purely hemolytic in origin. The tongue is swollen and is cherry red, and the same peculiar cherry-red color makes its appearance on the skin of the neck, trunk, and buttocks. The blood, if drawn, is bright cherry-red in color and will show by the spectroscope the presence of carbon monoxid.

Edsall ("Amer. Jour. Med. Sci.," 1907) believes that, with the exception of the spectroscope, the best test for carbon monoxid in the blood is the Hoppe-Seyler test. The blood containing carbon monoxid, if treated with twice its volume of a solution of sodium hydrate, yields a beautiful red color when spread on a porcelain plate, while blood not containing carbon monoxid is changed into a dirty, brownish mass.

Katagama's test consists in adding to 10 c.c. of blood, diluted with water, 2 c.c. of ammonium sulphate solution and 0.2 c.c. of 30 per cent. acetic acid. Carbon monoxid blood gives a bright red precipitate, while normal blood gives a greenish precipitate. These tests should be used in cases of coma of uncertain origin.

Pettenkofer insists upon the importance of searching for gas-poisoning as the cause of the trouble, when various persons in the same house have a tendency to wake with headache or nausea.

In the third stage there is coma with apnea. The pulse is more rapid and is weaker. It is often impossible to count the rate. The skin is usually cyanosed, is cold and dry, and occasionally there appear on the extremities the back blebs. Blebs usually occur in chains and are most apt to appear in cases in which the coma persists for twelve hours or longer. The blebs contain clear serum and have frequently been mistaken for burns from the application of hot-water bags. There flexes are abolished. There is paralysis of the sphincters. Coma may last for days and yet be followed by a recovery.

Gilman Thompson ("N. Y. Med. Record," July 9, 1904) considers it a bad sign if leukocytosis, which is usually present, is of high degree. Reported cases, however, do not seem to substantiate this belief. The leukocytosis varies from 10,000 to 22,000. Thompson, moreover, reports an increase in the number of red blood-cells. The same observation is made by McCombs, but is denied by Glenn Jones.

Glenn Jones ("Amer. Jour. Med. Sci.," 1909, vol. cxxxvii) found the red cells reduced in number. The specific gravity and the coagulability of the blood are increased.

The pathology of gas-poisoning consists principally of the characteristic cherry-red spots on the surface of the body, the cherry-red color of the blood, and the same color of many or all of the organs. There is usually intense hyperemia of, and occasionally small free hemorrhages into, all the organs. Nephritis is usually present and is apt to be of the acute hemorrhagic type. There may be small scattered hemorrhages throughout the brain and cysts may form as the result of softening. Cardiac dilatation, fatty degeneration of the heart, and splenic enlargement have been described by Koren in cases of supposed chronic gas-poisoning.

The sequelæ of gas-poisoning are principally nervous manifestations. In the milder class of cases nervousness, insomnia, and headache are usually present for several days; in the more severe cases, intention tremors, loss of sexual power, delirium, neuritis, transient hemiplegia, confusional insanity, leptomeningitis, and encephalomyelitis have been reported. Prolonged fever and glycosuria have also been found.

There may be acute congestion of the lungs, edema, emphysema, or bronchopneumonia. McCombs states that all of the sequelæ usually clear up within six months or less, and that cases with sequelæ constitute less than 2 per cent. of all cases, and are usually confined to those persons who have absorbed large amounts of the carbon monoxid. Sequelæ are, of course, more apt to occur in those of advanced years and feeble condition than in those who are young and vigorous.

The prognosis should be based upon the duration of the exposure, the age of the victim, the degree of coma, the condition of the blood, and the character

of the pulse and respiration. Usually a short exposure to illuminating gas means a case amenable to treatment and which will promptly recover. Longer exposure, or exposure of the aged and feeble, means a far worse prognosis.

Edsall cites 39 cases treated in the Episcopal Hospital of Philadelphia, of which 34 recovered. Of the 5 fatalities, several were due to sequelæ. If the onset of the symptoms are rapid, if edema develops, or if pronounced hemolytic changes are present, the prognosis is unfavorable. The persistence of coma is highly unfavorable, and Jones states that all cases that develop cutaneous blebs end fatally.

The treatment of poisoning by illuminating gas must be prompt and heroic. In the milder cases, in which there is neither coma nor apnea, the patient must be removed to an atmosphere free from the poison and must be given oxygen freely. The object of all treatment is to give oxygen in sufficient quantities to displace the carbon monoxid from the blood. Nausea and the feeling of fulness in the stomach can be relieved by the administration of effervescent sodium phosphate, Seidlitz powder, or a bottle of weiss beer. These procedures will usually cause vomiting and give relief. The patient should be kept quiet, an ice-bag being applied to the head to relieve the headache. Doses of bromid of potash will give comfort. Hoffmann's anodyne and aromatic spirits of ammonia act as useful stimulants and carminatives. Caffein, digitalis, strychnin, and camphor should be given if necessary. They will hardly be necessary, however, unless the victim is in the second stage or the stage of unconsciousness. When the patient is unconscious, venesection should be performed, followed by transfusion, or by the intravenous injection of salt solution.

The patient must be kept quiet, as many victims have died from sudden exertion. Venesection without the injection of salt solution or without transfusion is usually condemned, but Halsted reported 2 cases in which he thought it did good. Transfusion of defibrinated blood was practised in the 70's and 80's and successful cases were reported. Reinfusion of blood was attempted by Halsted in 1884, at which time he reported a successful case: 512 c.c. of blood were withdrawn from the radial artery, defibrinated and strained, and 280 c.c. were reinfused into the artery. In 1907 Crile and Lenhart reported their studies of transfusion, which procedure has supplanted all former methods of treatment.

Of course, in the third stage, that of coma with apnea, artificial respiration must be made, and for this purpose the pulmotor is most valuable, as it not only gives artificial respiration, but at the same time administers large quantities of oxygen under pressure.

Massage is an important feature of treatment. It increases circulation in the extremities. Transfusion, of course, requires the presence of a skilled surgeon, whereas intravenous injection of saline solution is not nearly so difficult a procedure. It is most important that artificial respiration should not be abandoned for a number of hours, as cases have been reported in which artificial respiration was finally successful after a period of six hours. In conditions which lead one to suspect that chronic poisoning may be present, the cause should be sought and, if found, removed, and the case be treated symptomatically. The general public should be instructed as to the danger of acute gas-poisoning.

## Diseases and Injuries of the Nose and Antrum

**Foreign bodies in the nose** (see Poulet on "Foreign Bodies in Surgery") are usually introduced through the anterior nares, but in rare instances during swallowing they enter by way of the posterior nares, a sudden expiration being the cause of the entry. During vomiting foreign bodies may enter the posterior nares. Small particles are often expelled spontaneously; larger

pieces collect mucus and epithelium and become fixed. Some materials swell after lodgment. Others become encrusted with lime salts. Seeds may sprout. In very rare cases insects enter and lodge. Cases are on record of leeches, taken in with drinking-water, passing into the nasal fossæ from the pharynx. In the tropics flies may deposit larvæ within the anterior nares and they develop with great rapidity. A foreign body is usually near the floor and may be between the vomer and turbinate bones. It may shift after lodgment.

**Treatment.**—In many cases general anesthesia is required. Illuminate the nostril, and, if the foreign body can be seen, insert a hook back of it and effect its removal by means of forceps. Some foreign bodies require to be pushed back into the nasopharynx. Occasionally expulsion may be effected by inserting a rubber tube into the unblocked nostril and telling the patient to blow forcibly through the tube. In serious cases a specialist should be summoned to remove a portion of the turbinated bone or to perform whatever operation he thinks best.

**Inflammation and Abscess of the Antrum of Highmore (the Maxillary Antrum).**—The source of this disease may be inflammation within the nose or periostitis around the roots of the teeth. In some cases the natural opening into the meatus is patent; in other cases it is partly or completely blocked. Caries and necrosis may arise. The **symptoms** are pain, edematous swelling of the face, and thinning of the bone so that it may crepitate under pressure. When pus has formed, if the antral opening is patent, certain positions of the head will cause a purulent flow from the nose, and if a speculum is inserted pus may be seen as it flows into the nose. The opening of the maxillary antrum into the nasal channel is at the summit of the antrum; hence the antrum drains when the head is inverted. The ethmoidal cells and frontal sinus drain best when the patient is upright. Wipe the interior of the nose and place the patient with his head between his knees. If the nostril fills with pus, it comes from the antrum (Cobb). In severe cases the jaw expands, the eye protrudes, and great tenderness of the alveolus exists. Percussion exhibits a dull note. In making a diagnosis it is well to take the patient into a dark room, insert an electric light into the mouth and note the diminution of light transmission on the diseased side as contrasted with the sound side. Transillumination may be easily practised by the use of a cautery electrode, protected by a small glass vial. Any cautery battery may be employed (plan suggested by Ohls). Exploratory puncture will settle a doubtful diagnosis. This may be by way of the lower meatus, the canine fossa, or the alveolar process.[1]

**Treatment.**—Before pus forms order the use of hot fomentations and remove any diseased teeth. When pus has formed, evacuate it at once. Before performing a severe operation try the effect of opening into the antrum from the nose, by means of Krause's trocar, followed by insufflation of iodoform. If this procedure fails, other means may be employed. If the disease arises from a carious tooth, pull the tooth and push a trocar through its socket into the antrum. If the teeth are sound, bore a hole with a large gimlet or with a bone-drill above the root of the second bicuspid tooth and 1 inch above the edge of the gum. A counteropening should be made into the inferior nasal meatus. A drainage-tube is pulled from the first opening into the nose and is allowed to protrude from the nostril. Irrigate daily with normal salt solution. In three or four days discontinue through-and-through drainage, but prevent the first opening closing until the discharge ceases to be purulent. In severe cases make a free incision through the canine fossa by means of a chisel.

**Distention and Abscess of the Frontal Sinus.**—The usual cause is an injury which may long antedate the symptoms. This injury causes or leads

[1] Cobb, in "Boston Med. and Surg. Jour.," May 7, 1896.

to blocking of the infundibulum; secretion accumulates and distends the sinus, and in some cases pus forms. In many cases the fluid slowly accumulates, and it may require years to produce marked symptoms. In other cases infection takes place early or existed from the start, and the symptoms are positive and violent. If the outlet into the nose is not permanently blocked, the fluid may discharge itself from time to time. In the chronic cases there is seldom much pain. The chief sign is a swelling of the inner or upper part of the orbit, which swelling progressively increases and finally displaces the eye. If at any time acute symptoms supervene, there will be pulsatile pain, discoloration, and tenderness.

**Treatment.**—In some cases it is possible to pass a trocar upward from the nose into the sinus, and so drain and irrigate. In most cases an incision should be made through the soft parts, and the sinus be opened by a trephine or chisel. After the sinus has been opened it must be curetted. The opening into the meatus should be restored and enlarged, and a drainage-tube must be passed from the forehead incision into the nostril. I usually prefer to open the sinus by making an osteoplastic flap in the anterior wall.

## Diseases and Injuries of the Larynx and Trachea

**Edema of the Larynx** (*Edema of the Glottis*).—The **causes** of edema of the larynx are: acute laryngitis; chronic diseases, such as tuberculosis, malignant disease, or syphilis; inflammatory disorders, such as diphtheria and erysipelas; acute infectious diseases; Bright's disease; aneurysm; whooping-cough; pneumonia; quinsy; wounds of the larynx; wounds of the neck; scalds and burns of the larynx, and the inhalation of irritating vapors, such as those of ammonia, nitic acid, or sulphur.

The **symptoms** are sudden and rapidly increasing dyspnea, respiratory stridor, huskiness of the voice, and finally aphonia. The swollen epiglottis may be felt with the finger and may be seen with the help of a mirror.

**Treatment.**—In cases in which edema of the larynx is not excessively acute, introduce a gag between the teeth, hold the mouth open, take a knife wrapped to within $\frac{1}{4}$ inch of its point, make multiple punctures into the epiglottis, and favor bleeding by the inhalation of steam. In severe cases perform intubation or tracheotomy.

**Wounds and Injuries of the Larynx.**—The larynx may be injured internally by foreign bodies, and externally by blows and cuts. A condition often met with is *cut throat*, the result usually of a suicidal attempt on the part of the patient or a homicidal effort on the part of an assailant. The cut of the suicide is usually in front; as a rule, it misses the great vessels, but divides the cricothyroid or thyrohyoid membrane. The epiglottis may be incised, or even be cut off. If a large vessel is cut, death rapidly occurs. The immediate dangers of cut throat are hemorrhage, suffocation by blood in the wind-pipe and bronchi, or by displacement of parts, and entrance of air into veins. The secondary dangers are bronchopneumonia, infection and sepsis, exhaustion, and secondary hemorrhage. The remote dangers are stricture and fistula (Keetley).

**Treatment.**—In wounds of the throat arrest hemorrhage, remove clots from the larynx and trachea, bring about reaction, asepticize the parts as well as possible, suture the deeper structures with silver wire, catgut, or kangaroo-tendon, and the superficial parts with silkworm-gut, dress antiseptically, and place a bandage around the head and chest so as to pull the chin toward the sternum. If laryngeal breathing is much interfered with, perform tracheotomy. Feed the patient through a tube until union is well advanced. The old method of leaving the wound open is to be condemned. When sutures are

used, primary union may be obtained. This fact was proved by Henry Morris.

**Scalds of the Glottis.**—(See section on Burns and Scalds.)

**Foreign Bodies in the Air-passages.**—The lodgment of foreign bodies in the air-passages is a frequent accident. A multitude of different things have been reported as having lodged in the air-passages. Small solid bodies are usually expelled by coughing. Liquids and solids rarely pass beyond the larynx (except in laryngeal disease or palsy, wounds of the floor of the mouth, cut throat, and in people unconscious or very drunk). In vomiting during or after the administration of an anesthetic or in the vomiting of drunkards the vomited matter may find its way into the larynx or lungs. There is great danger of this accident in an operation upon a patient with intestinal obstruction who has stercoraceous vomiting. In most instances of foreign bodies lodged in the air-passages it will be found that the object was being held in the mouth when a sudden deep inspiration was taken (often during laughter).

The **symptoms** are *immediate*, due to obstruction by the body and by spasm; and *secondary*, due to the situation of the body and the changes it undergoes or induces.

*Lodgment in the pharynx* causes violent dyspnea. The body can be seen or felt.

*Lodgment in the Larynx.*—A foreign body may lodge in the superior opening of the larynx, in the rima, or in the ventricle. In a severe case the patient fights madly for air; his face becomes livid and cyanotic; his veins stand out prominently; speech is impossible, though he may make noises and utter harsh cries; violent coughing begins, and then vomiting; he tries to force a finger down his throat and clutches at his neck; sweat pours from him; he feels a sense of impending dissolution, and he falls unconscious, with incontinence of feces and urine.[1] In a less severe case violent dyspnea gradually departs and the patient lies exhausted; but dyspnea and cough are liable to recur suddenly at any time because of spasm, and they may be induced by a change of position. These attacks of fierce spasmodic cough are not at first linked with expectoration, but after inflammation begins there is a profuse and often bloody expectoration. Inflammation follows more rapidly the lodgment of a sharp or irregular body than it does that of a round or smooth one. Inflammation is apt to produce edema of the glottis, bronchopneumonia, or ulceration and necrosis of the larynx. Any sort of foreign body in the larynx may at any moment produce spasmodic dyspnea, and is always very liable to cause edema of the glottis. The body if bony or metallic can be detected by the *x*-rays. A body may remain lodged in the ventricle for a long time without producing symptoms.

*Lodgment in the Trachea.*—The immediate symptoms of a foreign body in the trachea depend on the shape and weight of the body, and whether it becomes fixed in the mucous membrane or moves to and fro with the air-current. A smooth, heavy body falls to the tracheal bifurcation, and, if it does not enter a bronchus, moves with every breath, and by its movement causes violent laryngeal spasm, cough, and whooping inspiration without aphonia. The patient is often conscious of the movements of the foreign body, and the surgeon may detect them by the stethoscope. The foreign body may be found by the Röntgen rays. A foreign body in the trachea is liable to cause death by suffocation, or it may ascend so as to be caught in the larynx, or may even be expelled. Irregular or sharp bodies lodge in the mucous membrane, produce inflammation, frequent cough and expectoration, and finally lead to ulceration. Bodies which swell from heat and moisture tend to lodge and to become fixed (seeds may sprout).

[1] See Moullin's graphic description in his "Treatise on Surgery."

*Lodgment in a Bronchus.*—Foreign bodies in the bronchi seriously endanger life. They usually lodge in the right bronchus. The right bronchus is more nearly the direct continuation of the trachea than the left bronchus. When a small area of lung is obstructed the obstructed side shows diminished respiratory movement and murmur with occasional whistling sounds and large moist râles; the percussion-note is at first normal and later dull. When an

Fig. 558.—Author's case of pin in bronchus removed by low tracheotomy.

entire lobe is obstructed all respiratory sounds are absent over it, and over the unobstructed lung respiration is exaggerated; the percussion-note over the obstructed area is at first resonant, but becomes dull. The *x*-rays will enable the surgeon to detect many foreign bodies in a bronchus. Lodgment in a bronchus may cause bronchopneumonia, abscess, hemorrhage, and even gangrene. In some cases the body has been expelled spontaneously. In rare instances

Fig. 559.—Gibbon's case of tack in the right bronchus removed by low tracheotomy, child six years old.

people have lived for years with lodged foreign bodies. If death does not soon follow the lodgment of a foreign body, an abscess is very apt to form.

**Treatment.**—If a foreign body lodges in the pharynx, try to pull it forward; if this fails, it may be wise to push it back into the esophagus. In lodgment in the larynx or below, if the symptoms are very urgent, at once perform quick laryngotomy or tracheotomy. If the symptoms are

not so urgent, get a complete history of the accident and find out the nature of the foreign body. Be sure that a foreign body is really retained in the respiratory tract, and then try to determine what its situation may be. A person sometimes imagines a body is lodged when it has been expelled or even when it was never in the larynx, trachea, or bronchus at all, but was swallowed. Often a laryngologist can remove a foreign body from the larynx by means of forceps, a mirror and lamp being used for illumination. The fauces and upper portion of the larynx should have cocain applied to them to lessen pain and spasm. If the surgeon fails in extraction by forceps, and laryngotomy has been performed, continue the search through the opening in the cricothyroid membrane; if laryngotomy has not been performed, let the larynx be opened by *thyrotomy* (a vertical incision between the alæ of the thyroid cartilage, and the separation of these alæ to permit of exploration). After a thyrotomy suture the perichondrium with catgut. If the foreign body is in the trachea, perform ordinary tracheotomy; if it is in a bronchus, perform low tracheotomy. Tracheotomy prevents suffocation from laryngeal spasm or edema of the glottis. It may be possible to remove the body in the bronchus through the incision of a low tracheotomy, and this ought to be tried. By this method I succeeded in 5 cases, removing a pin, a bone, a bean, a tack, and a broken tracheotomy tube from the right bronchus. The foreign body may be expelled through the tracheotomy wound; if it is not expelled, search the trachea and bronchi with Gross's forceps, with probes, with hooks, or with the finger. If the foreign body cannot be found, put the patient to bed and maintain a moist atmosphere in the room. As a rule, when the foreign body is not found, insert a tube. If the foreign body be extracted, do not insert a tube (unless edema of the glottis exists or is likely to come on), do not suture the wound, but cover it with moist gauze and let it heal by granulation. Morphin and sedative cough-mixtures are given. Gross says that even when a foreign body has long been retained an operation should be performed if the air-passages are not seriously diseased. What shall be done when a foreign body is lodged in a bronchus and we are unable to extract it through a tracheotomy wound or by tracheobronchoscopy? Truc said if "the patient is in danger of death" cut through the chest-wall and attempt to remove the body. He said this with a full knowledge of the difficulty of locating the body. This difficulty has been partly overcome by the *x*-rays, and it seems now more certainly our duty to operate than it was a short time ago. Nasiloff proposed to reach the obstruction by the posterior route after rib resection. Curtis attempted this, and though the patient died, his operation proves that the method is feasible. An operation by the posterior route should be performed at once if low tracheotomy fails. The danger of pulmonary collapse will be abolished by the use of a suitable apparatus to prevent it (see pages 895 and 904).

*Tracheobronchoscopy.*—Killian, of Freiburg, devised a bronchoscope for introduction through a tracheotomy wound. I have used the endoscope in this manner. Later Killian devised a straight instrument which could be used through the mouth and larynx, tracheotomy being unnecessary. During its introduction the head was held far back.

Brüning, of Freiburg, improved the instrument. His tube is long enough to reach between the vocal cords to the divisions of the bronchi. In 1907 Killian was able to collect 164 cases of foreign bodies removed by this direct method.

Dr. Chevalier Jackson, of Pittsburgh, has devised a tracheobronchoscope through which he has succeeded in removing foreign bodies from the trachea and from a bronchus. In 10 cases of foreign body in the bronchus he removed the offender in 7. In 7 cases of foreign body in the trachea he was successful in each case, but 2 of the cases required tracheotomy. Whenever there is dyspnea he always prepares to do tracheotomy ("Annals of Surgery," March,

1908). In Jackson's trained hands the instrument is most useful, but an untrained man with it would be like an untrained man with a cystoscope. When used by a fully trained man the instrument prevents many cutting operations and saves many lives (see page 885).

### Operations On the Larynx and Trachea

**Tracheotomy.**—In a formal operation give ether or chloroform, but in an urgent emergency this cannot be done. The patient may be placed supine with a sand-pillow under the neck and with the head thrown over the end of the table. If a child, Liston used to wrap it up to the neck in a sheet to prevent movements of the limbs, would seat himself on a chair, place the child upon the nurse's lap, and take its head between his knees. The head must be exactly in the middle line and extended (in an adult this gives $2\frac{3}{4}$ inches of trachea above the manubrium; in a child of ten, $2\frac{1}{4}$ inches; in a child of six, about 2 inches). The operator stands to the right side when the patient is supine. If bleeding is profuse when the surgeon is ready to open the trachea, place the patient in the Trendelenburg position with the neck extended. The trachea may be opened above or below the isthmus of the thyroid gland. The isthmus in an adult

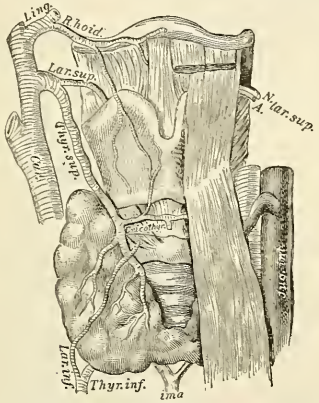



Fig. 560.—Blood-supply of the larynx and trachea (Esmarch and Kowalzig).

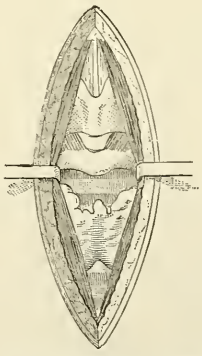


Fig. 561.—Parts exposed in tracheotomy (Esmarch and Kowalzig).

usually lies over the second and third rings (Figs. 560 and 561). The isthmus in a child usually lies over the first ring or even over the space between the cricoid cartilage and the first ring. The high operation is always chosen except in cases in which it is desired to search for a foreign body in a bronchus.

**High tracheotomy** is preferred because in this region the muscles are distinctly separated (Fig. 561), the main vessels of the neck and the inferior thyroid vessels are not encountered, the anterior jugular veins are small and have very few transverse branches, and the trachea is near the surface (Treves). The surgeon accurately locates the cricoid and thyroid cartilages. An incision is begun at the upper border of the cricoid cartilage, and is carried down precisely in the middle line for about $1\frac{1}{2}$ inches. Treves advises the operator to steady the skin of the neck with the fingers of the left hand and to cut with the unsupported right hand (if the hand be supported, the respirations will interfere with the operation). The skin, the superficial fascia, and the anterior layer of the cervical fascia are incised, the sternohyoid and sternothyroid muscles are separated, and the fascia over the trachea is divided. This fascia is attached above to the cricoid cartilage, and it divides below into two layers to invest the thyroid body and its isthmus. If veins are in the line of the incision, they are

pushed aside, but it is not necessary to take the time to apply double ligatures. Even if bleeding is profuse, as soon as the trachea is opened and air enters freely into the lungs, venous congestion is relieved and bleeding is apt to cease. If hemorrhage be violent and the veins are not at once caught by forceps, it may be well to place the patient in the Trendelenburg position before incising the windpipe, in order to prevent the entrance of blood into the lungs. Before opening the trachea the isthmus of the thyroid gland is pushed downward; if it cannot be pushed down sufficiently, a transverse incision is made through the fascia at the upper border of the cricoid cartilage, and the fascia and the isthmus with it are lifted off the trachea (Bose's method). A tenaculum is inserted into the cricoid cartilage in order to steady the tube. The back of the knife is turned toward the sternum, a finger being held upon the blade to prevent too deep a cut being made. The knife is plunged, as if it were a trocar, into the midline of the trachea above the isthmus, and two or three rings are divided from below upward. The hook is not removed until the operation is completed. If a foreign body is present, an attempt is made to remove it; if success attends the effort, no tube need be worn; but if the body is not found, a tube must be used. In croup or diphtheria remove membrane (by means of a feather and a solution composed of bicarbonate of sodium 2 oz., glycerin 1 oz., water 10 oz.—Parker) and insert a tube. The edge of the cut is grasped with the dissecting forceps, the mucous membrane being included in the bite; the head is placed erect, the tube is introduced, and the tenaculum is removed. Secure the tube by tapes, and suture the wound below the tube. Remove the tube at the first moment consistent with safety. After tracheotomy put a screen around the bed; have the air kept moist by steam; remove the inner tube and clean it every few hours at first; clean the outer tube whenever required. In croup or diphtheria put the patient in a croup tent and keep the air moist by a steam atomizer or a croup kettle. Remove and clean the inner tube every two hours. Clean the larynx and trachea from time to time by means of a feather and Parker's solution.

**Quick laryngotomy** must never be attempted upon a child under thirteen years of age, because of the small size of the cricothyroid space before this age (Treves). In view of the difficulty of introducing a tube and of wearing it so near the vocal cords, laryngotomy should not be performed for croup, diphtheria, or for any condition in which a tube must be long worn. The operation is performed as follows: Make an incision $1\frac{1}{4}$ inches in length in the middle line, from above the lower edge of the thyroid to below the lower border of the cricoid cartilage. Divide the skin, superficial fascia, and deep fascia, separate the cricothyroid and sternothyroid muscles, divide the deep layer of fascia, and cut the cricothyroid membrane transversely just above the cricoid cartilage. The tube must be shorter than the ordinary tracheotomy tube. An operation which opens vertically the cricothyroid membrane, the cricoid cartilage, and the upper rings of the trachea is called *laryngotracheotomy*.

**Intubation of the Larynx** (*O'Dwyer's Operation*).—Bouchot conceived the idea of intubation; O'Dwyer perfected it and made it a genuine scientific proceding. The instruments required for the performance of this operation are a mouth-gag, an instrument to hold the tube and introduce it, and an instrument for extracting the tube. The collar of the tube has a perforation through which a piece of silk is fastened to draw out the tube. The child is wrapped in a sheet to secure the limbs, is seated in a nurse's lap, and its head is held by an assistant. The jaws are opened and held apart by the self-retaining mouth-gag. The surgeon sits in front of the patient, wraps a piece of rubber plaster about the index-finger of his left hand, and passes the finger into the child's mouth until its tip touches the epiglottis. He introduces the holder and tube (observing if the silk is free) along the surface of the tongue until the

obturator touches the epiglottis; raises the epiglottis with the left index-finger, and passes the tube into the larynx; places the left index-finger against the tube, and withdraws the holder with the right hand. The silken thread is tied to the ear, and the nurse is directed to employ the thread to remove the obturator if it becomes obstructed or is coughed up. The tube is removed in two or three days; if breathing is easy, it is not reintroduced; but if dyspnea recurs, it is replaced for two or three days more. If, in introducing the tube, a mass of false membrane is pushed before it into the trachea, breathing ceases, and, if the mass is not at once coughed up, tracheotomy must be performed. Feed these patients on semisolids rather than upon liquids (mush, soft eggs, and cornstarch); and if trouble occurs in swallowing these articles, feed by the rectum or by means of a nasal or an oral tube. In opium-poisoning, in asphyxia, in acute traumatic pneumothorax, and in cerebral injuries, intubation may be associated with the use of Fell's apparatus (see page 896).

Fig. 562.—Direct laryngoscopy for the introduction of a tube for intratracheal insufflation anesthesia. The patient's head is on the table, with the forehead pushed strongly down backward, so that the vertex is under the chin, the head in extreme extension, and the hyoid bone thrown upward (Chevalier Jackson).

**Chevalier Jackson's Tracheobronchoscopy** (Dr. Chevalier Jackson did me the honor and the favor to write for me the following description of his operation).—"Direct inspection of the trachea and bronchi by means of a suitably illuminated tube passed through the mouth is now well established as a procedure of great value for the removal of foreign bodies and for diagnosis and direct local treatment of disease. In the earlier days of bronchoscopy a tracheotomy was done for the insertion of the bronchoscope, but this is now quite unnecessary, because perfection of instrumentarium and technic have rendered it very easy for those who have acquired the knack to insert the bronchoscope through the mouth and larynx. The first step in bronchoscopy is the exposure of the larynx by direct laryngoscopy.

"*Direct laryngoscopy* is so called in contradistinction to the ordinary methods of examination of the reflected image in a throat mirror. The latter pro-

cedure is invaluable for diagnosis, but for operative work the anteroposterior reversal of the image involves extreme difficulties. By direct laryngoscopy the larynx is exposed to direct view for operative work, removal of specimens of tissue, and for insertion of tubes for intratracheal insufflation anesthesia (Fig. 562). For laryngeal operative work direct laryngoscopy is usually done under local anesthesia in the sitting position in adults; and without anesthesia, general or local, in children. General anesthesia is particularly dangerous in any condition of the larynx associated with even a slight degree of dyspnea. The patient is usually in the sitting position for direct laryngoscopy in adults with local anesthesia (Fig 563), recumbent for direct laryngoscopy in children, and usually recumbent for bronchoscopy regardless of anesthesia,

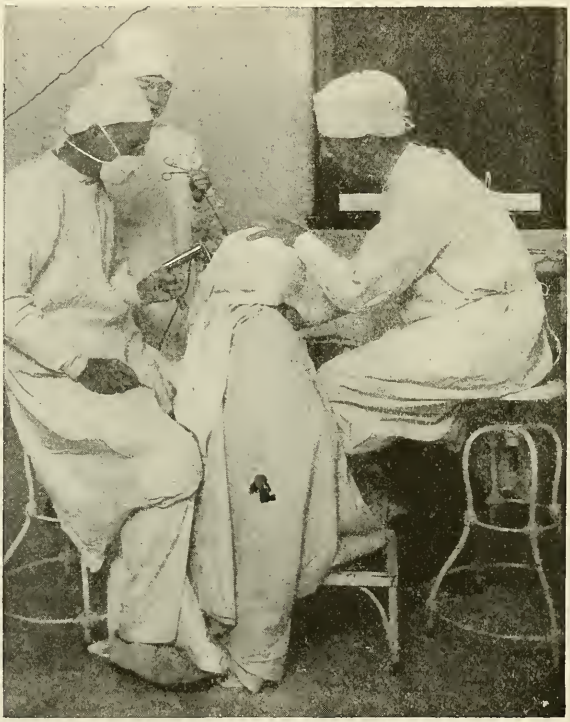


Fig. 563.—Direct laryngoscopy with the patient in the sitting position, for diagnosis and for endolaryngeal operation, and removal of specimens of tissue. For endobronchial diagnosis and local medication the bronchoscope may be passed in this position, but for foreign-body work in larynx or bronchus the recumbent position is better, in order to have the aid, instead of the opposition, of gravity (Chevalier Jackson).

though occasionally diagnostic bronchoscopies are done in the sitting position in adults. The technic of direct laryngoscopy and bronchoscopy is easily understood from Fig. 564, which represents the procedure in the recumbent position. The patient's head is fully extended by forcing the occiput down backward toward the shoulders, which throws the anterior part of the neck high up, elevating the hyoid bone and its attached structures. In doing this the *head must be raised*, never lowered. For this reason the schema is drawn with the head on the table, because the head should never be below the level of the table. For bronchoscopy the head and neck should be out in the air beyond the table and supported by an assistant, so that the head may be freely movable as needed. For instance, it must be moved to the right for

the bronchoscope to enter the left bronchus, and vice versâ to enter the right bronchus; and it must be slightly lowered to enter the middle lobe bronchus of the right side, raised to enter the posterior branch bronchi. For direct laryngoscopy only, however, the head should be on the table, and for the insertion of intratracheal insufflation tubes should be precisely as shown at A in Fig. 564. With the head properly placed, the next point of importance is to expose the larynx to direct view. To pass the speculum posterior to the tongue and to identify the epiglottis are easy. Now comes the most important movement in all peroral endoscopic procedures. The beak of the direct laryngoscope is passed posterior to the epiglottis for about 1 cm., when a strong lifting motion is given by the *beak* of the instrument, as if to lift the epiglottis out anteriorly through the neck in the direction of the dart. The upper teeth must not be used as a fulcrum. With the vocal cords in full view, the bronchoscope is inserted, with the handle horizontal, into the laryngoscope. The eye is now transferred from the laryngoscope to the bronchoscope. When the vocal cords separate at the next inspiration the bronchoscope is quickly passed through the glottis into the trachea, as shown at B. The bite-block is now inserted to prevent the patient damaging the thin-walled bronchoscope. The slide is then removed, as shown at C, leaving the bronchoscope in the trachea, as shown at D. The bronchial tree is exceedingly elastic and flexible, and may be explored by following the lumen by sight. Secretions may be removed by the sponge-holder (Fig. 565, C) uncontaminated by the secretions from the mouth for diagnosis or vaccine therapy. Suspected tissue may be carefully removed for examination by means of biting forceps (Fig. 565, E). Foreign bodies may be extracted from the trachea or bronchi with suitable forceps (Fig. 565, D). Compressive stenosis by the thymus gland, mediastinal tumors,

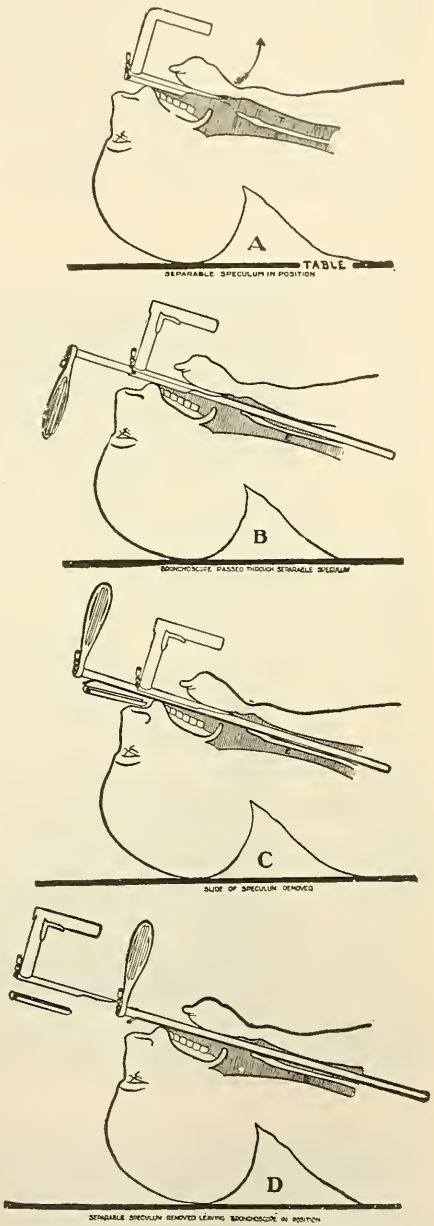



Fig. 564.—Schema showing the technic of direct laryngoscopy and bronchoscopy. The upper illustration (A) shows also the position of the patient and the direction of the force to be exerted on the direct laryngoscope in exposing the larynx for the insertion of intratracheal insufflation tubes for anesthesia (Chevalier Jackson).

and adenopathic masses may be diagnosticated. Deviation or compression of the trachea by goiter can be determined, and valuable aid can be rendered the surgeon by an accurate localization of the stenosis in all cases associated with dyspnea. No dyspneic case should ever be generally anesthetized without a preliminary examination of the larynx and trachea.

"*Esophagoscopy.*—For the passage of the esophagoscope no anesthetic, general or local, is really necessary, and none should be given in children, because cocain is dangerous in the very young, and general anesthesia assumes a graver risk especially in foreign-body cases in children because of the compression of the trachea by the bulk of the bube plus the bulk of the foreign

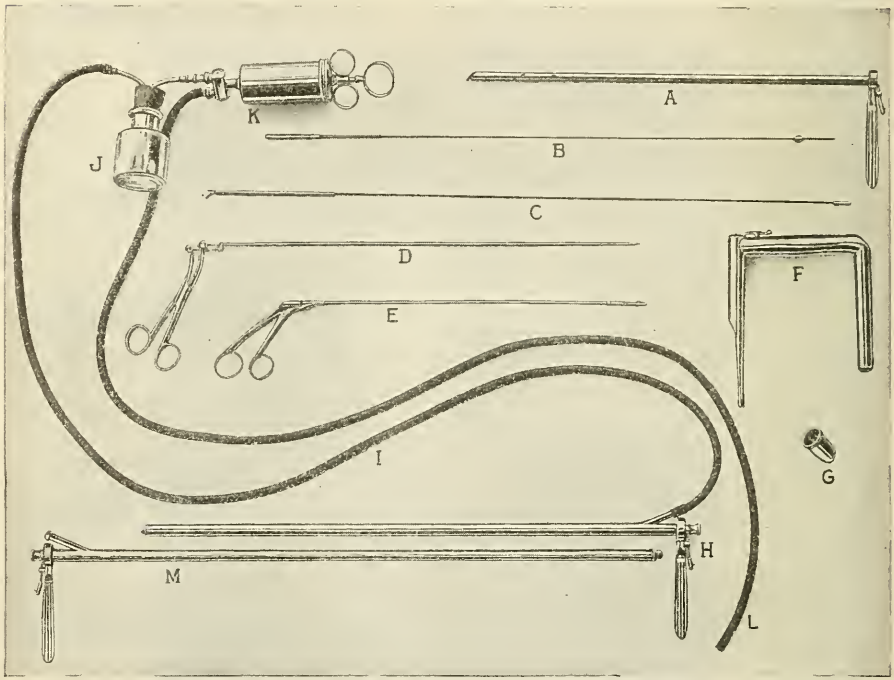



Fig. 565.—Jackson's instruments for direct laryngoscopy, bronchoscopy, and esophagoscopy: A, Bronchoscope; B, olive bougie for esophagoscopic bouginage; C, sponge-holder for sponging the field and obtaining specimens of secretion; D, forceps for removing foreign bodies; E, forceps for removing tissue; F, laryngeal speculum, called also slide speculum and direct laryngoscope; G, bite-block; H, esophagoscope with tubing (I) leading to aspirator (J, K) for removal of secretions. The tubing (L) is connected with the positive pressure side of the syringe (K) for use when needed to blow out obstructions, such as clots of pus, food, etc., that have been aspirated into the drainage canal of the esophagoscope. At M is shown a gastroscope.

body, especially if the latter is overriden or displaced. Respiratory arrest is likely to follow and the respiration can rarely be started except by bronchoscopic insufflation. In adults the lower pharynx and mouth of the esophagus may be anesthetized with an 8 per cent. solution of cocain. There is no need of anesthetizing the esophagus below the orifice, because it is quite insensitive there. Until the knack is acquired, the operator will find it difficult to pass the esophagoscope without deep general anesthesia, which should be by ether, never chloroform. The patient is placed in the Boyce position, previously described, the bite-block being held by the index of the assistant's right hand. The esophagoscope is inserted at the right side of the patient's tongue (Fig. 566) and the pyriform sinus sought immediately to the right of the right arytenoid.

Once the tube mouth is in the sinus, very gentle pressure is used, and the lumen of the mouth of the esophagus is watched for and followed at the moment of inspiration. Absolutely no force should be used, and no attempt to advance the tube should be made except at the moments when the patient takes a deep breath, which he must be encouraged to do. It is the spasm of the inferior constrictor at the cricoid level that gives the trouble. Once this is passed, the lumen of the cervical esophagus is easily explored, and the thoracic esophagus opens widely at each recurrence of the inspiratory negative intrathoracic pressure. Passing the hiatus esophageus requires a moment for the relaxation of spasm and the tube must be directed slightly upward (recumbent patient) to the left. Esophagoscopy is useful for the detection of acute and

Fig. 566.—Introduction of the esophagoscope under guidance of the eye. The tube-mouth is just entering the pyriform sinus. The assistant is holding the head in extreme extension and elevated. The nurse at the left is manipulating the aspirator for the removal of secretions without interruption of the operator's work (Chevalier Jackson).

chronic esophagitis, ulceration, diverticulum, dilatation, paralysis, and various stenotic diseases, such as cicatricial stenosis following the ulceration of syphilis, typhoid fever, corrosive poisoning by bichlorid of mercury, caustic alkalis, acids, etc. Cicatricial stenoses are dilated under guidance of the eye with olives, divulsers, and silkworm bougies left *in situ* for a few hours. Obstinate cases may require endoscopic esophagotomy. Stenosis may be due to neoplastic involvement of the esophageal wall when the growth will be visible endoscopically, or the stenosis may be compressive, when the esophageal walls will be seen to be covered with normal mucosa, but the lumen is obliterated by the compression, the place of the lumen being occupied by a slit-like crevice, the long axis of which is often curved and is almost always at right angles to

the direction of pressure. Specimens of endo-esophageal growths may be removed for examination, and it is reasonable to hope that when patients shall be esophagoscoped early for any abnormal sensation in the esophagus the surgeon will have a fair chance to resect the intrathoracic esophagus. Spastic stenosis of the esophagus, especially hiatal and abdominal esophagismus (so-called cardiospasm), is not only easily diagnosticated esophagoscopically, but esophagoscopic divulsion of the abdominal esophagus will cure almost all cases. The esophagoscopic removal of foreign bodies is possible in all cases, no matter how large the foreign body. The mortality is practically nil in careful hands."

## Diseases and Injuries of the Chest, Pleura, and Lungs

**Traumatic Asphyxia** (*Pressure Stasis; The Ecchymotic Mask*).—This is a condition that occasionally arises when the trunk is subjected to sudden and violent compression (caught under an elevator, caught under a heavy box, cave in of earth, crushed in the rush of a panic, etc.). The compression may be upon the chest, the abdomen, or both; and in the majority of cases it has been very temporary. The discoloration arises immediately, and is manifested over the head and neck down to and sometimes below the clavicle. The hue is a violet lividity. There are a great many spots in the skin in which the color is much deeper, which have been supposed to be hemorrhages, and similar spots exist on the labial, buccal, glossal, palatine, and pharyngeal mucous membranes. Subconjunctival hemorrhage is the rule. There may be bleeding from the nose, mouth, and ears. There is brief or prolonged unconsciousness, circulatory and respiratory depression, sometimes cough and bleeding from the lungs. There has never been a reported instance of intracerebral hemorrhage. There are (rarely) convulsions, there is no paralysis and no delirium. The lividity may clear up in a few hours. The spots seldom fade for days. The spots do not fade at all from pressure, the livid area fades but slightly.

If death occurs, it results from associated injuries. The condition in the cases without severe associated injuries has soon disappeared, and entire recovery has followed. The view generally taught is that traumatic asphyxia is the result of compression of the abdominal veins, causing distention of the superior cava and its tributary veins, this region of the body showing the effect more than the limbs, because of the comparative feebleness of the valves (Villemin). The blood is forced back along the veins and into the capillaries, and capillary paresis ensues. One thing is sure, and that is, that the condition is particularly apt to arise if the patient struggles violently to free himself from the compression; and many observers have held the opinion that actual vascular ruptures take place. There are certainly some cases, however, in which there is simply great venous and capillary distention in the skin without rupture, because pieces of skin have been excised and microscopical examination has indicated that there had been no blood effused. The selection of the face and neck as the regions of discoloration is due, perhaps, to absence or incompetence of valves in the jugular and facial veins. The fluid state of the blood which has been noted occurs in all forms of asphyxia. (See Winslow, "Medical News," Feb. 4, 1906; Birge, "Cleveland Medical Journal," Sept., 1905; Beach and Cobb, in "Annals of Surgery," April, 1904; Villemin, "Bull. et mém. de la Soc. Chir. de Paris," No. 9, 1906.) Despard ("Annals of Surgery," June, 1909) has reported 1 case and collected 17 from recent literature. Ettinger in 1907 collected 36 cases and added 1 of his own ("Wien. klin. Wochen.," 1907, vol. xx). I have seen 2 cases.

**Pleuritic effusion** may arise from the lodgment of foreign bodies, from injury by fragments of a broken rib, from tumors, and from inflammation of the

lung, but most usually is due to pleuritis. The commonest cause of primary pleuritis is tuberculosis. Inflammatory effusion is nearly always unilateral (except in tuberculous pleuritis; but even in this form it is often one-sided in origin).

The **signs** of pleuritic effusion are: dulness on percussion over the area of effusion, this dulness, when the patient is erect, being at the lower part of the chest and ascending higher posteriorly than anteriorly (alteration of position alters the situation of the dulness); the intercostal spaces are widened, the intercostal depressions are obliterated, the intercostal muscles are rigid, and their rigidity lessens the mobility of the ribs (Przewalski). No breath-sounds can be detected in the area of percussion flatness when the collection of fluid is large, but in small effusions deeply situated the breath-sounds are often audible; the percussion-note above the liquid is hyperresonant or tympanitic, and is often associated, at the edge of the liquid, with a friction-sound; posteriorly, high up and near the spine, there are bronchial respiration and bronchophony. In cases of pleurisy with effusion pain almost or quite disappears with the advent of effusion, dyspnea comes on, and the patient lies upon the diseased side. Cough always exists if there is pleuritic effusion, and fever is usually present. In serous effusions the diagnosis may be confirmed by the aseptic introduction of a clean aspirating-needle. Ramond has pointed out that in serofibrinous pleurisy the iliocostal and longus dorsi muscles are always noticeably enlarged on the side of the effusion ("Bull. de la Soc. Méd. des hôp.," 1910, vol. xxvii).

The **treatment** in this stage is to discontinue arterial sedatives and to stimulate if the circulation calls for it. The exudation is removed by the administration of salines, compound jalap powder, or elaterium. If these means fail, if the effusion is excessive, or if it is producing severe dyspnea, at once aspirate. Aspiration should be performed for an effusion which fills the whole chest, which produces great dyspnea, or which has lasted for three weeks. In tuberculous pleuritis early aspiration is not advisable, but aspiration should be performed if the fluid becomes purulent, if the effusion displaces the heart considerably, and if it adds notably to the dyspnea. If a non-tuberculous effusion becomes purulent, the proper procedure is incision, resection of a portion of a rib, and drainage.

**Empyema** is a collection of pus in the pleural cavity displacing and compressing the lung. It may begin suddenly, but rarely does so. Among the causes of empyema are those of serous effusion. Empyema is due to infection of the pleura, and in every case a bacteriological study should be made of the pus to discover the causative bacterium. The pneumococcus is the causative micro-organism in many of the cases which follow pneumonia. Pneumococci live but a short time, and in empyema due to pneumococci these micro-organisms may not be discoverable when the pus is evacuated. In most cases of empyema streptococci or staphylococci can be found in the pus. These micro-organisms may appear in an empyema induced originally by pneumococci (Stephen Paget). In empyema developing during or after typhoid fever typhoid bacilli may be discovered. In putrid empyema various bacteria are found. Bouchard thinks acute empyema has a special organism. Bacilli of tuberculosis are present for a time at least in tuberculous empyema, but may disappear, and are particularly apt to after mixed infection with pyogenic bacteria. In adults many cases, in children few cases, are secondary to tuberculosis. Empyema may be due to a wound or contusion, an attack of pneumonia, tuberculous pleuritis, phthisis, influenza, pyogenic infection of a serous effusion, caries of a rib, specific fevers, especially typhoid, peritonitis, abscess of the liver, suppurating hydatid cyst of the liver, subphrenic abscess, malignant disease of the pleura, gangrene of the lung, and pneumothorax.

Pneumonia is the common cause in children. In them we find streptococci in only 15 per cent. of the cases, and sterile fluid in 20 per cent. In 65 per cent. of the cases we find pneumococci. In adults the pus is often thin and very putrid. In children the pus is nearly always thick. Pneumococcic pus is fairly thick, contains some clots, and is white or greenish white.

**Acute Empyema.**—The *signs* are, in reality, those of pleuritis with effusion —viz., dulness on percussion, absent breath-sounds over the purulent matter, bulging of the intercostal spaces, and sometimes edema of the skin of the chest. The *symptoms* of acute empyema are dyspnea, pallor, cough, sweats, chills, and usually irregular fever, but fever may be absent. There is marked leukocytosis. The fingers may become clubbed. An empyema may pulsate, particularly an empyema of the left side. The cause of *pulsating empyema* has been much debated. The most probable explanation is that of W. J. Calvert ("Am. Jour. Med. Sciences," Nov., 1905). He says the requirements for such a condition are: "A firmly fixed, pulsating organ; distention of the pleural sac with fluid or air or solid material; and a collapsed condition of the lung." In all probability the thoracic aorta is the "fixed pulsating organ." The left parietal pleura is in close relation with the aorta, and most pulsating empyemas are left sided. The right parietal pleural may be "pushed against the aorta." If a lung contains air, it is elastic and compressible to a degree that enables it to absorb the aortic impulse; if it is collapsed and solid it cannot, and aortic pulsations are transmitted to fluid in the pleural cavity and the thoracic wall pulsates. A neglected empyema may break into the lung, esophagus, or pericardium, through an intercostal space, or may point in the lumbar region. When an empyema is pointing externally, the condition is called *empyema necessitatus*. A *total empyema* is a condition involving the entire pleural sac. In a *partial* or *localized empyema* the purulent matter is encapsulated. A *closed empyema* is one in which no opening has been made by the surgeon and no opening has formed spontaneously. In a closed empyema the pus is rarely putrid; in an *open empyema* the pus is often putrid. After an empyema ruptures spontaneously it rarely heals without surgical interference, a *pleural fistula*, as a rule, persisting. A *subphrenic abscess* may follow an empyema. When an empyema ruptures into a bronchus, pneumothorax arises, as a rule. Empyema may cause death by compression of the heart and lung, pulmonary embolism, pericarditis, peritonitis, cerebral embolism, cerebral abscess, septicemia, exhaustion, or rupture into a bronchus. Empyema is particularly fatal in childhood. In empyema of the early months of life nearly all the victims die. In those under one year of age 50 per cent. of the affected will die (Holt). In older children 30 per cent. die.

A small empyema due to pneumococci occasionally, though very rarely, undergoes spontaneous cure, the pus being absorbed (Stephen Paget).

A small empyema is occasionally cured by encapsulation by fibrous tissue.

Under exceptional circumstances even a large empyema may be cured by breaking externally or into a bronchus.

Empyema is so rarely cured spontaneously that it does not do to trust to Nature at all, and practically almost all cases die without surgical treatment.

*Double empyema* is a rare and extremely fatal condition.

**Chronic empyema** may follow acute empyema, or the condition may be chronic from the beginning. It is more common in adults than in children. In chronic empyema the lung is compressed, shrunken, does not expand and is strongly adherent, and the pleura is very thick. In some cases the pleura is over an inch thick. This thickening is brought about by the deposition of layer after layer of fibrin. In not a few cases a chronic empyema succeeds an acute one. Sometimes chronic empyema is maintained because a drainage-tube has slipped into the pleural cavity and remains lodged.

**Treatment of Empyema.**—The treatment is purely surgical, and the earlier it is applied the better. To delay allows the pleura to thicken and permits adhesions to form, conditions which prevent lung expansion and retard or even prevent cure. The results of operation for chronic empyema are better in children that are not very young than in adults. In acute empyema the prognosis is better in small collections than in large; in recent than in advanced cases; in pneumococcus empyema than in empyema due to other organisms. The surgical methods for various stages of empyema comprise aspiration, incision, rib-resection, the operation of Schede, the operation of Estlander, and the operation of Fowler (see pages 908 to 912 inclusive).

I do not believe in any of the appliances to drain with a small tube and maintain negative pressure in the chest to favor lung expansion. The Thiersch method consists in introducing a trocar into the cavity, passing a Nélaton catheter through the trocar, and removing the trocar. The external end of the catheter is attached to an easily collapsible rubber tube, the other end of which is in a bottle of water. The catheter is fixed to the side by rubber tissue and rubber plaster. "The theory is, that when the patient expires, the pus runs out through the tube, and on inspiration the collapsible walls of the tube are sucked together and prevent the entrance of air, and cause negative pressure in the chest, favoring expansion of the lung" (Lund, "Jour. Am. Med. Assoc.," August 26, 1911). In any method of suction so far devised the tube is so small that it blocks up, and though the drainage-tube may at first make an air-tight joint with the chest, it always loosens sooner or later and leaks air.

In acute empyema general practitioners are very apt to *aspirate*, and yet aspiration is almost never curative. It may cure a pneumococcus empyema in a child and an encysted empyema, but even in these it will usually fail. Aspiration is not to be considered a method of curative treatment. It is to be regarded as the surgical treatment only in a tuberculous empyema in a young person with rapidly progressing phthisis, because in such a case incision will probably prove fatal (Lockwood). It is a very useful diagnostic expedient, and enables the surgeon to prove the existence of pus, and the pus which is obtained can be examined bacteriologically. In a very large effusion it is wise to aspirate and withdraw part of the effusion a short time before operating. This enables the patient to take an anesthetic with greater safety and obviates the danger attending the rapid evacuation of a large amount of pus.

In a recent empyema, *incision* and *drainage* or *rib resection* and *drainage* will often cure the case, and yet many of the results are unsatisfactory. In some cases the discharge ceases and yet pulmonary function is not completely restored. In other cases a pleural fistula persists. If a profuse discharge is maintained, amyloid disease may arise. An acute empyema is to be drained by intercostal incision or by resection of a rib (see page 908). A chronic closed empyema is drained in the same manner, and if the lung will not fully expand and remains stationary for one year, Schede's or Estlander's operation is required. An open chronic empyema, in which the lung will not expand, requires the operation of Schede, Estlander, or Fowler (see pages 910 and 911). The results are best in children almost in the teens. Extensive decortication is sometimes impossible, and then Ransohoff's operation may be done. He calls it *discission of the pulmonary pleura* (see page 912). When there is an external opening which persists and which joins a long, narrow cavity, the condition is spoken of as *pleural fistula*, and pleural fistula is often produced by the prolonged use of a drainage-tube and sometimes by caries of a rib. Even if there is no opening on the cutaneous surface, there may be one into a bronchus. A pleural fistula may sometimes be cured by dilatation of the sinus. If this fails, and it usually does, it is the custom to resect one or more ribs.

Before resorting to operative treatment in chronic empyema we may try, in

some open cases at least, the injection of bismuth paste. We owe this method to Dr. Emil Beck. This plan will sometimes succeed. Ochsner has strongly commended it ("Annals of Surgery," July, 1909). The injection is made by a glass syringe and the material should fill but not stretch the cavity. After injecting the paste the external opening of the fistula is plugged with gauze. The treatment is begun with solution No. 1 (1 part of subnitrate of bismuth devoid of arsenic and 2 parts of yellow vaselin).

This is used every second day until pus has practically disappeared. Then solution No. 2 (30 parts of bismuth, 60 parts of yellow vaselin, and 10 parts of paraffin) is substituted for it. The injections are made often enough to keep the sinus and pocket full. They are made at first every day, then every other day, and so on until, finally, every eighth or tenth day is often enough. If poisoning should arise from these injections Beck advises the injection of olive oil at a temperature of 110° F. to dissolve the paste and favor its evacuation. McKelvey Bell recommends a paste which is more stimulating than Beck's. He takes 1 oz. of subnitrate of bismuth and 2 oz. of white petrolatum, dissolves over a hot-water bath, and stirs until cool. Then, while stirring, he adds 72 gr. of iodoform (5 per cent.). This must be kept in a dark place to prevent decomposition of the iodoform ("New York Med. Jour.," May 4, 1912). (See page 623.)

**Non-traumatic Pneumothorax.**—By the term "pneumothorax" is meant the presence of air in the pleural cavity. As a rule, besides air there is serous fluid or pus. It may be due to the rupture of an empyema into a bronchus; to the rupture into the pleural sac of a tuberculous area, an area of gangrene, an abscess of the lung, an air-cell in a state of emphysema, or of pulmonary tissue softened because of hemorrhagic infarction. The condition is by no means uncommon in phthisis. In 473 autopsies held in the Henry Phipps Institute of Philadelphia upon persons dying of pulmonary tuberculosis there were 41 cases of pneumothorax, or in 8.6 per cent. of the autopsies this lesion was found (J. M. Cruice, "Med. Record," Sept. 23, 1911). In 60 per cent. of the cases the lesion is on the left side. It is inaugurated by cough, straining at stool, vomiting, or lifting. It is more frequent in men than in women and is most common between the ages of thirty and forty. The immediate effect of the entrance of air into the pleural sac is to compress the lung, the degree of compression being in proportion to the amount of air present. In severe cases the lung is squeezed against the vertebral column, and the heart and diaphragm are displaced. In a right-sided case the liver may be displaced. In some cases, when the admission of air does not continue, the amount set free in the pleural sac is absorbed. In most cases pyopneumothorax (empyema) follows.

The **symptoms** usually arise suddenly (in three-fourths of the cases), and consist of distressing dyspnea, pain in the chest, lividity, and rapidity and weakness of the pulse. In some cases of phthisis the symptoms are gradual and not very severe. There may be pain and dyspnea or only dyspnea. In the latter case, if dyspnea existed before, the accident may be unrecognized. It has been pointed out that occasionally in phthisis pneumothorax seems actually to benefit the tuberculous area in the lung. There is nothing characteristic in the attitude. The patient usually lies on the opposite side, but may lie on the side of the trouble or on the back. He may sit erect or semi-erect. He may place himself in a knee-chest position. The physical signs of pneumothorax are as follows: The affected side of the chest is often bulged, movements are lessened or absent, and the heart is displaced, especially if the condition affects the left side. Palpation discovers that vocal fremitus is lessened or absent. On auscultation it is found that the breath-sounds are very feeble or absent in 70 per cent. of cases. Occasionally they are amphoric, bronchovesicular, or cavernous. The voice may be transmitted as a metallic sound (this is present in about half the cases), râles may sound metallic, and

on coughing there may be metallic tinkling. The percussion-note is hyperresonant or tympanitic in 95 per cent. of cases. Very seldom it is normal. In some rare cases the percussion-note is dull. When fluid gathers, there is a positively dull note on percussion over the fluid.

**Treatment.**—Osler says the treatment should be the same as that for pleurisy with effusion. In many cases it is wise to perform paracentesis without suction to remove air and serous effusion. If pus forms, a rib should be resected and a tube inserted (see Empyema). In pneumothorax occurring during chronic phthisis operation is of service. In cases with rapidly progressive phthisis it is practically useless.

If the opening into a bronchus or air-cell remains patent, aspiration will not get rid of air; the air will enter into the pleura as rapidly as the aspirator removes it. Incision has dangers of its own: the diaphragm is flapping during respiration and may be injured (Fowler), and when the pleura is opened there is a great alteration produced in the air-pressure in the chest, and the patient may "drown in his own secretions." After incision irrigation is not justifiable, because the fluid may enter a bronchus and produce suffocation (Fowler).

West's rules[1] are those I follow. West says early incision is dangerous. In an early stage use paracentesis without suction. This will often relieve the patient. If paracentesis does relieve him, wait a while and perhaps repeat the operation if the symptoms again become severe. If paracentesis does not relieve, incise, resect a portion of a rib, and drain. If pus forms, an incision must be made and a portion of a rib be resected to afford exit to the fluid.

Fowler pointed out that if the lung is bound down by adhesions, incision is dangerous but justifiable. Operation at the proper time often prevents the lung being bound down by adhesions.

**Acute Traumatic Pneumothorax.**—This is produced by the sudden admission of a quantity of air into the pleural cavity as a result of a wound of the chest wall. A small quantity of air, or the gradual introduction of considerable air, does not, as a rule, produce very serious symptoms. The sudden admission of a quantity of air causes very dangerous symptoms and even death. A quantity of air may be admitted rather suddenly as a result of an accident or during the performance of a surgical operation which opens the pleura. It sometimes arises during the removal of tumors from the chest wall, during operations upon the lung, and during empyema operations. As a rule, when pulmonary adhesions exist, dangerous symptoms do not arise, even when the pleura is widely opened, and adhesions exist in 25 per cent. of empyema cases seen by the surgeon.[2]

It was formerly taught whenever the pleura is opened there is a strong tendency to the development of pneumothorax, but West has shown that the surfaces of the pleura often cohere with a force superior to pulmonary elasticity, and in such cases pneumothorax does not arise.

In surgical operations in which it is necessary to open the pleura widely (as in operation for sarcoma of the chest wall) the surgeon endeavors to prevent acute pneumothorax, which may prove fatal. This may be done by operating in the Sauerbruch negative pressure chamber (see page 905) or by applying positive pressure (see page 905).

**Symptoms.**—When the pleura is opened during an operation or by an injury, the symptoms may be trivial and transitory, may be tolerably severe, may be extremely grave, and the patient may quickly die (Quénu and Longuet). Rudolph Matas[3] sets forth the symptoms as presented by the French observers.

[1] "British Medical Journal," Nov. 27, 1897.
[2] Rudolph Matas, "Annals of Surgery," April, 1899.
[3] Ibid.

The mild symptoms are weak, slow pulse and irregular, noisy respiration.

The severe symptoms are slow pulse, slow and irregular respiration, and dyspnea, continuing after the anesthetic has been withdrawn.

The grave symptoms are cyanosis; collapse; small, weak pulse; shallow and noisy respiration; and spells of syncope. Death may occur suddenly from inhibition, or later from mechanical asphyxia (Matas).

**Treatment.**—Various plans have been adopted: suturing the opening in the pleura; plugging the opening; pulling the diaphragm into the wound in the chest wall and suturing it; and grasping the lung and suturing it to the wound. Whenever the pleura is to be widely opened, operate in a Sauerbruch chamber or use a positive pressure apparatus, and when the operation is complete, suture the lung to the margin of the opening in the pleura with a continuous catgut suture. The apparatus for insufflation anesthesia accomplishes the object. Parham, Keen, and the author followed this plan, using the Fell-O'Dwyer emergency apparatus, and the lung was kept from collapsing.[1] This apparatus is shown in Fig. 567.

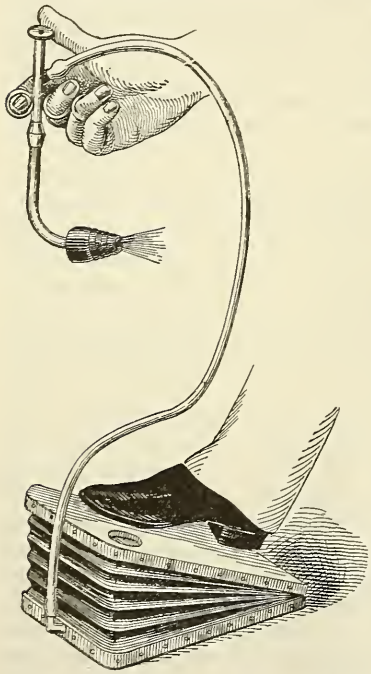


Fig. 567.—The Fell-O Dwyer apparatus. This illustration shows an early model; since then the bellows has been improved by the addition of a strong wooden frame, which holds it steadily, and is provided with a long arm that acts as a powerful foot-piece for compressing the machine with the least amount of muscular effort.

O'Dwyer's tube is introduced into the glottis as is the tube in intubation, and is attached to a bellows, the lung is inflated, respiration is maintained by the use of the bellows, and collapse, with all its dangers, is avoided. The modern positive pressure apparatus or the apparatus for insufflation anesthesia is better (see page 905). So is the pulmotor (see page 867).

**Contusions and Wounds of the Chest.—Contusions.**—A contusion may be trivial and limited to the superficial parts of the chest wall; it may involve the muscles; it may be associated with fracture of the ribs or sternum or with visceral injury.

*Symptoms.*—In an ordinary contusion without visceral injury there are considerable pain, discoloration, and often much swelling. The patient prefers to lie upon the back and the respiration is abdominal. After a severe blow upon the chest there is great shock and may even be instant death. The condition of shock so produced is called *concussion of the chest.* After a severe blow upon the chest a limited area of inflammation may arise in the pleura (*traumatic pleuritis*). Severe visceral injury is announced by positive symptoms. A *contusion of the lung* causes extravasation of blood and leads to pain, cough, expectoration of bloody mucus, dyspnea, and possibly distinct hemophthisis. Over the contused region the percussion-note is dull and on auscultation crepitus is audible. It may be mistaken for phthisis, but complete and early recovery soon dispels this fear. *Traumatic pneumonia* always follows. This usually involves a limited area of lung tissue, but genuine croupous pneu-

[1] F. W. Parham's paper on "Thoracic Resection for Tumors Growing from the Bony Walls of the Chest." Read before the Southern Surgical and Gynecological Association, November, 1898.

monia may arise after injury of the chest even when no rib was broken. The physical signs and symptoms are not evident until two or three days after the accident (Sir Thomas Oliver, in "Brit. Med. Jour.," April 30, 1910). Traumatic pneumonia may be caused by other things than external violence, viz., inhalation of illuminating gas, of dust, or of a foreign body.

In **rupture of the lung** the physical signs are dependent on the extent and situation of damage. A minute rupture would not produce definite physical signs. If the lung is ruptured and the pulmonary pleura is not, there will not be pneumothorax, but there may be cellular emphysema first becoming evident at the root of the neck. If the pleura is torn as well as the lung, there will be pneumothorax and hemothorax, the amount of hemorrhage depending upon the situation and extent of the injury.

Robt. G. Le Conte ("Annals of Surgery," March, 1908) points out five ways in which rupture may be caused: (1) *Bruising* (simply causes subpleural ecchymosis); (2) *bursting* (violent force—a lung unable to empty itself of air is broken as an inflated paper bag is broken by a sharp blow); the question as to whether the glottis must be closed for such an injury to occur is not settled; (3) *penetration* by a green-stick fracture of a rib; (4) *compression* of the lung against some resistant structure; (5) *tearing* (when the lung has previously been adherent to the wall of the chest).

These five causes might be sententiously designated as bruising, bursting, puncturing, squeezing, and tearing.

The *symptoms* are shock, dyspnea, cough with or without bloody expectoration, rapid and irregular pulse, cyanosis, emphysema (appearing first over the region of injury if a broken rib penetrated the lung, and first at the root of the neck is the lung is ruptured, but the pleura is not), and in some cases pneumothorax and hemothorax (Le Conte, Ibid.).

**Rupture of the diaphragm** causes pain, dyspnea, and often vomiting. The stomach or intestine may pass into the pleural sac. If this happens, there will be a tympanitic percussion-note over the displaced viscus and symptoms will vary with the viscus involved. Such a diaphragmatic hernia may become strangulated (see page 1152). In a case in the Jefferson Medical College Hospital, in which the stomach passed into the left pleural sac, there were persistent vomiting, violent pain in the chest and upper abdomen, great thirst, and displacement of the apex-beat. The condition may be confused with pulmonary rupture causing pneumothorax, but in rupture of the diaphragm persistent nausea and vomiting are prominent features; whereas, in pulmonary rupture, if they exist at all, they are early and temporary; further, in rupture of the diaphragm the tympanitic percussion-note is not found over the pleural apex, but in pulmonary rupture with pneumothorax a tympanitic note is found all over the entire pleural cavity (Le Conte, Ibid.).

**Treatment of Contusions of the Chest.**—A contusion of the chest wall is treated as directed in the section on Contusions (see page 259), and the chest is strapped with adhesive plaster, as in the treatment of fractured ribs. In concussion of the chest the treatment for shock is applied. It may be necessary to employ artificial respiration for a time. If a diaphragmatic hernia is diagnosticated, the abdomen should be opened, the displaced viscera restored to their proper abode, and the diaphragm sutured. The diaphragm may also be reached by resecting several ribs and opening the pleural sac. In contusion of the lung cold is applied to the chest early in the case, and any inflammation which arises is treated according to general rules. In rupture of the lung the case may be treated expectantly, but dangerous and continued bleeding or pneumothorax may render surgical interference necessary. For pneumothorax paracentesis without suction is employed. If this fails, it may be repeated. If it fails again, resect a portion of a rib and put in a tube. If bleeding is danger-

ously profuse resect a portion of a rib and insert a drainage-tube into the pleural cavity.

**Wounds of the Chest.**—Non-penetrating wounds are not particularly grave, and are treated according to general principles, the chest being immobilized. Penetrating wounds are extremely grave, as viscera are apt to be injured. In such a wound an intercostal artery may be severed or the internal mammary artery may be divided. An intercostal artery is rarely divided unless a rib is broken. The surgeon should always examine carefully in order to determine whether an intercostal artery or the internal mammary artery has been divided, and, in doing so, should bear in mind the admonition of Matas—that is, the bleeding from these vessels may be internal, the blood collecting in the pleural sac. The pericardium or heart may be injured (see page 404). A wound of the pleura is usually, but not always, associated with a wound of the lung. If the lung is injured, there are usually great shock, pain in the chest, dyspnea, and cough. In a large wound, damage to the lung will be indicated if air is sucked into the wound during inspiration and expelled during expiration, and if blood is forced out of the wound by coughing. The lung may be visible or may protrude (*protrusion of the lung*). In a small wound it is often difficult and sometimes impossible to determine whether the lung has been injured. Pneumothorax with pulmonary collapse proves it has. Severe hemothorax strongly suggests it. Spitting blood does not prove it. In some severe cases there is no hemoptysis; in some slight bruises the amount of blood coughed up is large. Emphysema about the wound does not prove lung injury. An incised wound of the lung is apt to produce rapid death from hemorrhage, especially if the wound is at the root of the lung. A pistol-bullet or a sporting-rifle bullet may not be productive of great primary hemorrhage; but infection will probably follow, and secondary hemorrhage will be apt to occur. The modern military-rifle ball passes through, rarely lodges, is aseptic, and often produces astonishingly little trouble. A pistol-bullet and an old-time rifle-bullet may lodge or may perforate.

*Treatment.*—Bring about reaction as previously directed (see page 262).

An incised wound of the chest, if large, should be carefully inspected. If the wound is small, cut down layer by layer until the depths of the wound are reached. Disinfect the wound and arrest hemorrhage. If the pleura is not open, proceed according to general rules. If the pleura is found to have been opened, suture it with catgut, close the superficial wound, dress with gauze, and immobilize the chest wall.

The above proceeding should be carried out whether it is or is not believed that the lung has been damaged, provided there is no pneumothorax and no violent hemorrhage. What course shall be pursued if the lung has been injured by a stab? If hemorrhage does not threaten life and there is no pneumothorax, the patient is kept at rest and observed. If pneumothorax occurs, the pleural sac must be drained by means of a tube, because clots must be evacuated and infection should be anticipated. If hemorrhage into the pleural sac persists, active measures become necessary. The use of ice-bags and drugs is but waste of time. Some surgeons believe that the mere closure of the external wound leads to arrest of hemorrhage, blood accumulating and making pressure. It is true that hemorrhage often ceases after suturing or plugging a wound and strapping the chest, but it is not probable that it ceases because of these measures. Blood in the pleura usually remains unclotted for several or many days. Further, as Le Conte[1] shows, as the blood is forced against the root of the lung the right heart is engorged, the blood-pressure is raised, and the bleeding continues.

Bleeding from the lung can often be arrested by inserting the end of a drain-

[1] "Annals of Surgery," April, 1899.

age-tube into the pleural sac. In cases in which a drainage-tube is inserted into the pleural cavity and free drainage established the pleura is immediately filled with air, and the muscles of respiration are kept from acting on the lung. The lung contracts by its own elastic tissue, as well as by the pressure exerted by the pneumothorax, and at the same time the presence of the air favors clotting in the severed vessels.[1] Baudet maintains that all grave wounds of the lung should be operated upon. If the insertion of a tube fails, or if the bleeding is rapid and obviously seriously threatens life, several ribs must be rapidly resected and the bleeding part explored. In some cases the bleeding may be arrested by ligation, in some cases by packing a small wound with gauze, in some cases by the suture-ligature. Two cases of lung suture have been recorded by Philadelphia colleagues (Jopson, in "Annals of Surgery," 1906, vol. xliii; Kelly, in "Annals of Surgery," 1910, vol. li). In a violent secondary hemorrhage following a gunshot-wound of the lung I packed the entire pleural cavity with sterile gauze to obtain a base of support, and arrested the bleeding by carrying iodoform gauze directly against the oozing surface.[2] The man recovered. I did the same thing on a Chinaman suffering from a gunshot-wound of the lung. The hemorrhage was arrested and he lived three weeks, dying finally from pericarditis. After directly arresting hemorrhage from the lung, turn clots out of the pleural sac and insert a drainage-tube. In a perforating wound inflicted by a bullet, reaction must be brought about, the wound dressed antiseptically, the chest strapped, and the patient kept quiet. If pneumothorax occurs, the pleura should be drained with a tube. If hemorrhage occurs, it should be met as directed above. In a wound in which the bullet has lodged, an examination should be made to see if the bullet is under the skin, and if it is, it is removed after the patient has reacted. It should always be borne in mind that a pistol-bullet may be deflected by a rib or may pass from the front to the back part of the chest by making a burrow under the skin (*a contour wound*). If a bullet is lodged, no attempt should be made to remove it unless an operation must be done for bleeding, unless the bullet causes trouble, or unless it is felt under the skin. Under no circumstances conduct a long search for a bullet. If emphysema of the chest walls is moderate, strapping or a bandage will control it; if it is great, make multiple punctures and then apply pressure. In protrusion of a portion of the lung try to restore the protrusion; but if restoration is impossible or if gangrene seems likely to occur, ligate the base of the protrusion with silk and cut away the mass.

Moller ("Archiv. f. klin, Chirurgie," 1909–10, vol. lvi) collected 26 cases of gunshot-wounds, which were operated upon, and 11 died (42 per cent.). In 20 suturing was done, with 7 deaths. In 2 the wound was sutured to the pleural opening, with 1 death. In 2 lung resection was followed by death. One, in which pleural packing was used, recovered. He collected 10 stab-wounds: 7 were sutured, with 1 death. In 3 the pleura was packed and all recovered.

**Wounds of the Chest Involving the Diaphragm.**—In such a case abdominal viscera may pass through the wound in the diaphragm and appear in the wound of the chest. If there is no indication of a wound of the abdominal viscera some surgeons would suture the wound of the diaphragm by way of the thorax. If there are indications of injury of abdominal viscera the abdomen must be opened. Personally, I prefer to open the abdomen in every chest wound implicating the diaphragm, even when symptoms of injury to abdominal viscera are not as yet manifest.

**Occluding Pulmonary Embolism.**—By this term we mean an embolism which completely blocks the pulmonary artery or some of its chief branches. Such cases occasionally follow a surgical operation. They are more frequent

[1] Le Conte, in "Annals of Surgery," April, 1899.
[2] The author, in "Annals of Surgery," Jan., 1898.

than used to be thought. The clot is derived from a vein. The calamity is most apt to occur between the second and fourth weeks after an operation (Bartlett and Thompson, in "Annals of Surgery," May, 1908). Pulmonary embolism may arise from fractures, inflamed hemorrhoids, or inflamed veins anywhere, and during or after pneumonia, erysipelas, typhoid fever, and any acute infection. In malignant endocarditis and mitral stenosis small emboli are common. If the pulmonary artery is completely blocked death occurs at once. If one branch only is blocked the patient may recover if the heart is sound and strong. If the artery proper is partly blocked the patient has a period of suffering and dyspnea before death, but death is sure to occur from subsequent complete blocking.

Bartlett and Thompson (Ibid.) report 22 cases of occlusive pulmonary embolism, 20 of which were fatal. The greatest cause in a surgical operation seems to be varicose veins, especially about an abdominal or pelvic tumor. I am convinced that most sudden deaths in infected cases are due to pulmonary embolism.

The **symptoms** of a mild case of embolism are rapid pulse, dyspnea, after some hours dulness of the base and impaired breath sounds, and, perhaps later, spitting of blood. In a real occluding embolism there are sudden collapse, cyanosis or pallor with livid lips, the pulse at the wrist is absent or very rapid, and irregular. There are pain in the chest, intense dyspnea, dilated pupils, and early unconsciousness. The right side of the heart distends greatly and the second sound is accentuated in the pulmonary area.

Schumacher in discussing these cases says there are three classes of them: Those in which almost immediate death occurs; those in which death occurs in a few minutes, and those which last much longer. In the latter cases a main branch becomes blocked and total obstruction is gradual (Willy Meyer, in "Annals of Surgery," August, 1913). Only the latter cases are suitable for operation. The surgeon must bear in mind the danger of embolism and endeavor to prevent it in operations by handling and exposing viscera as little as possible, by applying ligatures above all clots in veins and all varicosities, and removing the affected structures. Quénu proposed to lessen the danger of postoperative thrombosis by administering citric acid before operation. This agent lessens the coagulability of the blood. Tuffier fears this remedy, believing that it makes an existing thrombus more apt to break up and form emboli ("Presse Médicale," April 20, 1910).

**Treatment.**—In small, non-occluding emboli, in which condition the only symptoms are dyspnea, pain, rapidity of pulse, and, after some hours, dulness at the base and impaired breath sounds, give stimulants, dry cup the chest, and administer morphin if there is no cyanosis. In a very severe case death is rapid and there is nothing to do medically.

Trendelenburg, of Leipzig, suggests surgical treatment (German Congress of Surgery, 1908). He points out that death is not always sudden (in 7 cases out of 9 the victim lived from ten minutes to one hour), and advises opening of the pulmonary artery and removal of the embolus. The first patient operated on died before the completion of the operation. "Since then it was done, twelve times in all, at the Leipzig clinic, without one permanent recovery" (Willy Meyer in "Annals of Surgery," August, 1913). One patient lived four days; 1 died on the table; 1 died in fifteen hours from cardiac failure; 1 lived for thirty-seven hours and died of reactionary bleeding from the internal mammary artery. Kruger did one operation for embolism. The early result was apparently successful, but the patient died of infection on the eighteenth day ("Zentral. für Chir.," May 22, 1909). In Sauerbruch's clinic at Zurich the operation has been performed four times without a recovery (Willy Meyer, Loc. cit.).

Trendelenburg thinks that the surgeon has about fifteen minutes in which to

work. The operation is done under differential pressure. The pleura is opened, then the pericardium. A rubber tube is passed through the transverse sinus of the pericardium and the aorta and pulmonary artery are constricted. This constriction must be released within forty-five seconds. The artery is opened, the clot removed, the arterial wound clamped, and the elastic constriction released. The arterial wound is then sutured.

**Abscess of the lung** may follow ordinary pneumonia. It is more apt to follow aspiration-pneumonia. It is usually caused by streptococci or staphylococci, but it may result from pneumococci or colon bacilli. These germs may reach the pulmonary tissue by direct entrance from adjacent organs, by way of the blood, or by way of the bronchi and alveoli. Osler[1] tells us that pulmonary abscess may result from the aspiration of septic particles after "wounds of the neck, operations upon the throat," and suppurative lesions of the nose, larynx, or ear. Aspiration-pneumonia may develop when there is difficulty in swallowing from any cause, when there is profound exhaustion, and when there is palsy or incoördination of any of the muscles of deglutition. Cancer of the esophagus may be a cause; so may perforation of the lung by an abscess, wound of the lung, impaction of a foreign body in the lung, suppuration about a focus of tuberculosis or a metastatic abscess. A pulmonary abscess may be of trivial size or it may be very large, involving an entire lobe. There may be one abscess, several, or many. When suppuration results from aspiration-pneumonia or blood-infection, there are usually multiple abscesses.

**Symptoms.**—The expectoration is not frequent, but is profuse, and during a paroxysm mouthfuls are coughed up in rapid succession. The expectorated matter is sour or very offensive in odor and contains fragments or shreds of pulmonary tissue, which can be identified as such by the microscope. The patient lies upon the diseased side in order to keep the pus from running into the bronchi and causing cough. When the cavity fills and pus reaches the bronchi, violent cough and expectoration begin, continue until the cavity is partly or entirely emptied, and then subside, perhaps for several hours. If the abscess-cavity is large and full of pus, an area of dulness on percussion can be mapped out. When the pus is coughed out and the air enters, physical signs of a cavity are clear. The *x*-rays often show the situation of such a cavity.

The course of abscess of the lung is usually acute. There are fever of the hectic type, rapid loss of weight, weakness and rapidity of circulation, dyspnea, pallor, sleeplessness, and great weakness. Gangrene may arise; empyema or pyopneumothorax may develop; very rarely the abscess breaks through the chest wall; recovery may follow spontaneous evacuation or drainage by coughing up pus; death may result from exhaustion or secondary septic lesions. If operation is performed, from 70 to 80 per cent. of the patients will recover.

The **treatment** is purely surgical (*pneumotomy*). Make an incision over the cavity. Resect a portion of one or more ribs. Expose the pleura. If the two layers of the pleura are not adherent, suture them together and either wait two days, or surround the area to be incised by a coffer-dam of gauze and then operate. If they are adherent, always proceed at once. Search for the abscess with an aspirating needle. When the cavity is found, open into it with the actual cautery and insert a drainage-tube (see page 912). The direct mortality is about 20 per cent.

**Gangrene of the Lung.**—This term means the putrefaction of a devitalized portion of pulmonary tissue. The tissue is devitalized by the action of pyogenic micro-organisms. Gangrene may follow abscess, bronchitis, or pneu-

[1] "Practice of Medicine."

monia, or may be due to diabetes, to embolism of a branch of the pulmonary artery, bronchiectasis, tuberculosis, malignant disease, wounds, or the lodgment of foreign bodies. Gangrene may be circumscribed or diffused. There may be one cavity, small or large, or multiple cavities may form. The gangrenous area putrefies, softens, and the softened matter may be expectorated, a gangrenous cavity being formed. In the rare cases which undergo spontaneous cure the cavity is, after a time, surrounded by fibrous tissue and obliterated by granulations. The mortality from operation is about 30 per cent.

**Symptoms.**—Expectoration occurs only now and then, but at each seizure a great quantity of matter is brought up and this matter is horribly offensive. Occasionally there is no expectoration. The patient, as in lung abscess, lies upon the diseased side. The expectorated matter is mucopurulent, contains particles or shreds of pulmonary tissue, bacteria, and altered blood. The fetor of the pus is much greater than is the fetor of the pus of an abscess. The breath is very foul. Physical signs may indicate either consolidation or a cavity. There are hectic fever, great exhaustion, deathly pallor, and diarrhea. Pulmonary hemorrhage is not unusual, and complications spoken of in the article upon Abscess may occur (see page 901). Recovery sometimes ensues, the cavity closing by granulation. Death may take place in a few days. Often the patient lives for weeks, being sometimes better and sometimes worse, dying finally from exhaustion or from the effects of a complication.

The **treatment** is to operate as for pulmonary abscess.

**Surgical Treatment of Pulmonary Tuberculosis.**—For a number of years past surgical thought has been actively directed toward placing on a scientific footing operations for pulmonary phthisis. The matter is still in a transition stage, and operations at present have a very limited field of application, although Sonnenberg and others have reported cures. Baglivi, in 1643, endeavored to tap and inject tuberculous cavities. Hastings and Stuckè did the same thing in the eighteenth century. Mosler, a number of years ago, attempted to treat cavities by introducing a trocar into the cavity and injecting permanganate of potassium solution through the cannula. Patients were not benefited by this procedure. The plan was revived by Pepper in 1874. The results are bad and the operation is dangerous. Hillier tried injection of corrosive sublimate into the lung parenchyma, but the effect of the injections was disastrous. Vidal advocates counterirritation by the actual cautery and maintains that congestion improves nutrition. When the strength of the patient is well preserved and the pulmonary lesion is circumscribed and slowly progressive, it may, in some few cases, be justifiable to perform an operation, open the cavity, and treat it directly (*pneumotomy*). That pneumotomy might be performed successfully was suggested to surgeons by observing that some patients recovered after sword-thrusts into the lung. Fowler said it is not justifiable to operate if the disease has come "to a standstill." The same surgeon stated that the only accessible region is bounded above by the clavicle, to the inner side by the manubrium, to the outer side by the lesser pectoral muscle, and below by the second rib.[1] This operation does not cure any one, but it may cause distinct improvement when there is hectic from an ill-drained cavity containing the products of a mixed infection. In an advanced case there is usually more than one cavity, and if there is, the operation is contra-indicated. Before attempting it, be sure the case is advanced and not incipient and that the cavity is single. Locate the cavity by auscultation, percussion, and the *x*-rays. (See Willard, "Jour. Amer. Med. Assoc.," Sept. 20, 1902.) Tuffiier collected 45 cases and only 1 was a success.

[1] See the very full and thoughtful article by George Ryerson Fowler on "The Surgery of Intrathoracic Tuberculosis," "Annals of Surgery," Nov., 1896.

Mauclaise[1] says that pneumotomy is justifiable only in circumscribed tuberculous cavities without peripheral infiltration and in pulmonary abscesses. Bronchiectatic cavities are usually multiple; they are exceedingly difficult to locate, and treatment by pneumotomy should not be attempted. In the teatment of pulmonary tuberculosis resection of the diseased area was proposed by Ruggi (*pneumectomy*). Tuffier successfully performed this operation. Surgeons, as a rule, do not believe in pneumectomy. Reclus[2] voices the general opinion when he says the operation is not required if the area of disease is very limited, as such a condition is frequently curable by medicinal means, and it does no good if the area of disease is extensive.

Only two methods of surgical treatment seem to have won a distinct place: one is the artificial production of pneumothorax; the other, some form of plastic operation upon the thorax to collapse and immobilize the lung.

**Artificial Pneumothorax.**—It has long been known that pneumothorax might benefit a tuberculous lung. Carson, of Edinburgh, in 1843 tried to create artificial pneumothorax by making a puncture in the visceral pleura to allow the passage of air into the pleural sac. Farlanini suggested the introduction of a gas into the pleural sac and reported a successful case in 1894. In 1898 Murphy began the use of nitrogen. There are now on record about 400 cases (Mary E. Lapham, in "Am. Jour. Med. Sciences," April, 1912). The method should only be used in unilateral cases which are not far advanced. It is used when the patient continues to grow worse in spite of medical treatment. The method is useless if there are adhesions, and is contra-indicated if diabetes exists or if there is uncompensated valvular disease of the heart (Lapham, Ibid.). Adhesions forbid because they prevent pulmonary collapse. It is maintained that the operation occludes the lymph-channels, lessens the absorption of toxins (shown by the abatement of fever), prevents bleeding, compresses the lung, arrests its mobility, approximates the walls of cavities, squeezes masses of bacteria out of the tubes, favors the development of fibrous tissue, and leads to healing of cavities.

Certainly it seems that the once widely condemned therapeutic pneumothorax is of real value in proper cases. Nitrogen is the best gas to use. It is non-irritant and remains long in the pleural cavity. Our aim is to keep the lung compressed for one year. The nitrogen is introduced by means of a special apparatus (Lapham, Ibid.). At first injections are made daily, then every other day, then twice a week, then twice a month. The injection is made "over an area where the breath sounds and resonance are best" (Lapham, Ibid.). There is little danger in the method, but trouble may follow. There may be shock, dyspnea, or spasm of the glottis from pleural reflex. Other dangers are: gas embolism, convulsions, edema of the lung, empyema, pulmonary abscess (if the lung is stuck), cerebral embolism, and emphysema of the chest wall. A danger is an unfavorable influence on the other lung. (See Lapham, Ibid.; Mauclaire, "Abstract in Brit. Med. Jour.," from "Jour. des prat.," xxvi, 1911; Editorial in "N. Y. Med. Jour.," Oct. 26, 1912; Sorge, in "Wien. klin. Woch.," No. xxxiv, 1912.) (See page 907.)

**Plastic Operations on the Chest Wall (Mobilization of the Thorax).**—Freund and others asserted that a rigid thorax is a cause of phthisis. Allis[3] suggested that in extensive unilateral tuberculosis of the lung resection of a number of ribs will favor cure by permitting retraction of the chest wall. This operation is founded on the belief that the chief element in effecting a cure is the formation and contraction of fibrous tissue. Pulmonary collapse and abolition of movements favor the formation of fibrous tissue. In this

[1] "La Tribune médicale," Sept. 21, 1893.
[2] "Revue de Chirurgie," Nov. 11, 1895.
[3] To State Med. Soc. of Penna., in 1891.

operation the pleura is not opened. Quincke, Landerer, and others remove portions of ribs with periosteum along the axillary line. Some surgeons remove portions of ribs and periosteum from directly over the lesion. In tuberculosis of the apex in a person with a narrow and rigid chest the followers of Freund remove the first rib. Freeman removes portions of ribs without periosteum and applies a truss to push in the chest wall ("Annals of Surgery," July, 1909). Friedrich, of Marburg, removes all the ribs from the second to the tenth inclusive and mobilizes the first rib (*thoracicoplastic pleuropneumolysis* with *subcostal apicolysis*). The pleura is not opened. He used to remove the periosteum, but does so no longer. He now leaves the periosteum so that after a time enough bone will re-form to prevent lung bulging on coughing. Sauerbruch, Wilms, and others have individual operations. The results of none of the above operations seem very encouraging (Mauclaire, "Abs. in Brit. Med. Jour.," from "Jour. des prat.," xxvi, 1911; Editorial in "N. Y. Med. Jour.," Oct. 26, 1912; Sorge, in "Wien. klin. Woch.," No. xxxiv, 1912).

## Operations On Pleura and Lungs

**Intrathoracic Operations Under Positive or Negative Air-pressure.**—(This subject has been fully and judicially discussed by Jopson in "Annals of Surgery," May, 1911.) When under ordinary conditions the chest

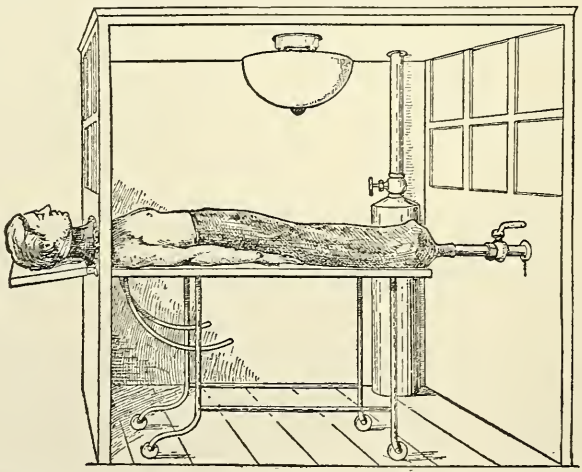


Fig. 568.—Sauerbruch's cabinet: Position of patient in chamber ready for operation under negative pressure.

wall is widely opened the lung often rapidly collapses and the patient is placed in deadly peril (see Acute Traumatic Pneumothorax on page 895). The reality of the danger has to a great extent retarded progress in the surgery of the heart, lungs, and lower portion of the esophagus. Of late, however, methods have been devised for maintaining normal respiratory movements and preventing pulmonary collapse during operations which open the pleura. There are two forms of pressure apparatus and each form finds warm advocates. Negative pressure is the form that is advocated by Sauerbruch. In Sauerbruch's negative pressure chamber the lung is kept from collapse by suction exerted upon its exposed surface. Positive pressure is advocated by Brauer. Positive pressure keeps the lung from collapsing by distending it from within. The two methods act similarly in many respects. Clinical observations and numer-

ous experiments seem to prove that "emphysema, persistent pneumothorax, difficulty of narcosis, and infection of the pleura are not dangers associated with the use of positive pressure as such" (Samuel Robinson and George Adams Leland, in "Surg., Gynecol., and Obstet.," March, 1909).

**The Sauerbruch Chamber** (Fig. 568).—This is an air-tight cabinet. The sides are of boards covered with tin, the corners being soldered. The roof is of glass. The sides contain air-tight windows. There is one air-tight door.

The room is lighted by electricity and contains a telephone. The larger chambers have a communicating room. Instruments which are wanted can be placed in this room so that the surgeon may reach them. The patient's head projects outside of the cabinet and a tightly fitting rubber collar is placed around the neck. The body and legs are surrounded by a canvas-covered rubber sac the interior of which is in communication with the external air.

The chamber is sufficiently large to hold the patient, the surgeon, and the assistant. By means of an electric suction air pump, the valve of which is in the wall, negative pressure is obtained and is continuously maintained in the cabinet. The patient's thorax is exposed to the suction of negative pressure, but the bronchioles are subjected to ordinary atmospheric pressure, hence, even when a wide opening is made in the chest wall, the lung does not collapse. The operator does not suffer from the negative pressure.

**The Positive Pressure Apparatus.**—Numerous apparatuses have been devised. Positive pressure used to be obtained by the Fell-O'Dwyer apparatus (see Fig. 567). The larynx was intubated and bellows were used. It can be obtained with this, but the apparatus is uncertain and difficult to use.

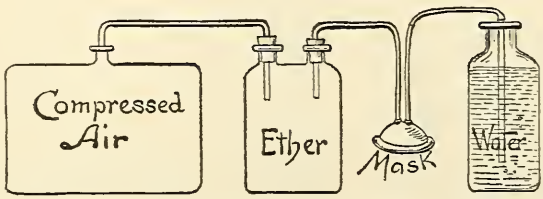



Fig. 569.—Scheme of apparatus for the maintenance of distention of the lung by positive pressure (Robinson and Leland).

Brauer advocated the following plan: When the patient has been anesthetized and the surgeon is ready to open the pleura, a glass case is placed over the patient's face and the air in the case is condensed by means of an apparatus.

Bauer subsequently modified the head chamber so that the hands and wrists of the anesthetist are admitted within it. Some surgeons have given compressed air by the nose, the mouth being sealed. Some give it by intubation from the mouth. The trouble with this method in man is that no completely satisfactory tube has yet been made. As a general thing the best way to give it is by a well-fitting face mask. A small motor runs a rotary air pump and thus we dispense with the trouble and uncertainty of clumsy reservoirs. Robinson and Leland (Ibid.) state that any positive pressure apparatus consists of four elements: (1) A supply of compressed air; (2) an anesthetizing segment; (3) a device for introducing air and ether into the respiratory tract; (4) a means of varying the resistance of exhaled air (Fig. 569).

**Intratracheal Insufflation (Method of Meltzer and Auer).**—This method is highly valuable. It can be used to maintain respiration and, at the same time, to give ether. The patient is first anesthetized by the ordinary method. A flexible and elastic tube is then carried through the larynx well into the trachea. In order to get it into the larynx use a tube director (Cotton and Boothby), or use Chevalier Jackson's direct laryngoscope, as do Elsberg and

Peck. The tube usually enters the right bronchus before it blocks. When it blocks it should be drawn back 5 or 6 cm. (Meltzer, in "Keen's Surgery," vol. vi). The tube is an English catheter or, as Elsberg prefers, a woven silk catheter. The eye should be at the end. It is always decidedly less in diameter than the trachea. Meltzer says the largest size used should not exceed 8 mm. in diameter. The tube is attached to the insufflation apparatus. This forces the air, the air and ether, or the oxygen and ether into the lungs, and the vapor returns between the tube and the tracheal wall.

Elsberg's apparatus is shown in Fig. 570. An electric current is turned on at the switch A. It is carried by wire to the motor C. This motor drives blower D. The air passes through a tube E, an oil filter F, and a tube G, into a bottle H, containing hot water. The air is then forced through the tube I to a rubber tube connected to the tracheal catheter. The tube I is also connected to the ether jar J. The tube P is joined to a foot bellows. This is a "safety device," for use if the motor or blower fails or if electricity is not available.

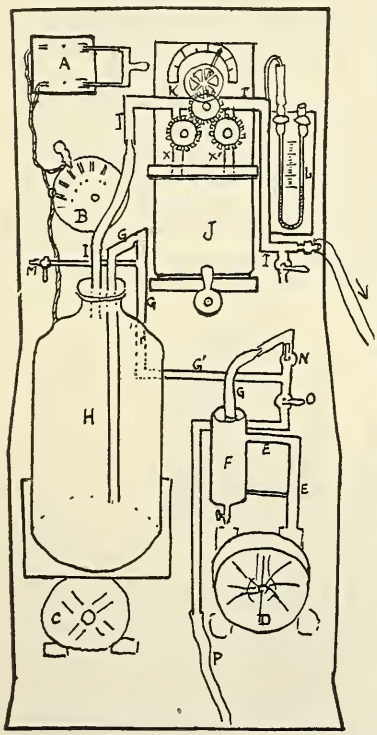


Fig. 570.—Diagram of Elsberg's apparatus showing essential parts.

**Exploratory Puncture of the Pleural Sac.**—Puncture often gives valuable information as to the existence of fluid in the pleural sac and as to the nature of the fluid. The operation must be performed with aseptic care, otherwise a serous effusion might be converted into a purulent effusion, and either a serous or a purulent effusion might be rendered putrid. A large hypodermatic syringe with a long and strong needle is used for exploratory puncture. A slender needle breaks easily and is unsafe. In order to prevent breaking the needle impress upon the patient the absolute necessity of keeping quiet and avoiding any violent respiratory or general movement during the operation. It is not desirable to stick the lung, although harm rarely results from such an accident. If no fluid is found in the pleura on one trial, several other punctures should be made. What is known as a dry tap may be due to the entire absence of fluid, to encapsulation of fluid in a region not invaded by the needle, to the lodgment of the point of the needle in thickened pleura or in an adhesion, or to blocking of the lumen of the needle with coagula. Fowler[1] points out that if a person has been recumbent for a long time the upper layer of fluid may be clear, while the lower layer is purulent. The fluid should be collected in a sterile glass tube and subjected to a careful bacteriological study.

**Paracentesis Thoracis.**—The operation of tapping with a trocar and allowing the fluid to flow out through the cannula is no longer practised except in an emergency, when an aspirator cannot be obtained, or in an early stage of non-traumatic pneumothorax. An aspirator is a much better instrument.

**Aspiration** consists in the introduction into the pleural sac of the tip of a hollow needle, the other end of which is attached by means of a rub-

[1] "Annals of Surgery," November, 1896.

ber tube to a bottle from which the air has been exhausted. The fluid does not run out, but is sucked out, air is excluded, and bacteria do not enter the pleural sac. Fig. 434 shows a pneumatic aspirator. No anesthetic is required. The patient's skin, the instruments, and the surgeon's hands must be thoroughly asepticized. The patient is given a little whisky, and, unless he is very weak, he assumes a semi-erect attitude, with the arm hanging by the side. The trocar is introduced in the fifth interspace, just in front of the angle of the scapula. The surgeon marks the upper border of the sixth rib with the index-finger, and plunges in the trocar just above the finger, thus avoiding the intercostal artery, which lies along the lower border of the rib above. He guards the needle with the index-finger to prevent its going in too far. The fluid is withdrawn rather slowly in order that the patient may escape syncope and violent cough. If the patient becomes very faint the operation should be abandoned. All the fluid present should not be removed at one sitting—complete removal of a large effusion is not safe. The operation can be repeated if necessary. After withdrawing the cannula place iodoform collodion over the opening in the chest. In an early stage of non-traumatic pneumothorax perform paracentesis without suction. In non-purulent pleuritic effusion, if the lungs will not expand after tappings, perform thoracotomy. In some cases aspiration is followed by pulmonary embolism or embolism at a distance. Syncope is a not unusual result. Convulsions occasionally occur. In rare cases the sudden withdrawal of a large effusion is followed by *albuminous expectoration*, as was pointed out by Pinault in 1853. It usually begins from a few minutes to half an hour after aspiration. When this complication arises the pulse is very weak, there are severe dyspnea, cyanosis, cough, and the expectoration of quantities of a yellow, frothy fluid. Riesman ("Amer. Jour. Med. Sciences," April, 1902) demonstrates that the condition is due to pulmonary edema and not to puncture of the lung. The sudden withdrawal of fluid by aspiration relieves the pressure which was compressing the lung, the lung becomes congested with blood (*congestion by recoil*, Riesman calls it), the blood distends weakened vessels, and profuse transudation takes place into the air-cells. Most cases recover in a few hours or a day or two. Severe cases die from asphyxia. Terrilon collected 23 cases, with 2 deaths. If albuminous expectoration arises, dry cup the chest and counterirritate with mustard plasters. Perform venesection. Give oxygen by inhalation. Administer atropin hypodermatically. Employ artificial respiration if necessary.

**The Operation for Creating Artificial Pneumothorax.**—Murphy's apparatus as modified by Brauer is shown in Fig. 571. The manometer is on the left-hand side of the figure. The jar for nitrogen (*A*) contains a solution of corrosive sublimate through which the gas flows. The water jar (*B*) is lowered and the nitrogen tube is opened, and as the stream of nitrogen passes along the glass tube it goes through a filter of sterile cotton. The tube from the jar of nitrogen joins a three-way stopcock (*C*). The nitrogen may be made to flow through the needle (*H*), or deliver the intrapleural pressure to a water manometer and a mercury manometer. After determining that the functional capacity of the other lung is sufficient to sustain the demands about to be put upon it, proceed with the operation. Follow Brauer's plan because it is the safest. Select a spot where the healthy sounds and resonance are best heard (Mary E. Lapham, "Amer. Jour. Med. Sciences," April, 1912). Expose the pleura by an incision. If adhesions are absent, puncture by a blunt instrument and explore by a catheter to be sure there is a pleural cavity. If a cavity exists attach the needle to the apparatus and inject the nitrogen. After injection suture the wound. Subsequent injections are made by the needle alone, no incision being required.

**Thoracotomy** is an incision into the cavity of the pleura. It may be merely an intercostal incision, or may be an opening into the chest after resecting a portion of a rib. Often in a child with empyema good drainage can be obtained by an intercostal incision, but in most children and in all adults a rib should be resected.

If there is very little dyspnea, ether may be given. If there is considerable dyspnea, chloroform should be given. If there is severe dyspnea, no general

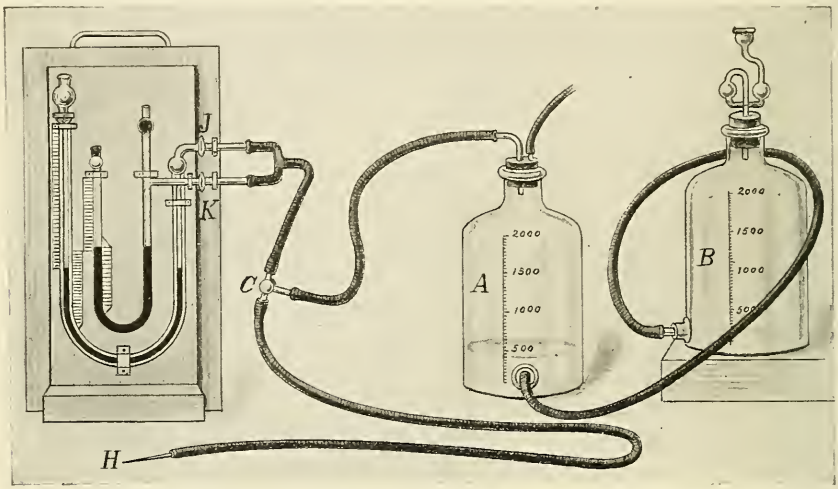



Fig. 571.—Brauer and Spengler's modification of Murphy's apparatus for nitrogen injections.

anesthetic is admissible. In severe dyspnea the patient is using certain voluntary muscles to aid him in obtaining air. A general anesthetic abolishes the activity of the voluntary muscles of respiration, and so might cause suffocation. In such cases the operation can be done with fair satisfaction after the injection of eucain or after infiltrating the superficial tissues of the chest wall with Schleich's fluid, or, what is better, preliminary aspiration can be performed. Aspiration will permit of the subsequent administration of a general anesthetic. The patient on whom thoracotomy is to be performed is placed supine, the diseased side being at or over the edge of the table. He must never be placed on the sound side, because he breathes only with that side, and pressure on it may be dangerous.

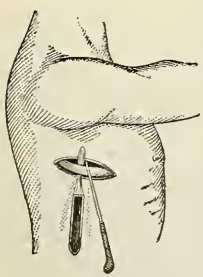


Fig. 572.—Resection of a rib (Esmarch and Kowalzig).

The arm of the diseased side should be elevated to a right angle with the body. If the surgeon desires to obtain only intercostal drainage, he should make a longitudinal incision about 3 inches in length at the upper border of the sixth or seventh rib, and the middle of this incision should correspond to the midaxillary line. This incision is carried, layer by layer, to the pleura. If, as will usually be the case, he wishes to remove a portion of a rib, he will make an incision about 3 inches in length directly upon the outer surface of the rib he wishes to remove, and the middle of this incision corresponds to the midaxillary line. Some surgeons resect a portion of the fifth rib, some remove a bit of the eighth rib, and Munro[1] shows that at the level of the eighth rib there is no danger of

[1] "Medical News," Sept. 2, 1899.

injuring the diaphragm. By many operators a portion of the seventh or eighth rib is removed in front of the line of the posterior axillary fold.

I agree with Hutton that a portion of the sixth rib in the midaxillary line should be removed.[1] The reasons given by Hutton for the selection of this rib are: (1) It is over the portion of the lung which expands last. An empyema is drained only partly by gravity, and most of the fluid is really forced out and the cavity is obliterated by lung expansion. If an incision is made anterior or posterior to this point the expanding lung will block the drainage opening, and a pus-cavity without drainage will remain in the midaxillary line. (2) Such an incision permits a patient to lie on his back without making pressure on the drainage-tube.

The periosteum of the outer surface of the rib must be divided in the same direction as the superficial incision. The exposed rib is stripped of periosteum front and back by means of a periosteal separator, and with the periosteum at the lower border of the rib the intercostal artery is lifted out of harm's way. The rib can be divided by means of cutting forceps or a Gigli saw. The usual method is to push a periosteal separator under the rib and saw the bone in two places by means of a metacarpal saw (Fig. 572). I prefer a costotome, as it accomplishes the section most rapidly. An inch or more of the rib should be removed. The intercostal artery is ligated at each end of the incision, the periosteum is removed, and the pleura is opened. The object of removing the periosteum is to prevent the rapid formation of bone which might narrow the opening and interfere with drainage. The actual opening of the pleura is carried out in the same way in intercostal incision and after rib resection. A grooved director is pushed into the pleural sac, and the opening is enlarged by means of the forceps and the finger.

The finger removes all masses of tuberculous material or aplastic lymph within reach. If the finger finds the lung firmly bound down by dense adhesions so that it cannot expand, simple rib-resection will not cure the patient. If the adhesions between the parietal and visceral pleura can be separated by the finger the lung may expand. In order to accomplish this separation a piece of more than one rib must be removed, because it is necessary to insert more than two fingers (Samuel Lloyd, quoted by Lund, in "Jour. Am. Med. Assoc.," August 26, 1911). If adhesions cannot be separated so that the lung can expand, Estlander's, Schede's, or Fowler's operation should be done. Some surgeons advocate immediate irrigation after opening an acute empyema, but this procedure is unsafe. It is true that in most cases irrigation does no harm, but in no case will it sterilize the cavity, and in some cases it is very dangerous. The pleura is very susceptible to the action of irritants. This is especially true of young children. It happens occasionally that the injection of the blandest fluid is followed by intense dyspnea, great shock, disturbances of respiration and circulation, convulsions, and even death (Quénu). The convulsions which occasionally follow pleural irrigation were called by de Cerenville *pleural epilepsy*. In putrid empyema it is proper to irrigate. Irrigation will remove part of the actively poisonous putrid matter, and the retention of putrid matter is a greater danger than irrigation. It was formerly a common custom to make a counteropening by cutting down upon the long probe pushed against the chest wall after being introduced through the incision, but a counteropening is of no particular use. A drainage-tube about 2 inches in length is introduced and stitched in place. The tube must not be long enough to touch the lung. A safety-pin is clamped upon the tube to keep it from slipping into the chest. A tape should be fastened to each side of the tube and tied about the chest to prevent it from slipping out. Arrest bleeding, suture the skin, dress with gauze and

[1] See W. Menzies Hutton on "Empyema," in "Brit. Med. Jour.," Oct, 29, 1898.

a binder, and have the dressings changed as soon as they become soaked at one point. Several times a day change the patient's position. At each change of dressings direct him to lie on the diseased side with the foot of the bed raised for half an hour. Healing takes place by ascent of the diaphragm, expansion of the lung, and retraction of the chest wall. Expansion of the lung is favored by expiratory acts; hence cause the patient several times a day to blow through a rubber tube into a 1-gallon Wolff bottle filled with water. The water is blown into another bottle attached to the first by a tube. Remove the drainage-tube when the discharge becomes thin and scanty (about the eighth or tenth day, as a rule). If an empyema ceases to improve and remains stationary for months after it has been drained, firm adhesions exist. If after one year has passed a cavity still exists and there is a flow of pus, the surgeon must perform the operation of Schede, Estlander, Fowler, or Ransohoff.

**Thoracoplasty** (**Estlander's operation**) was first proposed by Warren Stone, an American surgeon, but was set forth in detail by Estlander, of Helsingfors, in 1879. It is employed in old cases of empyema in which drainage has failed and in cases with retracted chest wall, collapsed lung, thickened pleura, and cavities whose rigid walls will not collapse. The procedure recognizes the fact that after pus is evacuated, if the lung is adherent, it cannot expand to fill the space once occupied by fluid, and that the rigid chest wall cannot fall in as a substitute for the lung. It seeks to destroy the rigidity of the chest wall and to permit it to collapse and thus obliterate the cavity of the empyema. In this operation a piece is removed from every rib which overlies the cavity. When the surgeon resects a rib and finds a cavity with uncollapsible walls, or a lung bound down with firm adhesions, he should perform thoracoplasty. This operation causes the obliteration of the cavity by collapsing that portion of the chest wall overlying it. The cavity is usually in the upper or central part of the pleural space. The instruments required are the same as those for resection of a rib. The position is the same as that for rib resection. The length of the incision depends on the size of the cavity. The surgeon usually removes portions of the second, third, fourth, fifth, sixth, and seventh ribs. Make a transverse incision along the center of an intercostal space, and through this incision remove the ribs above and below by the method set forth on page 908 (the removal of six ribs will require three incisions). Instead of this incision, we can make a vertical incision or a U-shaped flap. Always take away the periosteum in order to prevent reproduction of the ribs. In cavities which are surrounded by firm adhesions, and in old cases in which the pleura is greatly thickened, irrigation is safe. If the cavity is small, it should be packed with iodoform gauze and allowed to granulate; if large, it should be drained by a large tube, the skin being sutured by silkworm-gut.

**Schede's Operation.**—Schede, of Hamburg, showed that when the pleura is much thickened, even Estlander's operation will not permit the chest wall to collapse and fill the cavity once occupied by the fluid. The instruments used are the same as for Estlander's operation. A U-shaped flap is made from the level of the axilla in front to the level of the second rib and between the scapula and spine behind. The lowest level of this incision corresponds to the lowest limit of the pleura (Fig. 573). The flap is loosened and raised and the scapula is lifted with it. The ribs from the second rib down and from the costal cartilages to the tubercles are removed, along with the intercostal muscles and the pleura. This is accomplished by cutting with bone-shears and scissors. Hemorrhage is arrested. The pleura is curetted. A drainage-tube or a piece of iodoform gauze is introduced, and the raw flap is laid against the visceral layer of the pleura. The superficial incision is

sutured, except at the point where the tube or the gauze emerges. The average mortality from Schede's operation is from 15 to 20 per cent. The operation is far more often necessary in adults, but the results are much better in children.

**Total Pleurectomy or Pulmonary Decortication (Fowler's Operation).**—In the spring of 1893 de Lorme, of the Val de Grace, performed some experiments on dogs looking to the development of the operation. In October, 1893, the late George Ryerson Fowler, of Brooklyn, having no knowledge of de Lorme's investigation, operated on a man and cured a chronic empyema. The French surgeon's first operation was months later. De Lorme sought to do without the great mutilation of the Schede operation. His idea was to make an opening in the chest wall large enough to work through (but not nearly so large as that caused by Schede's operation), incise the dense fibrous membrane which binds down the lung, and allow the lung to expand and fill the cavity. De Lorme makes a trap-door incision. Fowler resected ribs extensively to obtain room. The thickened fibrous membrane is removed from the chest wall, lung, pericardium, and diaphragm, any sinus is extirpated, and all granulation tissue is taken away. The shrunken lung expands to fill the cavity.

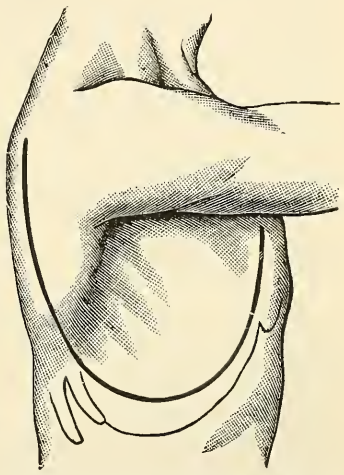


Fig. 573.—Incision for Schede's operation of thoracoplasty (Esmarch and Kowalzig).

Lund describes the operation as follows ("Jour. Am. Med. Assoc.," August 26, 1911): "In regard to the technic of the operation, the method of the resection of $1\frac{1}{2}$ inches of five or six ribs, through an incision running upward and forward from the anterior end of the old drainage incision, has proved, to my mind, very satisfactory. In slitting up the thickened pleura beneath the ribs I have had, in one or two cases, to grab the intercostal artery, but have been very much surprised to find how little trouble there has been from bleeding. The visceral pleura, which is about $\frac{1}{4}$ inch thick and which is more like the sole of an old rubber shoe than anything else, is carefully incised with a knife over the lower part of the lung. The finger is inserted through the incision and, as soon as the soft surface of the lung is felt, is swept to an fro with the pulp of the finger toward the pleura and pressing outward so as to cause the least possible damage to the lung. Then a pair of blunt-pointed scissors is inserted and the membrane slit clear up to the top of the chest, and cleared off from the lung with the finger. A wound of one or two of the air-cells resulted in 2 or 3 of my cases, allowing the escape of bubbles of air on exploration, but so small an area of the lung was affected that, apparently, no harm resulted. In regard to the after-treatment, it is probable that after the patient is put to bed the lung does retract to a certain extent, but the occasional coughing keeps up the expansion. The thick, pus-soaked dressing, if tightly applied, to my mind, acts as a more efficient valve than any mechanical valve which one could employ."

Fowler made a report of 30 cases. Eleven cases were completely cured. In 17 cases the empyema was cured, but 6 of them had tuberculosis. There were 3 deaths. The combined statistics of Fowler, de Lorme, and Cestan show 35.7 per cent. cured, 19.7 per cent. improved, 33.9 per cent. not cured, and 10 per cent. died (Kurpjweit, in "Beiträge für klinischen Chirurgie," Bd. xxxiii, H. 3).

**Discission of the Pulmonary Pleura (Ransohoff's Operation).**—This operation was devised by Ransohoff, of Cincinnati. It can be employed when decortication is impossible, and it may be used as a substitute for decortication in certain cases. It permits the shrunken lung to expand. It is founded on the observation that if the thickened pleura over a shrunken lung is incised the cut widens with each respiration and quickly becomes a groove (Ransohoff, in "Annals of Surgery," April, 1906). The pulmonary pleura is divided by numerous parallel incisions $\frac{1}{4}$ inch apart, and then similar incisions are made to cross these. An incision is also carried through the costal side of the angle of reflection of the pulmonary and costal pleura.

**Pneumotomy for Abscess and Gangrene of the Lung.**—Pneumotomy is employed for abscess, gangrene, and bronchiectasis. Give chloroform or use a local anesthetic. Place the patient recumbent with the shoulders a little raised. Make a U-shaped flap over the seat of disease. Resect a portion of a rib. If it is found that adhesions do not exist between the pulmonary and costal layers of the pleura, stitch these layers together with catgut, and either postpone further operation for forty-eight hours or surround the area by gauze and operate at once. If adhesions exist, proceed at once. Chloroform can be put aside when the pleura is exposed. Fowler called attention to the fact that lung tissue is so insensitive that the administration of an anesthetic can be suspended as soon as the pleura has been opened. Incise the agglutinated layers of the pleura, and pass an aspirating needle into the lung in various directions. When the abscess is located, open it by the cautery. Carry the Paquelin cautery slowly into the lung in the direction of the abscess-cavity. The cautery knife should be at a dull-red heat.

When the cautery opens the cavity of the abscess, withdraw the instrument and insert a drainage-tube, and suture the flap of superficial tissue. If the abscess is not found after one or two punctures by the aspirating needle, abandon the attempt.

Tuffier explores for an abscess by what he calls *décollement of the parietal pleura*. He exposes the parietal layer of the pleura, passes his hand between this layer and the chest wall, strips the pleura off over a considerable area, and is able to feel the lung beneath and thus determine its condition.

Hartmann ("Presse Médicale," April 27, 1912) states that Garré collected 96 cases of abscess, with 19 deaths, and 122 cases of gangrene, with 42 deaths. In bronchiectasis pneumotomy may be employed. Hartmann (Ibid.) combines the cases of Körte and Sauerbruch, and states that in 149 cases there were 46 cures. In Sauerbruch's 133 cases there were 40 cures, 43 deaths, and 7 were improved.

## XXVII. DISEASES AND INJURIES OF THE UPPER DIGESTIVE TRACT

**Injuries and Diseases of the Face, Nose, Mouth, Salivary Glands, Tongue, Jaws, and Esophagus.—Closure of the Jaws.**—This condition may be caused by tetanus, by the irritation of a non-erupted wisdom tooth, carious teeth, by cancer of the mouth, sarcoma of the jaws, alveolar abscess, cicatricial contractions due to burns or noma, and temporomaxillary ankylosis.

**Temporomaxillary Ankylosis.**—Ankylosis of the temporomaxillary joint may result from gonorrheal arthritis, rheumatoid arthritis, or fracture of the condyle. Even when one joint is completely ankylosed, the jaws upon the sound side can be somewhat separated because of the elasticity of the mandible.

*Treatment.*—Gradual dilatation by means of box-wood screws is useless. Violent separation is without value. After either method the condition always

recurs rapidly. Esmarch removed a wedge-shaped piece of bone from the angle of the jaw in order to form a false joint. Other operators do a like operation on the ramus. A simple osteotomy is certain to fail because bony union will occur. That is the trouble with Swain's operation (sawing the body at the angle). Bony union may be prevented by resecting the zygoma and putting a flap of temporal muscle between the fragments (Helferich). Removal of the condyle and a portion of the neck is usually efficient. In double ankylosis both condyles should be resected. Verneuil, in 1860, suggested mobilizing the joint and interposing an attached flap of temporal muscle between the condyle and the socket. Such an operation may give a gratifying result.

**Alveolar Abscess.**—This condition is caused by a decayed tooth. A superficial abscess is known as a *gum-boil*. The process may spread to the jaw-bone, causing necrosis. From the maxilla the suppuration may enter the antrum. From the lower jaw pus may track into the neck.

*Treatment.*—Early and free incision, usually, but not always, the extraction of the offending tooth, and drainage.

**Necrosis of the Jaw.**—Extensive necrosis is much more common in the lower than in the upper jaw. Necrosis of the alveolar process of either jaw may be due to suppurative periostitis, the result of carious teeth. In suppurative periostitis of this form an alveolar abscess arises which is followed perhaps by circumscribed necrosis. In rare cases widespread suppurative periostitis occurs which may result in extensive necrosis.

Syphilis produces periostitis and osteomyelitis, but more commonly causes caries than necrosis. Tuberculous periostitis may result in limited necrosis. Its most common site is the orbital margin of the maxilla.

Actinomycosis is a rare cause of necrosis. In mercurial salivation extensive necrosis is prone to occur. A child suffering during dentition from an exanthematous fever or other virulent infection is liable to a violently acute suppurative periostitis and osteomyelitis with evidence of severe general infection. In the lower jaw particularly extensive and usually symmetrical necrosis follows.

*Treatment.*—In an acute suppurative periostitis remove a carious tooth (if one exists), incise the gum to the bone; if osteomyelitis exists open the outer plate of the bone, order frequent cleansing of the mouth by antiseptic washes. In syphilitic caries place the patient on antisyphilitic treatment and use a curet to remove carious bone. In tuberculous caries use a curet and employ antituberculous treatment.

When necrosis occurs it is usual to wait until the sequestrum loosens and to then remove it through the mouth or nose. Early sequestrectomy is a better plan. If there is profuse suppuration, remove the dead bone by subperiosteal resection (Tillmanns's "Text-Book of Surgery").

**Phosphorus Necrosis.**—This condition was first described in 1845 by Lorinser. It is seen among those who make matches from yellow phosphorus. Recent legislation against yellow phosphorus is wiping out the disease. It is most common in the lower jaw. The fumes enter carious teeth. About such teeth ossifying periostitis or suppurative periostitis and osteomyelitis occur. Even when the process begins as ossifying periostitis suppuration occurs, either between the periosteum and the new bone or between the new bone and the older bone. The bone enlarges enormously and very extensive necrosis takes place. The entire lower jaw may be lost. Bones of the base of the skull may be destroyed. Tillmanns (quoting Maas, Binz, and others) says that arsenic and pyrogallic acid tend, like phosphorus, to produce ossifying periostitis.

*Treatment.*—Entire removal of the victim from the fumes, living in the open air, the frequent use of antiseptic mouth-washes, incisions if pus forms, early subperiosteal sequestrectomy by means of a chisel and mallet.

**Wounds of the Salivary Glands.**—An aseptic wound usually heals and rarely results in a salivary fistula, although after healing it is not unusual for an encysted collection of saliva to gather under the skin. Such a collection of saliva, if it does not disappear spontaneously, can usually be gotten rid of by continued pressure. When a wound of a salivary gland is infected a single fistula or multiple fistulæ may be left as a legacy. A salivary fistula is very annoying, because the saliva flows constantly. A fistula of a gland usually heals spontaneously after a long time, but healing may be quickly brought about by touching the orifice with the Paquelin cautery.

*Wound of Steno's duct is apt to cause a fistula*, and the condition is often difficult to cure. In this condition, when the duct was cut across, the central end grows fast to the cutaneous surface. Fistula of Steno's duct may also be caused by obstruction and rupture of the duct and by suppurative or gangrenous processes.

In wounds of the duct the ends should be brought as near together as possible by catgut sutures which do not enter the lumen of the duct; if the mucous membrane is not already opened an incision should be made through it to permit drainage of saliva into the mouth, and the skin should be sutured. In some cases the central end of the duct may be carried into the mouth and sutured to the mucous membrane. If, after an injury of Steno's duct, saliva gathers under the skin, make an incision through the mucous membrane to give a route for the saliva to enter the mouth, and apply pressure externally. When an external fistula forms, it may perhaps be cured by the cautery and pressure, but, if the peripheral portion of the duct is obliterated (which can be determined by a sound) a cutting operation must be performed. Tillmanns advocates cutting out the external portion of the fistula by two elliptical incisions. A trocar is passed through the bottom of the wound in two places, about $\frac{1}{2}$ cm. apart; a piece of stout silk is drawn through the holes and tied tightly and the superficial incision is closed. The silk cuts through and makes an internal fistula. Another method is to make an incision, find and isolate the central end of the duct, open the mucous membrane, suture the duct to it, and close the superficial wound.

Fig. 574.—De Guise's operation for salivary fistula (Esmarch and Kowalzig).

De Guise's operation is shown in Fig. 574. He threads a piece of silk through two needles and carries the needles into the mouth so that the silk will embrace a bit of tissue $\frac{1}{2}$ cm. in length. The silk is tied tightly within the mouth, the ends are cut off, and the margins of the fistula at the surface are freshened and sutured. I prefer silver wire to silk.

**Parotitis.**—Mumps, or epidemic parotitis, is treated by the physician. In this condition the submaxillary and sublingual glands are usually involved as well as the parotid. In pyemia, metastatic abscesses may form in the parotid gland. Great swelling arises, respiration is often embarrassed, and early incision is necessary. Parotid inflammation other than mumps is usually due to the passage of bacteria up Steno's duct, the source of the microbes being a foul condition of the mouth, particularly noma or stomatitis. Hence such inflammation is most common during the existence of acute infectious diseases and sepsis. Suppuration or even gangrene may occur. As a rule, only one

gland is attacked, but both may be. It is a well-known fact that occasionally, though very rarely, after an abdominal operation inflammation of the parotid gland occurs. The condition is more common in adults than in children. This form of parotitis may, of course, be due to septic metastatis and may be produced by trauma, but I am satisfied that most cases result from foul mouths, the infection ascending from the mouth along the duct. Oral cleanliness tends strongly to prevent the so-called *sympathetic parotitis*. In about one-third of the cases the condition is not to be distinguished from mumps and is recovered from in seven to eight days. Mild cases seldom suppurate, and if they do, the pus may flow down the duct into the mouth. In nearly one-half of the cases, according to Marchetti ("Epitome of Surgery," in "Brit. Med. Jour.," March 6, 1909), there is phlegmonous inflammation with necrosis and suppuration of the tissues and formation of a salivary fistula. In non-suppurative parotitis there are pain, tenderness, obvious swelling, and hyperemia of the skin, and it is difficult to open the mouth or swallow. When suppuration occurs, all of the above symptoms are intensified, the discoloration becomes dusky, the skin becomes shiny and edematous, the constitutional symptoms of pus formation exist, and there is usually delirium.

*Treatment.*—In the non-suppurative form apply heat. Wash the mouth out frequently with an antiseptic wash and apply ichthyol and lanolin to the swollen region. In the suppurative form make several openings by Hilton's method, seeking for points of softening; apply hot antiseptic fomentations, wash the mouth frequently with an antiseptic fluid, and combat sepsis by appropriate constitutional treatment.

**Salivary Concretions.**—The saliva contains in solution certain salts which may be deposited. Deposited on the teeth they constitute tartar. Deposited in a salivary duct or the acini of a gland they constitute a calculus. The salts deposited are carbonate and phosphate of lime. A calculus may consist purely of these two salts or there may be a foreign-body nucleus. A calculus is a possible result of an inflammation which blocks, constricts, or roughens a duct or acinus and decomposes saliva. Small concretions are often passed. Concretions the size of a bean are retained. A concretion may attain the size of an English walnut. A concretion does not block a duct continuously, but does so now and then, causing swelling and tenderness of the gland. A retained calculus can be palpated by a finger in the mouth and a finger externally.

*Treatment.*—A calculus in a duct is extracted by making an incision through the mucous membrane. If a very large calculus forms in the submaxillary gland, the gland should be removed through an external incision.

**Lymphomata of the Salivary and Lachrymal Glands (Mikulicz's Disease).**—Mikulicz, of Breslau, described this condition in 1888. It is a chronic, brawny, and non-inflammatory swelling, painless though sometimes tender, and unconnected with any known systemic condition. In Mikulicz's early cases the lachrymal, parotid, and submaxillary salivary glands of both sides were enlarged, hence he regarded it as symmetrical. We now know that non-symmetrical cases occur, in fact, only one gland may be enlarged, although, of course, such cases may eventually develop growths on the other side. In some cases the sublinguals have been involved, in some the accessory lachrymals, in some the glands of Nuhn and Blandin. In a case of Osler's there was enlargement of the spleen, tonsils, and cervical lymph-glands. In a typical case the cheeks are much broader than natural, and the eyelids droop on the temporal side like "those of a bloodhound" (Ziegler, in "New York Med. Jour.," Dec. 11, 1909). The mouth is very dry because of deficiency of salivary secretion. The conjunctiva is dry for want of enough tears. Chronic inflammation in the nasopharynx is not unusual.

The condition may occur at practically any age after three or four. A case four years of age has been reported. The glands may undergo regression during pneumonia, appendicitis, or some other infection. The cause is doubtful. Some think it is due to bacteria, but the tissue is not inflammatory, being merely hyperplastic lymph tissue. Others regard it as due to a toxic material in the blood. Ziegler believes that the causal toxic material comes from the nasal sinuses.

*Treatment.*—Arsenic internally will perhaps produce cure. The iodids are used by some and pilocarpin has been recommended. The *x*-rays should be applied. Operative removal has not been successful. All diseased conditions of the nasopharynx should be corrected, if possible. (See Ziegler's thorough study, in "New York Med. Jour.," Dec. 11, 1909.)

**Harelip and Cleft-palate.**—*Harelip* is a congenital cleft in the upper lip due to defective development. *Cleft-palate* is a congenital fissure in the soft palate or in both the hard and soft palates. In harelip the cleft is usually complete, through the entire lip into the nostril, but in rare cases it may show only as a furrow in the mucous edge or as a split from the nostril partly into the lip. It is most common on the left side. In double harelip the central portion of the lip is often adherent to the tip of the nose. Double harelip may be free from complication, but is often associated with a malformation of the alveolus and palate. The term "harelip" is a poor one, as the cleft in a hare's lip is the shape of the letter Y, the stem of the Y being median and an arm entering each nostril. Median harelip is exceedingly rare. Dupuytren said it never occurred, but at least 9 cases have been reported (Ransohoff, in "Lancet Clinic," Nov. 2, 1912). Ordinary or lateral harelip is due to failure of fusion of the lateral maxillary and frontal processes. Median harelip is due to "failure of union between the lateral tubercles of the frontonasal process which is placed on each side of the middle line" (Ransohoff, Ibid.). We recall His's teaching, that the central portion of the upper lip is formed from the lower portion of the frontonasal process by the fusion of its two buds (Ransohoff, Ibid.). In cleft-palate the septum of the nose is usually adherent to the palatine process opposite the side upon which the fissure exists. In those rare cases of cleft-palate double in front, the nasal septum is attached only to the premaxillary bone, and the premaxillary bone is not attached at all to the superior maxillary bone. In harelip there is frequently a cleft in the alveolus, and almost always flattening of the corresponding side of the nose. Harelip is often associated with cleft-palate, talipes, and other deformities. It is a great deformity, and interferes with sucking, swallowing, and articulation.

Operation for harelip uncomplicated by cleft-palate should be performed between the third and sixth months of life in a child in good health, free from stomach trouble, cough, or coryza, but operation is not advisable in the early weeks of life. Always, if possible, operate before dentition begins (seventh month). If the child is in poor health, postpone the operation until restoration has so far advanced as to render operation safe. While waiting for operation be sure the child is getting enough food. If it cannot suck, feed it with a spoon. If a cleft exists in the palate, we sometimes operate first upon the lip, because the pressure of the parts after the edges of the gap are approximated aids in the closure of the bony cleft. In other cases we operate first on the palate. Cleft-palate interferes with sucking, deglutition, mastication, and articulation. In severe cases the food passes into the nose and excites inflammation. Loss of control of the palate muscles always exists, and liquids and solids are liable to pass into the wind-pipe. Clefts in the hard palate should not be operated on until the second year, but should be operated upon then, otherwise speech will be permanently affected. Some surgeons refuse to operare until the tenth or twelfth year, but operation done this late will not correct

speech defect. The patient at the period of operation should be well and free from cough. In many cases the passage of food and drink into the nose can largely be prevented by the use of a diaphragm.

*Operation for Harelip.*—Wrap the child in a sheet; place it in the Trendelenburg position, and rest the head upon a sand-pillow. The surgeon stands to the right side of the patient. Ether or chloroform is given. For *single harelip*, separate with the scissors the upper lip from the bone on each side of the cleft until approximation of the edges can be effected without tension. If the premaxillary bone of one side projects more than its fellow, grasp it with sequestrum forceps and bend it back (Jacobson and Treves). Clamp the upper lip at each angle of the mouth to prevent hemorrhage. If the edges are of equal or nearly equal length, and if the gap is not very wide, perform Malgaigne's operation. This is performed as follows: A flap is detached on each side, the detachment beginning at the upper angle of the gap; each flap is detached above, but remains attached below. The flaps are separated from the bone, and are drawn downward so as to form a prominence at the vermilion border (Fig. 575). If the edges are pared so that in closure the vermilion border is even, when the parts are healed a gutter will be visible at the line of union. The edges are approximated by an assistant, and silkworm-gut sutures or silver wires are passed by means of a straight needle. Each suture goes down to the mucous membrane. The first suture is passed through the middle of the lip, $\frac{1}{3}$ inch from the cleft. Three or four

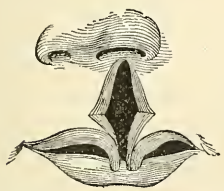


Fig. 575.—Malgaigne's operation for harelip.

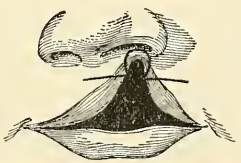


Fig. 576.—Mirault's operation for single harelip (Esmarch).

Fig. 577.—Incisions for double harelip (Esmarch and Kowalzig).

main sutures are passed through the thickness of the lip, and are tied and cut off. Two or three fine silk or catgut sutures are passed by a curved needle through the vermilion border of the lip and the mucous membrane of the mouth, and are tied and cut off. A small piece of gauze is placed over the lip and is held in place by straps of rubber plaster. After operation prevent the child crying by feeding it often and giving it small doses of laudanum. Heath orders 2 drops of laudanum in 1 oz. of distilled water, a teaspoonful to be given every two or three hours. About the sixth day one-half the sutures are taken out, and on the eighth or ninth day the remaining ones are removed. In many cases no further procedure is necessary, but if after some weeks the prominence at the lip border does not shrink, it can be readily clipped away. Harelip-pins are not used at the present time, and are not needed if the lip is well separated from the bone. If the edges of the cleft are of unequal length, Edmund Owen's operation can be performed (see below, under Double Harelip), or we can perform Mirault's operation, as shown in Fig. 576.

In *double harelip* the operation is similar to that for single harelip. If the intervening piece is vertical and is covered with healthy skin, complete each operation as for single harelip, closing both fissures at once with silver wire in a strong, healthy child, closing them at intervals of three weeks in one not so lusty (Fig. 577). Excise the septum if it is deformed. The premaxillary bone should in most instances be removed, the skin over it being preserved. Sir William Fergusson was accustomed to incise the mucous membrane and shell out

this bone. The premaxillary bone can be forced back into line, being held, if necessary, by catgut suture of the periosteum; but if saved, it is liable to necrose and its teeth soon decay. Heath removes this bone two weeks before operating on the lip. If there is much hemorrhage after removal of the bone, arrest it with a hot wire or with Horsley's wax. Figure 577 shows incisions for double harelip. Edmund Owen's operation is very useful (Figs. 578, 579). In this operation very thick flaps are cut. The prolabium and incisive bone are removed. The flaps are cut as shown in Fig. 578, on one side by a line *a–b*, and on the other side the piece *c–d–e* is removed; *a* is brought to *e*, *b* is brought to *d*, *f* is brought to *c*, and sutures are applied (Fig. 579).

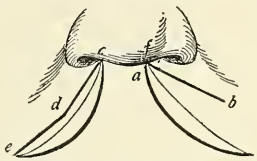



Fig. 578.—Double harelip, the prolabium and incisive bone having been removed (Owen).

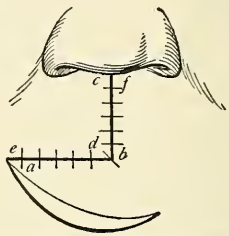



Fig. 579.—The two sides of the lip drawn together and secured by sutures (Owen).

*Operation for Cleft-palate.*—It is true that during the early years of its growth a cleft diminishes in size, and particularly if a harelip is closed, but to wait too long before we operate means permanent speech impairment. Bony clefts should be operated upon early in the second year. Clefts of the soft palate only may be operated upon during the first six months of life. If both the hard and soft palates are cleft, close both at one operation. In an ill-nourished child in which the covering of the bone is obviously thin it is best to postpone any operation upon a bony cleft until the end of the third year. I agree with Berry that operation is justifiable up to the age of twenty, but early operation is highly desirable. Edmund Owen put forth a convincing plea for early operation.[1] He says he is operating earlier and earlier, and quotes Chilton as the gentleman who led him to do so. Owen maintains that if speech is to be improved, operation must be done early, and he formulates some very valuable rules for preparation and care. I have never been convinced that operation in early infancy is sufficiently safe or has any notable advantages. When one comes to treat congenital clefts of the lip, the alveolar process, and the hard and soft palate, the necessities one should seek to obtain are the surgical closure of the clefts, the establishment of the function of the involved tissues, the correction of the congenital deformity, and the prevention of postoperative or acquired deformity. There are few if any cases of cleft-palate that cannot be successfully treated by surgical means; and it is a very unusual thing for a case really to need any mechanical appliance, such as the obturator and velum.

In deciding upon the time for operating and the nature of the operation, the safety of the patient should be the first consideration. One must carefully consider the physical condition, especially in respect to nutrition. An operative method that has a greater mortality than is incident to minor surgery ought not to be selected, and no operation should be performed until the condition of the patient justifies it. Having considered the physical condition of the patient and the relative safety of different operative plans, a careful study of the individual case should be made, and in this study each of the four requirements above set forth must be attentively regarded. If we succeed in closing the cleft without establishing the function of the tissues, without correcting congenital deformity and without preventing postoperative

[1] "Lancet," Jan. 4, 1896.

or acquired deformity, we leave the patient worse off than he was before, and perhaps render subsequent satisfactory treatment impossible.

We should attempt to secure closure of the cleft with the least possible formation of cicatricial tissue. The simplest technic is the best, and we should endeavor to avoid all unnecessary additional traumatism. One should refrain from passing additional approximation sutures, from bruising the tissues by overtension or by traction-forceps, and from using large needles and coarse suture materials, which make large suture-cicatrices. The amount of scar tissue bears directly upon the functional result. In addition, when dealing with the lip, and especially with the soft palate, one must seek to avoid incisions that involve muscles, and particularly the nerve-supply of muscles.

The periosteal-flap operation separates portions of the soft palate from the palatine bones. A large amount of cicatricial tissue is necessary to effect repair, and this mass of new tissue lessens the good functional results. In the periosteal-flap operation the repaired soft palate is anterior and inferior to the position secured by the osteoplastic method and, to that extent, interferes with the closure of the nasopharynx. Nevertheless, in my opinion, the operation which uses the soft tissues only is by far the safest and is the one I usually employ. In cases of complete cleft associated congenital deformities are especially manifest in the nose, lips, premaxilla, and maxillæ.

To correct congenital deformities and to prevent postoperative or acquired deformities is the most neglected and the least understood phase of the subject, and it is a very complicated question to hope to make clear in a brief statement. The key to the difficulty is the normal contour of the face as established by the proper occlusion of the permanent teeth. In finding this out there is no better guide than the rules laid down by Dr. Angle in the latest edition of his "Orthodontia." He maintains that every tooth must be held in its proper relation and occlusion; and that if any teeth are lost they must be replaced in order to establish or restore the proper expression and contour of the face. To comply with the foregoing requirements one should avoid any operation that would not maintain or would fail to replace the normal position of the premaxilla and the maxillæ and their future complement of teeth. If the premaxilla is only slightly in advance of its normal position, the early closure of the cleft of the lip will help to replace it. If, however, the premaxilla is far in advance of its normal position, it is hopeless to expect the pressure of a reunited lip to restore it to position. In such a case sufficient of the nasal septum posterior to the premaxilla must be resected, and the premaxilla must be carried back and sutured in position; but this operation should be done after the closure of the cleft soft and hard palates, and seldom at the same operation. If the cleft is unilateral in relation to the premaxilla and that bone is swung to the opposite side, and anterior to its normal position, the pressure exerted by an early repaired lip will often correct the condition. Until the deciduous incisors have erupted it is difficult to determine how far the intermaxillary bone really protrudes; and it is often surprising to observe how little correction is needed in what had appeared to be marked protrusion of the premaxilla in a unilateral or bilateral cleft. If in doubt about this point it is better to wait until the eruption of the deciduous incisors, when one may decide with certainty whether there is enough anterior protrusion to warrant the closure of the lip before operation on the palate. Early closure of the cleft of the lip brings very considerable pressure to bear, especially in double cleft or the typical harelip, as the lateral portions are comparatively short and the lip is usually quite tense. Sometimes this pressure is quite efficient, when exerted upon these cases of protrusion of the premaxilla, which are frequent, and of lateral separation of the maxillæ, which are infrequent; but when such pressure is exerted upon cases without protru-

sion or separation, it produces an unfortunate postoperative deformity, and one that is too frequently encountered. It causes the alveolar arch to lose its parabolic curve, and what should be an arch is frequently V shaped or triangular, and not infrequently the cuspid teeth are closely approximated. One thus gets marked flattening of the anterior lateral region of the face or cheek, with loss of contour and position of the upper lip and apparent protrusion of the lower lip and chin. It is true that such a postoperative deformity can be corrected by modern orthodontic methods, but it is better to prevent it than to be obliged subsequently to correct it.

From the preceding remarks it is evident that it is to be regarded as advisable in many cases to close the cleft in the soft and hard palates before operating upon the lip. The best time for operating is just before the patient begins to employ articulate speech. In most cases the cleft in the alveolar process, including the floor of one or both nares, should be repaired separately, and subsequently to the repair of the hard and soft palates. A comparatively short time after operating upon the palate the lip may be repaired, and the lip also should be repaired before the establishment of articulate speech. An advantage in operating with the harelip still unclosed is that one can see better and work better during the operation on the palate, and can give the palate better local care after the operation. Unfortunately, however, many children with cleft palates are never brought for advice until they have cultivated articulate speech. It is always very difficult and often impossible to correct the manner of speaking that they have taught themselves. Only long training and much perseverance is of any avail. The earlier the operation is performed, the better will be the result—not only from the functional standpoint, but also as regards the correction of existing deformity and the prevention of future deformity. So far as obtaining good surgical results go there is practically no set age limit.

If operation is refused for cleft of the hard palate, if it offers no real hope, or if it is very dangerous, an obturator must be worn. An obturator is made by a dentist. In preparing a child for operation I follow Edmund Owen's rules, viz.: Have the child in the best condition, free from cough and stomach disorder. Operate in summer. Place the child under the charge of a nurse several days before the operation.

*Operation for Suture of the Soft Palate* (*Staphylorrhaphy*).—The operation of staphylorrhaphy, which is applied to clefts of the soft palate alone, is a comparatively easy procedure. In performing this operation the patient should be anesthetized and be placed in the Trendelenburg position, or else with the head hanging over the end of the operating table. The mouth is held open by Whitehead's gag, and an assistant holds an electric light and a reflector to illuminate the oral cavity. If the patient is not a young child, the operation may be done under cocain, with the subject sitting erect in a chair and the surgeon sitting directly in front of him.

The surgeon should have at hand several knives of different shapes. The double-edged, pointed knife is an excellent one for freshening the margins of the palate. Special forms of needle-holders have been devised for the purpose of carrying the needle. The heavy, curved, sharp-pointed bistoury is the best instrument for dividing the muscles of the palate; and a sharp hook should be at hand, in order to catch the edge of the cleft, if necessary.

The surgeon first of all separates the soft palate from the posterior edge of the palate bones and from the nasal mucous membrane. This step is necessary in order that the edges may meet in the middle line (Berry). One edge of the cleft uvula is now grasped with a pair of forceps or a sharp hook, and is pulled upon to make it tense. This edge is then pared from below

upward, the piece being continuous from the base to the apex of the cleft. This piece is severed, and then the other margin of the cleft is pared in the same way. It is now advisable to free the margins of the wound from tension. These lateral incisions not only relieve tension, but temporarily paralyze the soft palate. Figures 580 and 581 show the incisions as recommended by Berry. These incisions divide the tendons of the levator palati and the palatopharyngeus muscles and temporarily paralyze the palate. The impairment of palate function is not permanent, as the nerves to the muscles are not cut.

The sutures are inserted by means of a special needle-holder, so arranged that the needle may be directed in many different positions when grasped. The sutures are introduced from below upward, silkworm-gut being used for the uvula and the lower part of the velum, and silver wire for the balance of the cleft. Each suture, as it is passed, is tied or twisted, and it is not cut off until the next suture is inserted, and thus serves as a handle. If there is too much tension to allow of the sutures being tied as they are inserted, all the sutures are passed and lightly twisted before one is tied.

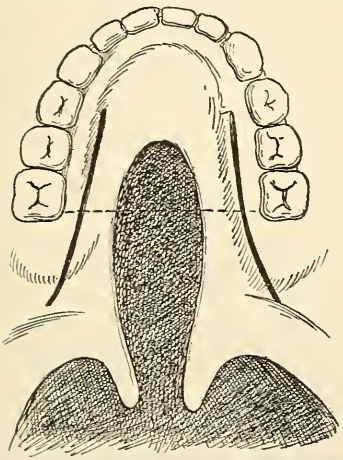


Fig. 580.—Cleft of soft and part of hard palate. Shows exact situation in which the lateral incisions should be made (Berry).

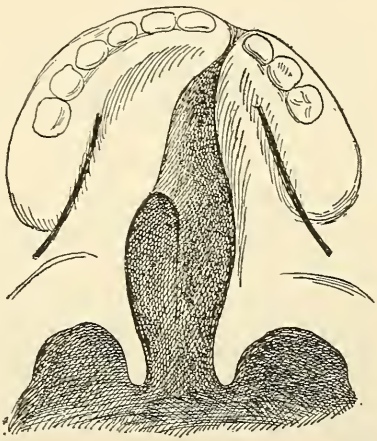


Fig. 581.—Semidiagrammatic view of complete left cleft palate. The septum nasi is attached to the palate on the (patient's) right side. The mucous membrane on the left side of the septum may be detached and brought down if necessary to help in the closure of the anterior half of the cleft. Shows exact situation in which the lateral incisions should be made (Berry).

*Closure of Clefts in the Hard Palate* (*Uranoplasty*).—As previously stated, the best time to perform these operations is during the second year of life. In some few cases we postpone the operation until the end of the third year. If the child learns to talk with the palate cleft, articulation will never be very greatly improved, even by operation. One should, therefore, try to operate before the child learns to talk. Even after the closure of the cleft the speech does not become entirely normal; in fact, as Berry says, it never becomes even very good. One should exercise the greatest care in forming the soft palate, because good articulation is largely dependent upon a well-formed soft palate (Berry, in "Brit. Med. Jour.," Oct. 7, 1905). The surgeon may be able to close the entire gap at one operation; or, owing to undue tension, he may be forced to close it but partly, completing the closure at some subsequent period.

The operation that to my mind is the best is one that uses the soft tissues alone—such a one as is advised by Berry. I have entirely abandoned the operation of wedging the bone over with a chisel. I am satisfied that it

is far more dangerous than is the other method; it is more liable to fail; and, if it fails because of necrosis, it is difficult or impossible to cure the defect by a second operation. The essence of a successful operation, using the soft tissues alone, is, as Berry insists, the complete detachment of the soft palate from the posterior edge of the palate-bone (Fig. 582), because, if one fails to secure this, the edges of the gap will not approximate in the median line. One should also separate the soft palate from the mucous membrane of the nose (Fig. 582).

A second very important point is the imperative necessity of making incisions to the sides to relieve tension and to paralyze for a time the soft palate. The incisions, as recommended by Berry, are shown in Figs. 580, 581. The cut is close to the teeth, and is taken as far posterior as the middle of the soft palate, at the junction of that structure with the lateral pharyngeal wall. In this cut there is some risk of dividing the anterior palatine artery, but hemorrhage from this vessel can be arrested by pressure. Berry insists that the incision need not go forward more than the level of one or two premolar teeth; or, in older children, to the first or second molars. The edges of the fissure are pared on each side, from the tip of the uvula to the top of the gap. Strips of the mucoperiosteum are lifted up on each side of the gap and shifted toward the cleft, and at this stage the posterior border of the soft palate is separated from the posterior border of the hard palate (Fig. 582).

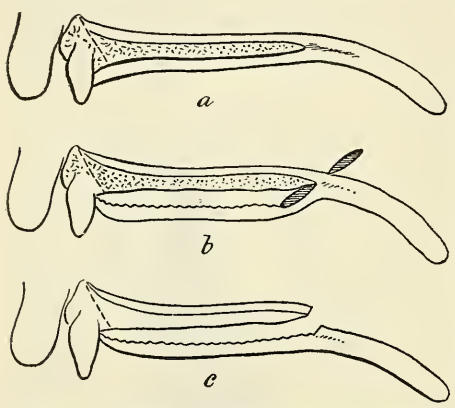



Fig. 582.—Longitudinal vertical section through the hard and soft palates: *a*, Before operation; *b*, palatine mucoperiosteum detached and brought down, blades of scissors introduced to cut attachment of soft palate to the bony palate and to the nasal mucous membrane; *c*, the same after the cut has been made and the soft palate thus brought down (Berry).

The parts are sutured with silver wire, following the advice of Edmund Owen to twist and cut each wire, leaving an end $\frac{1}{8}$ inch in length. This procedure causes the child to keep his tongue from the suture line.

For the first twenty-four hours only water is given. After this period the patient is fed with jelly and liquids. Only fluid or soft food is used for two or three weeks. Talking is forbidden. A day or two after the operation the child should be taken into the open air and kept in it all day. As Owen shows, this greatly stimulates vital resistance and lessens, to a considerable extent, the danger of sloughing of the suture line. The mouth is washed frequently, and always after taking food, with Condy's fluid. The sutures are allowed to remain between two and three weeks.

*Sir William Fergusson's Operation.*—In this operation the mucous edges are pared, the bones are drilled for wires, and the sutures are inserted, but not tied. An incision is made on each side of the cleft down to the bone, each incision being midway between the cleft and the corresponding alveolus. The bone is divided on each side, by means of a chisel, to the full length of the incision; and the chisel is used as a lever to force each half of the bone toward the gap. The sutures are tied, and each lateral incision is plugged with iodoform gauze.

*Brophy's Operation.*—This operation is employed particularly for children under three months of age, and cannot be used when the child is over six months. In this operation the palate is closed before the harelip is touched. Operating at this time the bones are soft, and by leaving the harelip untouched

the surgeon has more room to work. The author of the operation believes that when it is performed at this early age the palate muscles do not atrophy, but develop, and that the patient does not form the evil habit of talking through the nose.

In performing this operation the very strong-handled needles of Brophy are necessary. The patient is anesthetized and put into the Trendelenburg position and a strong piece of silk is put through the tip of the tongue as a traction-suture. The edges of the cleft in the hard palate are pared, a little of the bone being taken away with the paring. Then the edges of the cleft in the soft palate are pared. The needle is threaded with strong silk; the cheek is lifted, and the threaded needle is forced through the superior maxillary bone from without inward, starting just back of the malar process and just above the palate. As the needle shows in the cleft the thread is picked up with a pair of forceps, and the needle is pulled out, the loop of thread remaining in the cleft. Through a part of the opposite superior maxillary corresponding with this first point of entrance the needle is entered again and another loop is got into the cleft. The second loop is caught into the first loop, and when the former is pulled out it carries the latter with it. This thread now passes through both the superior maxillary bones and usually through the nasal septum as well. This thread is used to pull a piece of strong silver wire through. One other silver wire is introduced in the same manner more to the front. The silver wire ends are threaded through perforated lead plates, which fit the external outline of the bones on each side. The wires are tightened and twisted. For instance, on one side the end of the anterior wire is twisted to the end of the posterior wire, and so on. The thumbs are used to jam the two ends of the maxillary bones forcibly together, thus closing the cleft, and then the wires are twisted more firmly to hold the edges in contact. The cleft in the soft palate is then sutured, although the surgeon may deem it advisable to wait one day before doing so. After the palate heals the harelip is closed.

**Carcinoma of the Lower Lip.**—Cancer frequently arises in the lower lip, very rarely in the upper lip. Males suffer frequently, but females are not very often attacked. In some cases it seems to arise in smokers at the point on the lip where the pipe habitually rests. A short-stemmed clay pipe, which grows hot when it is smoked, is particularly apt to lead to the causal irritation. The region of the lip which is most liable to cancer is the junction of the skin and mucous membrane. The growth may begin in a fissure or abrasion, may start in an eczematous area, but most frequently arises as an indurated area which quickly ulcerates. After a cancer has existed for a variable time the submental, submaxillary, and cervical lymphatic glands become diseased. These glands are usually involved within three months of the beginning of the cancer. In a case of my own they were found to contain carcinoma cells in less than three months after the origin of the carcinoma of the lip. This involvement cannot be detected by external manipulation in the earliest stages, hence it is not proper to conclude that the glandular involvement is absent simply because it cannot be palpated. It occasionally happens that glands enlarge because of septic absorption, and this enlargement may even precede carcinomatous involvement. From an operative point of view the glands should always be regarded as carcinomatous. If cancer is not operated upon it destroys the lip, extensively involves the glands of the neck, the floor of the mouth, the periosteum and the lower jaw, and produces death in from three to five years. If the jaw is involved the prognosis is bad, and it is almost hopeless if the floor of the mouth is involved.

The *treatment* consists in the early and thorough removal of the growth by the knife, and also in the removal of the fatty tissue and glands from the

submaxillary triangles, from the submental region, and down to the carotid bifurcation. The growth must be thoroughly removed, that is, the incision must be at least ½ inch wide of the disease. For many years a favorite operation was the V-shaped incision, the skin edges being sutured by silkworm-gut, the sutures being passed almost to the mucous membrane and being inserted so as to compress the vessels when tied, and the mucous membrane being sutured with fine silk or catgut. The V-shaped incision should be used only for a very small and very recent growth. After the removal of the growth from the lip a vertical incision is made from the point of the V over the cricoid cartilage, and from the origin of this incision incisions are made in each direction along the under surface of the body of the jaw. The glandular area is thus exposed, and after the removal of the fat and glands the wound is sutured with silkworm-gut. Far better than the V-shaped incision is the operation devised by W. W. Grant, of Denver.[1] In this operation the growth is removed and cheiloplasty is performed.

*Grant's Operation for Cancer of the Lip.*—This operation gives a useful mouth and a more natural-looking lip than does the ordinary operation, and there is decidedly less tension on the suture line. Furthermore, the suture line in a man is apt to be soon covered with a beard. The procedure has

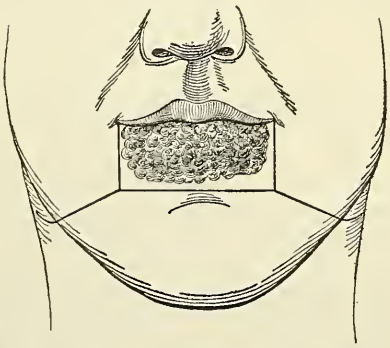


Fig. 583.—Grant's method for removal of carcinoma of the lower lip. The incision.

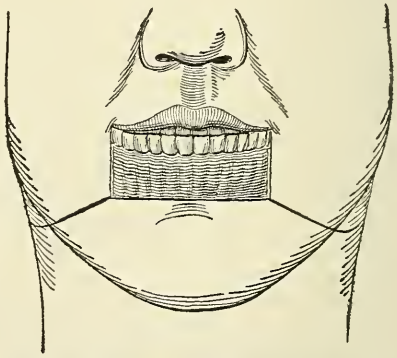


Fig. 584.—Grant's method for removal of carcinoma of the lower lip. Second step. The mass removed.

great advantages over the ordinary V-shaped operation, which greatly lessens the size of the mouth, making it what is known as a sucker-mouth, and the new lip is rigid and ugly.

In Grant's operation two vertical incisions are made, one on each side of the growth, and these are connected by a horizontal incision at the base (Figs. 583, 584). Thus, a quadrangular gap is formed, which must be filled by flaps. An incision is made on each side from each inferior angle of the wound, obliquely downward and backward beneath the maxilla, on a line about midway between the angle of that bone and the apex of the chin (Fig. 583). Its further extension is determined by the amount of lip removed and by the degree of glandular involvement.

The submaxillary lymph-glands are removed through these incisions. The glands in the midline, however, beneath the chin may require a separate incision. If the lip is extensively involved, the cheek ought to be completely separated from the inferior maxillary bone to the middle of the masseter muscle (Fig. 585). When the glands have been removed, the triangular flaps are brought together and united, first of all, in the middle line (Fig. 586). If the tension is marked, owing to the amount of tissue excised, it is wise to insert

[1] "Medical Record," May 27, 1899.

a traction suture, $\frac{3}{4}$ inch from the center line, and tie it over pads of gauze covered with muslin. One thus prevents undue tension upon the sutures in the center of the flap. The stitches that unite the cheek posteriorly are inserted and tied, and the entire thickness of the cheek must be included. Silkworm-gut sutures are used. A drainage-tube is inserted in the posterior angle of the wound on each side. It is very useful to use also a T-drainage-tube as advised by Grant. This tube is about the diameter of a lead pencil and the cross-piece rests behind the incisor teeth or symphysis and beneath the tip of the tongue. It drains away all of the mouth secretions, saves the lines of incision from being constantly bathed in them, and renders very frequent changes of dressing unnecessary.

I have employed this operation repeatedly, and regard it as the most useful method we have for the purpose. Thorough removal of the carcinoma of the lip and of the related glands will cure from 60 to 70 per cent. of cases.

**Carbuncle of the Upper Lip.**—In contrast to carbuncle in other regions of the body, facial and labial carbuncles are most common in young persons. Carbuncle of the lip is due to staphylococcus infection and begins as a papule. Numerous pustules appear, and sloughing usually takes place. There may or may not be serious constitutional involvement. The condition is

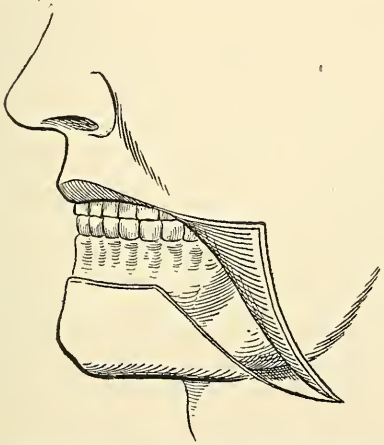


Fig. 585.—Grant's method for removal of carcinoma of the lower lip. Dissection preliminary to suturing.

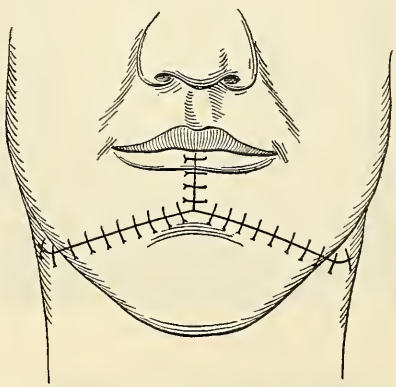


Fig. 586.—Grant's method for removal of carcinoma of the lower lip. The wound sutured.

very dangerous, as thrombophlebitis may arise and track up into the cranium by way of the ophthalmic vein and cavernous sinus. I have known two persons to die from carbuncle of the lip.

*Treatment.*—Excise if possible. If excision is impossible, make a crucial incision, cutting away the corners and edges with scissors. Scrape out the carbuncle with a sharp and strong curet, swab with pure carbolic acid, pack with iodoform gauze, and dress with antiseptic poultices.

**Tongue-tie** (*congenital ankyloglossia* or *adherent tongue*) is congenital shortness of the frenum, the tip of the tongue adhering to the floor of the mouth. It is due to the projecting portion of the tongue being incompletely developed from the tuberculum impar. "In many of the slighter cases the development has merely lagged behind, and will be completed as the child grows after birth" ("Diseases of the Tongue," by Henry T. Butlin, Second Edition). The tongue cannot be protruded beyond the incisor teeth. Swallowing is interfered with, and later in life articulation is impeded. It is not very unusual in infants, but in the great majority of cases disappears as the

child grows older. Persisting tongue-tie, Butlin says, is one of the rarest of conditions, and my experience is in absolute accord with his—in fact, I have never seen a single case. Many unnecessary or even harmful operations are done for a condition which, if let alone, will usually correct itself. Improper operation may result in fatal hemorrhage or in "swallowing of the tongue." The operation usually done is to tear up the frenum with a thumb-nail. This is unsurgical and makes a lacerated wound. A better way is to raise the tip of the tongue to make the bands tense, and then snip with the scissors close to the mucous membrane of the lower jaw. The slit in the handle of the grooved director was placed there to catch the frenum in, but a short frenum will not enter it (Butlin).

**Ranula** is a retention-cyst of the duct of the submaxillary or the duct of the sublingual gland. A ranula when first formed contains saliva, but after a time the saliva undergoes a change, and in appearance comes to resemble mucus. *Mucous cysts* occur in the floor of the mouth, resulting from obstruction of the ducts of the *mucous glands of Nuhn and Blandin.* These glands lie on each side of the frenum of the tongue. Such a cyst is often spoken of as a ranula. A *cyst of the incisive gland* forms just back of the lower jaw and lifts up the frenum. A true ranula appears upon the floor of the mouth on one side and pushes the tongue toward the opposite side (Fig. 587). The *treatment* of a mucous cyst is by excision of a portion of the cyst wall and cauterization of the interior with pure carbolic acid; or by cutting a flap from the cyst wall and stitching it aside so as to keep a permanent opening. Such an operation may cure a genuine ranula, but will often fail. In true ranula an external incision should be made, and through this both the cyst and the gland should be removed. This plan is recommended by Mintz.[1]

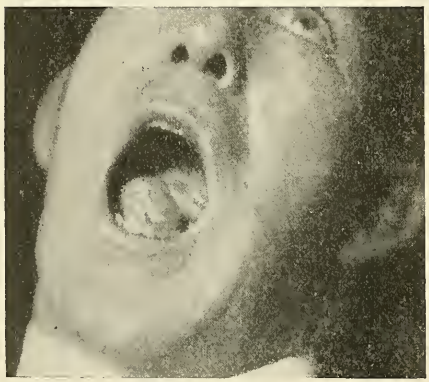


Fig. 587.—Ranula.

**Thyrolingual or Thyroglossal Cysts and Sinuses.**—In early embryonal life the thyroid gland has a duct which passes from the thyroid isthmus to the foramen cæcum of the dorsum of the tongue. The duct may be lined with one layer, two layers, or several layers of epithelium, and there are mucous glands and lymph-follicles in its walls, these structures being derived from the mucous membrane of the tongue. The wall of the duct presents numerous irregularly placed and irregularly shaped diverticula. It is known as the thyroglossal or thyrolingual duct. The duct runs from the base of the tongue down the midline of the neck. It is connected with the body of the hyoid bone, with the periosteum in front of the bone, and with the thyrohyoid bursa behind the bone. It passes to the upper portion of the front surface of the trachea, where it bifurcates, each branch passing to a lateral lobe of the thyroid gland. This fetal structure under normal conditions begins to atrophy in the fifth week and closes by the eighth week, the foramen cæcum marking its old orifice on the dorsum of the tongue. When the duct is obliterated, it becomes a cord of epithelium. In more than 30 per cent. of bodies the remains of this primitive passage can be found (Weglowski, in "Zentralb. f. Chir.," 1908, xxxv, 289). The duct may persist between the foramen cæcum and the hyoid bone, developing, it may be, into a *sublingual dermoid.* The portion behind and below the

[1] "Zeitschrift für Chirurgie," March, 1899.

hyoid may remain and develop into a *subhyoid cyst.* The part inferior to the hyoid may persist, give origin to a cyst which ruptures, and constitute an *incomplete median cervical fistula.* The duct may remain open from the mouth and make, by bursting an opening in the neck, a *complete median cervical fistula.* A patent duct may exist for years and announce its existence by some acute inflammatory process. The small diameter of a cervical fistula renders probing to any depth impossible. Some have told us to determine if a fistula is complete by injecting quassia solution into the lower end. The patient will perhaps experience a bitter taste. If we inject a colored fluid we may see it if it runs from the mouth. I have never succeeded in doing either. Tumors may spring from the duct.

*Treatment.*—If a thyroglossal cyst or tumor arises on the dorsum of the tongue, and if it is increasing in size and interferes with swallowing and speech, it must be removed through the mouth. A general anesthetic should be given. In some cases preliminary tracheotomy is necessary.

A cyst, tumor, or fistula about the hyoid bone requires excision, the patient being under the influence of a general anesthetic. A portion of the cyst wall adheres strongly to the posterior surface of the hyoid bone and must be carefully removed even if it is necessary to split the bone to accomplish it. In treating fistula the surgeon makes an elliptical incision of the skin about its orifice so as to free the fistula from the subcutaneous tissue. When traction is made upon the cutaneous end of the duct it will stand out clearly and can be dissected out (M. S. Seelig, in "Surg., Gynecol., and Obstet.," May, 1907). It is useless to try to cure a fistula by cauterization. A fistula requires the complete removal of its epithelial-lined walls. No lesser operation will cure. In 1 case I operated four times before securing success. In another case I divided the hyoid bone, removed the fistula, sutured the bone by chromic gut, and obtained a cure.

**Carcinoma of the Tongue.**—This is one of the most dreadful forms of cancer. It is a quite common disease. In most of the cases I see it is far advanced when first brought to the hospital. The only form of cancer which attacks the tongue is epithelioma. It is much more common in men than in women. It is a disease of adult life and is very rare before the age of thirty-five. It begins, as a rule, near the tip, on the side or at the base of the anterior two-thirds of the tongue, as a warty growth, as an ulcer having at first a papillary structure, as a fissure which indurates, or as an indurated area which ulcerates. The cause of the growth may sometimes be traced to the irritation of a jagged tooth or an ill-fitting plate, or to the smoking of a pipe, or to holding nails in the mouth, as is done by those who nail laths. Cancer may follow a chronic inflammation—leukoplakia, for instance. Chronic ulcers are liable to become cancerous and any indurated ulcer has potentialities of deadly peril and should be promptly removed. Fournier regards syphilis as an influential cause, and states that in 184 cases of cancer of the mouth or tongue 155 had had syphilis. There has been no such proportion of syphilitics in my personal cases. In Whitehead's 104 cases only 7 had had syphilis. As in cancer of the lip, men are much more frequently affected than women. In most cases the disease spreads rapidly; produces early and extensive glandular involvement; disease of the floor of the mouth; dribbling of saliva; difficulty in masticating, swallowing, and talking; foulness of the breath; severe pain which usually radiates toward the ear, and often a fatal septic trouble. Cases not operated upon usually die decidedly within two years. There is a very rare form of carcinoma described by Wölfler, which grows very slowly or even remains latent for years.

One reason why cancer of the tongue grows so rapidly has been pointed out by Heidenhain, of Greifswald. The lingual muscles are contracting almost

constantly, and as a result cancer-cells are forced along the lymph-spaces to healthy areas.

*Treatment.*—A cancer of the tongue should be removed radically at the earliest possible moment. Specific treatment for diagnostic purposes should not be continued beyond a very few weeks. In doubtful cases a Wassermann test is made, and if it is positive, salvarsan is given intravenously. If still in doubt as to the nature of the growth, remove it and have a pathologist at hand to immediately study it by frozen sections (Warren, in "Annals of Surgery," Oct., 1908). Have permission beforehand to proceed at once to radical operation if conditions demand it. The study of a small piece is always of uncertain value, hence, take out the growth. Whatever it is, it should be removed. Before any operation is undertaken all stumps of teeth should be extracted and a dentist should clean tartar from the teeth. During several days preceding an operation the teeth should be scrubbed twice a day with a brush and soap and the mouth rinsed with hydrogen peroxid. The nares and nasopharynx should be sprayed with peroxid of hydrogen and then with boric acid solution every second or third hour when the patient is awake.

In some cases the entire tongue is removed; in some, half of it; in some, only a piece of it. Not only the diseased tongue, but also the adjacent lymphatic glands must be removed. Cancer of the tip of the tongue, as a rule, involves the submental and sublingual group of glands early. Cancer of the anterior two-thirds of the dorsum of the tongue first involves the lingual and submaxillary lymph-nodes. Cancer of the under surface of the tip of the tongue first involves the submaxillary glands. Sooner or later the superior deep cervical glands about the carotid bifurcation become involved as a result of cancer of the tip or edges of the anterior portion of the tongue. In cancer of the dorsum the deep cervical glands become involved as well as the superficial nodes. The lymphatic system of the base of the tongue is distinct from that of the balance of the organ. It drains into the deep cervical groups.

It was formerly my belief that in a very recent and limited case only the glands on the diseased side require removal, but that in an advanced case the glands must be removed from *both sides* of the neck. Experience has convinced me that in any case the glands on both sides should be removed. Kuttner, of Tübingen, has demonstrated that lymph from one side of the tongue may flow to glands on the same side of the neck, but some also may flow to the opposite side of the tongue. Remove the obviously involved glands by the *block dissection* of Crile. In a bad case everything is removed but the carotid arteries. The sternocleidomastoid muscle, the omohyoid, the jugular vein, even the pneumogastric and phrenic nerves of one side may be taken away. After a week or two the other side of the neck should be operated upon. It seldom requires a wide removal of structures. If the pneumogastric or phrenic were cut on one side it must be preserved on the other. The jugular vein can be removed after a collateral circulation has been established subsequent to removing the jugular of one side. Two operations are to be considered: partial removal and complete removal.

*Partial Removal of the Tongue.*—This operation is restricted to recent cases in which one side only of the anterior portion of the tongue is involved. The operation does not offer as good a chance of cure as complete excision, because lymph containing cancer-cells may have reached the opposite side of the tongue. Even in partial removal the glands should be removed from both sides. Intratracheal anesthesia is employed.

In performing the operation of partial excision introduce a mouth-gag, pass a silk ligature through each half of the tip of the tongue, and draw the organ out of the mouth. Place the patient recumbent with the head a little raised. Split the tongue back in the middle line by the scissors, and loosen

the cancerous side from the floor and side of the mouth. Pass a stout silk ligature through the base of the tongue posterior to the cancer. Draw the organ out and cut off the diseased side in front of the ligature, but well back of the disease. Tie the vessels, remove the traction threads, and treat subsequently as in cases of complete removal.

*Complete Removal of the Tongue (Kocher's Method).*—Kocher recommends a preliminary tracheotomy in tongue excision, but the Trendelenburg position renders this procedure unnecessary so far as fear of the passage of blood into the larynx and trachea is concerned. I operated many times with the patient in that position. At present I operate with the head a little raised and the patient taking ether by intratracheal insufflation. Because of the insufflation there is no respiratory difficulty and the stream of escaping air and ether keeps blood out of the bronchial tubes. The method is most satisfactory. The surgeon stands to the side. Ether is given by intratracheal insufflation (see page 1199). Ligate the lingual artery on the side opposite to the one where the main incision is to be made. Remove the glands on that side and suture the wound. An incision is then made on the side opposite to that on which the artery was ligated. This incision passes from behind the lobe of the ear, along the anterior edge of the sternocleidomastoid to about the middle of the margin of this muscle. From this point the incision is carried to the level of the hyoid bone and then to the symphysis menti, along the anterior belly of the digastric muscle (Fig. 588). The flap is dissected and turned up; the facial and lingual arteries are ligated; "the submaxillary fossa is evacuated" (Treves); the sublingual and submaxillary glands are removed; the mylohyoid muscle is divided; the mucous membrane is incised close to the jaw, and the tongue, caught by tenaculum forceps, is drawn through the opening. The tongue is split in the middle by scissors, and the near half is removed, bleeding is arrested, the remaining half of the tongue is cut through, and the vessels are tied. Stitch the mucous membrane of the stump to the mucous membrane of the floor of the mouth with catgut sutures. Kocher does not suture the skin wound. I prefer to suture it and employ drainage-tubes. I follow the suggestions of Treves as to after-treatment. Some hours after the operation, when oozing has ceased, dust the mouth wound with iodoform. The patient, as soon as possible, is propped up in bed, and he must not swallow the discharges if it can be avoided. The mouth, every half-hour, is sprayed with peroxid of hydrogen and washed with a carbolic solution (1 : 60). Every three hours, after washing the floor of the mouth and the stump, the parts should be dried with absorbent cotton and dusted with iodoform. For twenty-four hours after the operation nothing is given by the mouth except a little cracked ice, the patient being fed by rectum. At the end of twenty-four or forty-eight hours some liquid food is given from a feeding-cup. The patient will soon learn to swallow; but if he cannot swallow easily, he is fed by a tube. Treves, in his clear and positive directions for after-treatment, states that nutrient enemata are to be continued until sufficient nourishment is taken by the mouth; that the mouth should be flushed by irrigation, and must be washed immediately after taking food; that morphin is to be avoided, and that the patient can usually leave the hospital in from seven to ten days.

Fig. 588.—Kocher's excision of tongue (Esmarch and Kowalzig).

*Whitehead's Operation.*—Whitehead removes one-half of or the entire tongue from within the mouth by the use of scissors. He passes a ligature through the tip, cuts the frenum, draws the tongue strongly forward, and separates by a series of clips with the scissors. The lingual arteries are tied as cut. "The stump should be kept under control, as regards hemorrhage, by a stout silk ligature passed through the remains of the glosso-epiglottidean fold and retained for twenty-four hours."[1]

Heath has shown that if the forefinger be passed to the epiglottis and used to "hook forward" the hyoid bone, the lingual arteries are stretched and portions of the tongue can be removed almost without bleeding. It is rarely desirable in Whitehead's operation to remove the glands and the tongue at one séance. To do so increases shock and the danger of death. The rule of procedure set forth by W. Watson Cheyne[2] is eminently wise. This rule is as follows: If glandular involvement is trivial or not detectable, it is perfectly proper to remove the tongue first, and after a week or so remove the glands. If the glandular involvement is marked, growth in the glands will be much more rapid than growth in the tongue. In such a case the glands should be removed before the tongue, because, if the tongue is removed before the triangles are cleared, in the week or two of waiting the case may become inoperable. In the majority of cases clear out the triangle before removing the tongue, doing the other operation in one or two weeks when the wound in the neck is healed. If the disease in the mouth is far advanced, do both operations at one séance.

**Examination of the Esophagus.**—The *x*-rays are of great value not only in detecting foreign bodies, but in finding carcinoma, pouches, and constrictions. As Waggett ("Brit. Med. Jour.," Oct. 19, 1912) says, by means of the *x*-rays we may learn that there is a stricture (or are strictures), where it is (or they are), how narrow it is (or they are), and whether or not there is extrinsic pressure. In this examination the patient first swallows material through which the rays pass with difficulty. A salt of bismuth is generally used. The carbonate of bismuth is a safe and satisfactory salt. It is given in glutoid capsules (Kohler, Ibid.).

*Esophageal Sounds and Bougies.*—These instruments were long our only mechanical means of diagnosis. They are used far less than formerly. They possess certain dangers. For instance, if an aneurysm exists and we are misled in believing that the condition is stricture, the rigid sound and even the flexible bougie may penetrate the sac of the aneurysm and cause death. I have personal knowledge of such a case. If a person has ever brought up blood, neither a sound nor bougie should be used. Again, neither the bougie nor sound can prove the existence of a slight stricture. They give no information at all as to the nature of a stricture. I find their greatest diagnostic use is to locate the situation of a constriction before passing the esophagoscope. In view of the fact that such an examination may cause bleeding, and that blood interferes with an examination by the esophagoscope, the bougie or sound, if used at all, should be employed the day before the introduction of the esophagoscope.

The olive tip bougie is flexible, and is made from elastic web. It is a safer instrument than the bulbous sound (see Fig. 597, E). The latter has a series of removable ivory or metal tips which vary in size. Used diagnostically, the olive tip bougie is to impart information as it enters; the bulbous sound, as it enters and as it is within. On withdrawing the bulbous sound it may catch upon the lower border of a constriction which it passed on entering.

Before being passed the bougie or sound should be warmed and greased with glycerin. The patient sits in a chair and throws the head well back against the breast of an assistant. The mouth is opened widely and is held open by a

[1] "American Text-Book of Surgery."
[2] "The Practitioner," April, 1899.

large cork or by a gag. The patient is directed to breathe deeply and regularly while the instrument is being passed. Depress the tongue with the finger and carry the instrument beyond the glottis. As it reaches the back of the pharynx the patient will gag and choke. Tell him to swallow and breathe regularly. The fact that he can breathe regularly shows that the instrument is not within the larynx. It may then be gently urged along the gullet. All maneuvers are to be conducted with the utmost gentleness. Force means danger.

Remember that in an adult the esophageal orifice is 5 or 6 inches from the incisor teeth, that the esophagus has a length of from 9 to 10 inches, hence, that the cardiac orifice of the stomach is from 14 to 16 inches from the incisor teeth (Maylard's "Surgery of the Alimentary Canal"). It is further important to remember that the normal esophagus is of smaller caliber in some regions than

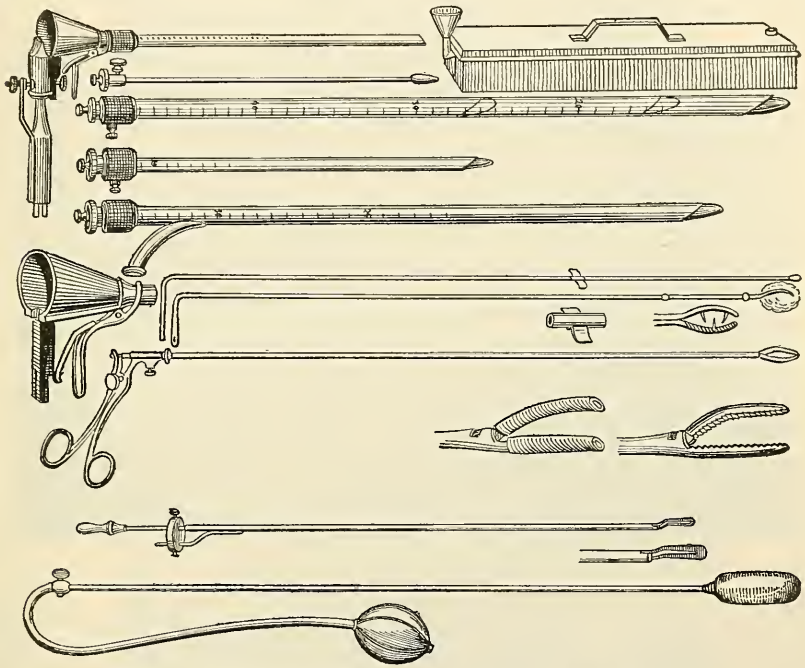


Fig. 589.—Von Mikulicz's set of instruments for esophagoscopy.

in others. There are four points of physiological narrowing, viz.: On a level with the cricoid cartilage, where it is crossed by the aorta, where it is crossed by the left bronchus, and where the tube passes through the diaphragm.

*Auscultation* may enable the surgeon to hear the food bolus rub against a stenosis and to note delay in the passage of liquid. Fluid normally passes in four seconds.

*The Esophagoscope.*—This instrument is of the highest value, a value just beginning to be properly appreciated. It is not altogether free from danger. Deaths have occurred from it. The mandrel does the harm, and should not be used at all or should be withdrawn as soon as the tube has passed the cricoid constriction. The rest of the way the instrument is carried along while the surgeon is looking through it and seeing what is ahead. It is not carried into a constriction; it is not carried by an ulcer or a pulsating lump. It enables us to make diagnoses otherwise impossible, to diagnosticate cancer in an early stage, to treat local conditions, and to remove a fragment of tissue for examination.

I prefer to pass the instrument without general anesthesia except in children, highly nervous people, or cases of very severe spasm. Apply cocain to the back of the tongue, pillars of the fauces, epiglottis, and pharynx. Place the patient on the right side with the head thrown back. This is known as *Starck's position*, and, as Gottstein shows, it relaxes the diaphragmatic crura. An assistant supports the head and follows every movement of the surgeon. The patient is cautioned to breathe tranquilly, and is told if the pain or annoyance becomes intolerable to raise his left arm, when the surgeon will cease for a time. The surgeon must, of course, keep his word on this point. Artificial teeth are removed. The instrument is kept in the midline and is inserted as is a sound or bougie. As soon as it passes the cricoid narrowing the mandrel is withdrawn, and the instrument is passed slowly and gently along while the surgeon is looking through and ahead of it. For Chevalier Jackson's method of esophagoscopy see page 888.

Fig. 590.—Position of patient during esophagoscopy (after von Mikulicz).

The cervical esophagus is closed and seems to unroll before the instrument. The thoracic esophagus is open, and while the esophagoscope enters into the upper part of this region, if the gullet is normal, the surgeon can see all the way down to the diaphragm. At the diaphragm the esophagus bends forward and to the left and this point must not be mistaken for the cardia (Kohler, in "Brit. Med. Jour.," Oct. 19, 1912).

The cardia may be found closed or may be seen to open and close with respiration. This instrument is of great value in the extraction of foreign bodies, in the diagnosis and treatment of many esophageal diseases, and in the diagnosis of certain peri-esophageal conditions. Every surgeon should be able to use the esophagoscope. (See Gottstein, in "Keen's Surgery," vols. iii and vi; Waggett, in "Brit. Med. Jour.," Oct. 19, 1912; Kohler, in "Brit. Med. Jour.," Oct. 19, 1912; "Reports of Eightieth Meeting of the British Medical Assoc.," July, 1912; Lewisohn, in "Annals of Surgery," Jan., 1913.) For Chevalier Jackson's directions as to the use of the esophagoscope see page 888.

**Stricture of the Esophagus.**—*Fibrous* or *cicatricial* stricture or scar of the esophagus is due to the healing of an ulcer, and results from traumatism, chronic inflammation, scarlet fever, syphilis, tuberculosis, chronic ulcer, prolonged vomiting, variola, gout, or to swallowing a corrosive substance or a boiling

liquid. In about 15 per cent. of cases of scarlet fever there is inflammation of the esophagus and larynx and stricture may result. Fibrous stricture is commonest in the young, and is apt to be situated opposite the cricoid cartilage, at the tracheal bifurcation or near the cardiac end. Cicatricial strictures, except when due to boiling or corrosive liquid, are usually single, but may be multiple. A cicatrix may be of irregular shape, may be cylindrical, may be annular, may be narrow, or may be broad. Stricture following impaction of a foreign body is located at the seat of impaction unless the tube has been injured by efforts at extraction, in which case multiple strictures may exist (Maylard). Strictures which result from swallowing boiling fluid or corrosive liquid are usually very extensive, may be multiple, and give early symptoms. In

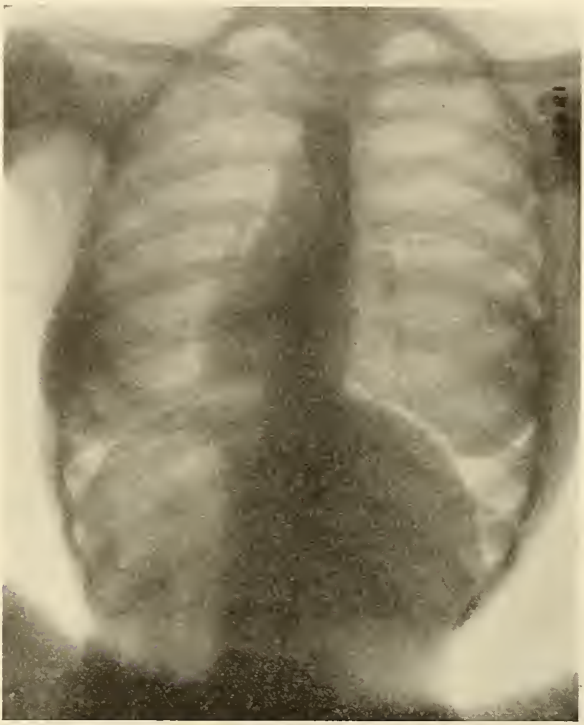


Fig. 591.—Cicatricial stricture of esophagus.

some cases they are slight and may not give symptoms for years after the injury. Syphilitic stenosis is due to the healing of a gummatous ulceration, but there is nothing characteristic in this kind of stenosis. Tuberculous stenosis is extremely rare. The esophagus above an extensive scar is usually dilated.

*Symptoms of Cicatricial Stenosis.*—The condition is most common in youth, but may begin at any age. The chief symptom is difficulty in swallowing, at first slight, but becoming more and more pronounced until swallowing is almost or quite impossible. The dysphagia is first manifested to dry solids, then to all solids, and finally to liquids. In some cases vomiting occurs after swallowing. If the stricture is high up, the vomiting is almost immediate; if it is low down, the vomiting is delayed, especially if the canal is dilated above the stricture. From time to time the patient vomits independently of taking food, the ejected matter containing no gastric juice, only saliva and mucus which gathered in the dilated gullet about the scar. The vomited matter is not bloody. The

patient feels weak, hungry, and thirsty, becomes exhausted and emaciated, and suffers from flatulence, gastralgia, and constipation.

There is occasionally slight uneasiness or even pain in the region of the stricture, possibly "about the epigastrium or between the shoulder-blades" ("The Surgery of the Alimentary Canal," by Maylard). If there is certainly no aneurysm and if blood has never been brought up, the flexible bougie may be used first and then the solid tipped sound, in order to find a stricture. The stricture may be located by auscultation over the spine on a line with the supposed obstruction. While a patient is swallowing water, the arrest of the fluid at the seat of stricture may be audible. Even if the fluid passes, it will be delayed for a time and the duration of deglutition is thus prolonged. In order to determine the time of deglutition put the ear just below the angle of the left scapula, or else between the left sternocostal margin and the xiphoid cartilage, place a finger on the patient's Adam's apple, and hold a watch in the other hand. Have the patient take a drink of water. Count the time from the moment the Adam's apple begins to rise until the fluid is heard to gurgle into the stomach (Ogston's method). It ordinarily requires four seconds for fluid to pass from the mouth into the stomach (Maylard, Ibid.). The *x*-rays are used to diagnosticate stricture and to locate it. They are valuable in diagnosis. An emulsion of bismuth is swallowed and a skiagraph is taken. The bismuth is seen on the plate as a black mass extending above the seat of constriction (Fig. 591). A bougie can be passed until it reaches the block and a skiagraph may be taken with the bougie in position.

In a case reported by Seelig ("Surgery, Gynecology, and Obstetrics," Sept., 1908) the patient was directed to swallow a fine gold chain as thick as ordinary wrapping twine. The chain was about 2 feet long. If a diverticulum exists the chain will fill the sac and a skiagraph will show the position of the diverticulum. If no diverticulum exists the plate will show the chain nearly in the middle line of the body. The esophagoscope should be used (see page 931).

The history of the case is of much importance in diagnosis. The surgeon must inquire about impaction of a foreign body, or swallowing of acids, alkalis, or boiling fluids; and must examine for evidence of syphilis. If there is no history of injury, syphilis, tuberculosis, scarlatina, variola, or prolonged vomiting, and the patient is over forty years of age, the indications point to cancer rather than cicatricial stenosis. The easy passage of a bougie when the patient is anesthetized shows that spasm is the cause, and not organic disease. Narrowing due to external pressure (*compression stenosis*) is marked by positive symptoms of the causative disease.[1] Compression stenosis may arise in goiter, vertebral growths, enlargement of the heart, glandular enlargement, peri-esophageal abscess, aneurysm, lordosis, and mediastinal tumor (Kohler, in "Brit. Med. Jour.," Oct. 19, 1912).

*Treatment.*—Thiosinamin is given by some physicians, but I have never seen it accomplish the slightest good. Telleky[2] recommends it in old scars without inflammation. He makes a 15 per cent. alcoholic solution and injects from ½ to 1 syringeful at a dose, throwing the fluid beneath the skin between the scapulæ. He uses twenty doses in the course of two weeks. *Gradual dilatation* through the mouth is a method employed for at least a time in almost every case. It is the method of choice when it can be carried out, and usually it can be carried out. Begin with the largest flexible bougie which will easily pass. Warm the bougie, oil it, pass it gently, and hold it in position for several minutes, prolonging the time of retention of the bougie as treatment progresses. Pass an instrument every second or third day, gradually increasing the size. Plummer ("Northwestern Lancet," Jan. 15, 1912) dwells on

[1] See the valuable article in Maylard's "Surgery of the Alimentary Canal."
[2] "Wien. klin. Woch.," Feb. 20, 1902.

the danger of using bougies, especially in cancerous constriction, and advocates the use of the string guide of Mixter in tight or tortuous stenoses. Mixter's plan is to have a patient put a piece of silk on the back of the tongue and swallow it while drinking water. The string may thus be floated through. Plummer proceeds as follows: He takes a spool of buttonhole twist and inserts a safety-pin so as to make a reel. He unwinds the thread, marks it with black ink at intervals of 1 yard, rewinds it, takes a few inches of the free end of the thread, moistens it, places it on the back of the patient's tongue, and gives the patient a swallow of water. The patient is directed to swallow 2 or 3 yards before bedtime, "and an equal amount at the rate of a foot an hour the following forenoon." The first portion, in a state of snarl, enters the intestine during the night. By afternoon the thread may be rendered taut by pulling upon it, yet it will not be pulled out. The thread may remain in place for some time, dilating the stricture. It may be used as a guide to direct a bougie through a tight or tortuous constriction, the eyed bougie being threaded upon the strand (Plummer, in "Collected Papers of the Mayo Staff in Rochester," 1911). If the stenosis involves a considerable portion of the esophagus, gradual dilatation will almost certainly fail to cure.

Symonds advocates the insertion of a tube through the stricture and leaving it in place until there is decided dilatation, and then replacing the tube with a larger instrument. The patient is fed through the tube (Fig. 592). In some cases in which it is impossible to pass a bougie through the stricture

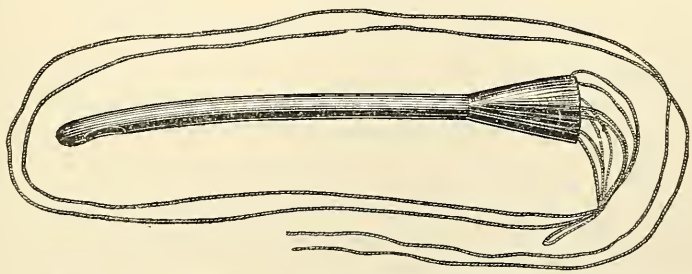


Fig. 592.—Symonds's short tube for intubation of the esophagus (Morrow).

by the ordinary plan it is possible to pass one when viewing the opening through the stricture by means of the esophagoscope. Whalebone or olive-tipped instruments may be passed in increasing sizes. Strands of silkworm-gut may be gotten through. If they are, they can be left in place a few hours, when a larger bundle of gut or perhaps an instrument can be passed. Surgeons have divulsed strictures and performed internal esophagostomy through an esophagoscope. Either of these plans is preferable to forcible dilatation or internal esophagostomy by a special instrument, but without the esophagoscope.

Electrolysis has been advocated by Fort and others. Gradual dilatation from below has been practised in cases in which a bougie could not be passed from the mouth. A gastrostomy is performed, and after the fistula has become sound the patient is made to swallow "a shot to which is attached a silk thread" (Maylard). The silk thread is brought out through the fistulous orifice and is attached to a bougie, and the dilating instrument is pulled up through the esophagus. *Forcible dilatation* can be employed through a gastrotomy opening, by means of bougies, tents, or divulsing instruments. A fibrous stenosis in the region of the cricoid cartilage which is not cured by gradual dilatation should be treated by the operation of *external esophagotomy*. In this operation the stricture is divided by a longitudinal incision; "funnel-shaped retraction of the cut portion is caused by adhesion to the

external tissues divided, and it lessens future contraction."[1] If dilatation fails in the case of a stenosis anywhere above the line of the aortic arch, the esophagus may be opened above the stricture (external esophagotomy). If the stricture is below the wound a tenotome may be introduced through the wound, the stricture is cut and well dilated by the passage of instruments. This operation is known as Gussenbauer's combined esophagotomy.

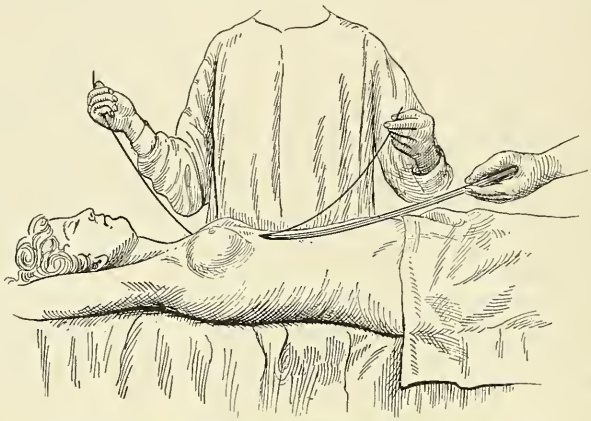


Fig. 593.—Abbe's method of cutting esophageal strictures.

If a stricture is impassable from above, the stomach should be opened and retrograde dilatation be carried out. Billroth showed years ago that a stricture impassable from above may be passable from below. This is because the esophagus above the stricture is basin shaped and immediately below the stricture is funnel shaped (Abbe). If a fine bougie is carried from the stomach to the mouth it is used to carry a piece of string through the same route, and this string is used to pull bougie after bougie through the stricture. A firm, non-dilatable stricture in the thoracic portion of the esophagus can be treated by Abbe's method (Figs. 593, 594). He performs a gastrotomy, sutures the stomach to the abdominal wound to prevent contamination of the peritoneum, and seeks for the esophageal opening by two fingers passed within the stomach. Abbe points out that finding the orifice would seem much more easy than it is. In a recent case I found it only after a prolonged search, the entire esophageal region feeling smooth to the touch. Abbe says that "this surface is maintained by the circular sphincter muscle layers, and it is not until a moment's pressure of the finger at the right place causes them to yield that it slips upward into the esophagus" ("Med. Record," Nov. 30, 1907). Abbe then passes a long filiform whalebone bougie from the stomach into the mouth, ties a piece of braided silk to the bougie, withdraws the instrument, and leaves the silk in place. One end of the silk emerges from the mouth and the other end from the gastrotomy wound. In some cases he opens the stomach and also opens the esophagus above the stricture; one end of the string

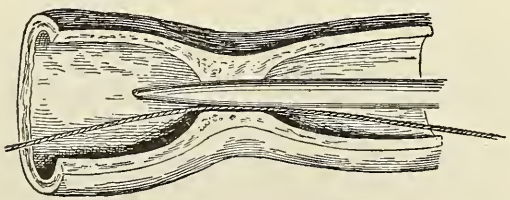


Fig. 594.—The bougie engaged in the stricture while the string-saw is being used.

[1] W. J. Mayo, "Jour. Amer. Med. Assoc.," July 29, 1899.

comes out of the esophagotomy wound and the other end out of the gastrotomy wound. A large dilating bougie is then passed from the stomach into the esophagus and pushed as forcibly as is safe into the lumen of the stricture. The string is used as a string- or bow-saw, and the stricture is divided, the dilating bougie being pushed firmly upward while the saw is being used. If this is not done the saw will not cut. Only stretched tissue will be divided. When the stricture has been divided the silk is withdrawn, full-sized bougies are passed, a temporary gastrostomy is usually made, and the wound or wounds are sutured.

An operation devised by A. J. Ochsner is thus described by Mayo[1]: "The anterior wall of the stomach is drawn out of a left oblique incision through the abdominal coverings; a small opening is made into the stomach sufficient in size to introduce the finger. A whalebone probe, to the tip of which a silk string guide has been tied, is now passed through the esophagus either from above or retrograde, as in the Abbe method. With this guide a loop of silk is drawn out of the gastric incision in such manner as to leave the guide as a third string. Into this loop a small soft-rubber drainage-tube 3 feet or more in length is caught in the middle by traction on the ends of the doubled thread through the mouth; this loop of rubber tube is drawn through the stomach and made to engage in the stricture.

"The greater the amount of traction, the smaller the stretched rubber tube, until it is sufficiently reduced in size to enter the stenosed portion; by alternating the direction of the pull the tube is drawn out by its free ends and in by the silk loop. Increasing sizes of tubes can be employed, and if necessary the third string can be used as a string-saw, after the Abbe plan of procedure." In a very severe case of stenosis gastrostomy is performed to keep the patient from starving. In a case of fibrous stenosis in charge of the author it was found impossible to insert any instrument from above or from below. Gastrostomy was performed by Kader's method. The patient was fed through the artificial opening and the esophagus was thus put at rest. Two weeks after the operation it became possible to pass a bougie from the mouth. The gullet was gradually dilated to its normal caliber and the gastrostomy wound was closed. This case demonstrates that a stricture of the esophagus, like a stricture of the urethra, may become temporarily impassable from inflammation, edema, and spasm; but, after the part is put at rest, may again permit the passage of an instrument.

In some cases of incurable stricture *cervical esophagostomy* is performed below the stenosis, and the patient is fed permanently through the opening. The operation is performed like esophagotomy, except that the mucous membrane is sutured to the skin.

**Carcinoma of the Esophagus.**—Cancer causes obstruction of the esophagus. It arises in those beyond middle life, and is far more common in men than in women. The disease may begin at any portion of the gullet, but is least often met with in the central portion (Maylard, Butlin). Epithelioma is the usual form, but scirrhus or encephaloid may occur. Cancer soon ulcerates, involves adjacent parts, and affects the deep cervical and posterior mediastinal glands. In at least 75 per cent. of cases of chronic obstruction of the esophagus cancer is the cause.

*Symptoms of Cancerous Stenosis.*—The patient is over forty years of age, is usually a male, and presents the same difficulty of swallowing met with in cicatricial stenosis. Regurgitation is common. The regurgitated matter is alkaline and is apt to contain blood. There is generally decided pain and very rapid and great emaciation occurs. The seat of obstruction may be located by the very gentle use of a soft or semisolid bougie, but it is wiser to use no bougie

[1] "Jour. Amer. Med. Assoc.," July 29, 1899.

and to rely on the *x*-rays and the esophagoscope. In a very recent case diagnosis is possible only by esophagoscopy. The stomach is the seat of pain; the mouth is dry, and there is often great thirst. As the disease infiltrates, the involvement of adjacent regions produces other symptoms. Dyspnea may result from tracheal pressure. Pleuritis, pericarditis, or pneumonia may arise. There may be paralysis of the sympathetic or recurrent laryngeal nerves.

In suspected cases of cancer never try to pass unguided bougies or sounds through the constriction. In a cancer case dilating instruments are weapons rather than tools. If a bougie is used to locate the constriction it will probably start bleeding and be bloody when withdrawn. A solid instrument might perforate the esophagus. If it does, death will follow. The *x*-rays should first be used and then the esophagoscope.

*Treatment.*—The disease is, of necessity, fatal, and treatment is only palliative. Complete excision of the cancer is scarcely feasible even in the cervical region. I know of but one successful resection of the thoracic esophagus, which was the case reported by Torek. At present the justifiability of such an operation has not yet been conclusively demonstrated. The patient should be put upon a soft, bland diet, small quantities being given frequently. When trouble is experienced in swallowing the bland and soft food, pass a soft bougie every third or fourth day. When the patient becomes entirely unable to swallow soft food, we may insert a Symonds tube (see Fig. 592) or do an esophagostomy (if this can be performed below the stricture), or perform gastrostomy. In every doubtful case of esophageal stricture give a course of iodid of potassium before performing any operation.

**Spasmodic Stricture of the Esophagus** (*Esophagismus; Hysterical Stricture*).—By this term is meant a spasm of the circular muscular fibers of the gullet, which is most common near the larynx or the cardia. This condition not unusually arises in a hysterical individual, in which case it will be associated with the stigmata of hysteria, especially globus hystericus. In some cases evidences of hysteria are wanting, although the patient is neurotic and ill-nourished, the condition being due to a reflex irritation. A spasm of the muscular fibers of the esophagus may be clonic or may be tonic. A clonic spasm may arise during vomiting or from some reflex cause; it may affect one part of the tube for a time and then shift to another, or may develop only in one particular region. Globus is a spasm which moves upward. Tonic spasm is in one fixed place. Most reflex spasms are tonic and result from cancer of the liver, cancer of the stomach, tonsillitis, glossitis, pharyngitis, or inflammation of the epiglottis (A. L. Benedict, in "Am. Jour. Med. Sci.," Aug., 1904). It occasionally occurs in tetanus, sometimes in epilepsy. Spasmodic stricture may also arise during pregnancy and as a result of laryngeal ulceration. I have seen several instances due to cancer of the stomach. In 1 of these cases the esophageal spasm entirely disappeared after the performance of pylorectomy.

*Symptoms of Spasmodic Stenosis.*—It arises suddenly in a hysterical or neurotic individual. It may last for a time and suddenly pass away, or may persist for a considerable time. The difficulty in swallowing is irregular, rarely interfering seriously with nourishment. Usually fluids are taken more easily than solids, but sometimes solids are taken more readily than fluids.

There may be regurgitation; but in recent cases, if it occurs, it does so at once on swallowing food. Examination with a bougie detects the obstruction. If the bougie is held firmly against it, in most cases the spasm will, after a time, relax suddenly or gradually and let the instrument pass. A medium-sized instrument or a large one may not pass until the patient has been anesthetized, but in every case a bougie can be passed after an anesthetic has been given.

*Treatment of Spasmodic Stenosis Above the Cardia.*—The systematic passage of bougies. Occasionally the passage of an instrument but once will cure a

case. The general health must be improved, and in persistent cases it may be necessary to use electricity within the esophagus, employ cold locally, and administer the bromids.

**Cardiospasm.**—When the cardiac sphincter contracts the condition is known as cardiospasm. It may or may not be associated with cancer, ulcer, or some other disease of the stomach, with gall-bladder disease or cancer of the liver. The attacks are periodic, with a variable time between them, but sooner or later diffuse dilatation occurs. Before dilatation the patient has periodic attacks of difficulty in swallowing, being perfectly well between the attacks. One of Plummer's cases had periodical attacks of dysphagia lasting from three to fourteen days, and during these attacks it was impossible to swallow either solids or liquids. In many cases the attacks are neither so severe nor prolonged. They usually begin suddenly while swallowing, but may begin at a time when no food is being taken. The attack is a feeling of choking or obstruction felt in the cardiac region and the back. Soon after these attacks originate the patient begins to suffer very soon after eating from regurgitation into the mouth. As the esophagus dilates the regurgitation is postponed longer and longer after eating, until finally the gullet is never completely empty (Plummer, "Northwestern Lancet," Oct. 1, 1906). The matter which is regurgitated is not sour, because it contains no gastric elements. Plummer points out that a stomach-tube cannot be passed, but a large-sized sound can be passed easily. The fact that a large sound can be passed proves that there is no organic stricture. When external causes of pressure are excluded the diagnosis rests between diverticulum and dilatation of the esophagus (Plummer, Ibid.). The *x*-rays aid in diagnosis. Plummer gives methods in detail (Ibid.).

*Treatment.*—Russell's plan is to dilate the cardia with a silk-covered balloon of rubber. Plummer describes how to construct it ("Collected Papers of the Staff of St. Mary's Hospital, Mayo Clinic, 1905–1909"). The distention is to be gradual, and at once suspended if there is violent pain indicating laceration. Mikulicz and others have performed gastrotomy and dilated the cardia. Willy Meyer ("Am. Jour. Surg.," June, 1912) has opened the thorax under positive pressure, plicated the dilated portion of the esophagus into two longitudinal double folds, and separated the pneumogastric nerves, tearing off the minute esophageal branches (*esophagoplication* and *vagolysis*). This patient was cured. This operation was attempted on 2 other patients. One was improved. The other turned out to have organic stricture. In another case Wendel performed cardioplasty in the same manner as pyloroplasty is done. He did it by the abdominal route. Meyer suggests that it may be done transthoracically.

**Diverticula of the Esophagus.**—Rokitansky in 1849 described traction diverticula and pressure diverticula. Maylard tells us that these pouches may be due to one of four causes: they may be congenital; may be due to stricture; may be caused by pressure from within upon a weak spot of the wall; may be due to traction from without by the healing and contraction of an area of inflammation. To these another cause should be added, muscular weakness resulting in dilatation. The usual situation for such a pouch is on the posterior wall of the gullet on a level with the cricoid cartilage. At this point there is a space devoid or nearly devoid of muscle, called the *Lannier-Hackerman area* (Charles H. Mayo, "Jour. Am. Med. Assoc.," July 22, 1912). As the pouch enlarges the fundus comes toward the side, usually the left side. Pouches are rare in the thoracic esophagus.

*Symptoms.*—In spite of the statement that a diverticulum may be congenital, we encounter the condition clinically only in adults. The first symptom is difficulty in swallowing, like that in cancer. There may be cough from nerve irritation and dyspnea from pressure on the trachea (Chas. H. Mayo, "Annals of Surgery," June, 1910). Regurgitation may occur, the regurgitated matter

being free from gastric juice. As the opening of the sac is usually a prolongation of the esophageal canal a sound or bougie tends to enter the sac. When the diverticulum is in the neck a lump forms during deglutition, and this lump may be obliterated by pressure. Food will pass into the stomach only when the diverticulum is full. A bougie can seldom be passed unless the pouch is full of food, at which time it may pass or may not. Sometimes it enters the pouch. This striking symptom, the variability in the passage of the bougie, is evidence suggesting the diagnosis of intrathoracic diverticulum. By listening through a stethoscope fluid may be heard to pass into the pouch. The diverticulum causes obstruction. "The depth of the obstruction can be measured with a stomach-tube, bougie, or acorn probe, but, as a matter of fact, these procedures do not differentiate between diverticula and strictures which are pervious to liquids and yet impassable to sounds" (Charles H. Mayo, "Annals of Surgery," June, 1910). After a patient swallows an emulsion of bismuth or food mixed with a salt of bismuth a diverticulum may be skiagraphed. When a bougie is passed as far as it will go a skiagraph should be taken with the bougie in position. The plate may show that the instrument is so much deviated to the side that it must be in a pouch. If a fine gold chain is swallowed it may fill up the pouch, and if it does, a skiagraph will indicate the diverticulum. The opening of the pouch may be seen by means of an esophagoscope. Plummer's test is valuable. He has the patient swallow a string guide, as described on page 935. The thread is passed through the eye of an olive tip set on a whalebone stem. The instrument is passed onward until it meets an obstruction. "Should the trouble be due to stricture the tip will not change its level when the thread is tightened, but if there is a diverticulum the probe will be elevated to the level of the opening in the lower esophagus, proving at once the existence of a pocket and also its depth by the amount of elevation of the probe upon tightening the thread" (Charles H. Mayo, Ibid.).

*Treatment.*—In very early cases dilatation may cure. In advanced cases in the cervical esophagus we must perform extirpation and suture, as done by von Bergmann, Hearn, the author, and others. For five days after operation no food is given by the mouth. No attempt should be made to extirpate a pouch in the thoracic esophagus. In such a case gastrostomy may be necessary.

**Injuries of the Esophagus from Within.**—Injuries of the internal surface are more common than injuries from without. Burns and scalds are among these injuries. Wounds may be inflicted by foreign bodies. Injuries of the gullet cause pain on swallowing, and a wound induces bleeding, the blood being both coughed up and vomited. A severe wound may involve a large vessel and cause violent or even fatal hemorrhage. If the bronchus or trachea is involved, there will be "cough and expectoration of blood, mucus, and food" (Maylard). The pleural or pericardiac sacs may be perforated.

*Treatment.*—Feed only by the rectum. Give morphin hypodermatically. Do not feed by the mouth for ten days, and even then give only fluid food and jelly. Symptoms are met as they arise. After burns by caustic, administer the antidote; give large drafts of water and wash out the stomach. From two to four weeks after a caustic has been swallowed and after a burn or scald the use of sounds should be begun (provided there is no raw surface), and sounding should be persisted in for a considerable time to prevent contraction.

**Injuries of the Esophagus from Without, Other Structures Not Being Seriously Involved.**—Such injuries are rare. Esophageal injuries, as a rule, are associated with serious damage to adjacent structures. Injuries may be due to stabs or to bullets. Besides the obvious external signs of the injury there will be difficulty in swallowing, cough, bloody expectoration, or vomiting; and mucus or the contents of the stomach may run out of the wound.

*Treatment.*—Suture the wound, and feed by the rectum for ten days.

**Foreign Bodies Lodged in the Esophagus.**—These accidents occur especially in children and lunatics, and women are more apt to suffer from them than men. A list of various bodies which have been swallowed will be found in Poulet's elaborate treatise. There are three regions where a foreign body is especially apt to lodge—viz., opposite the cricoid cartilage, at the level of the diaphragm, and at the point where the left bronchus crosses the gullet. Small and sharp bodies may lodge anywhere.

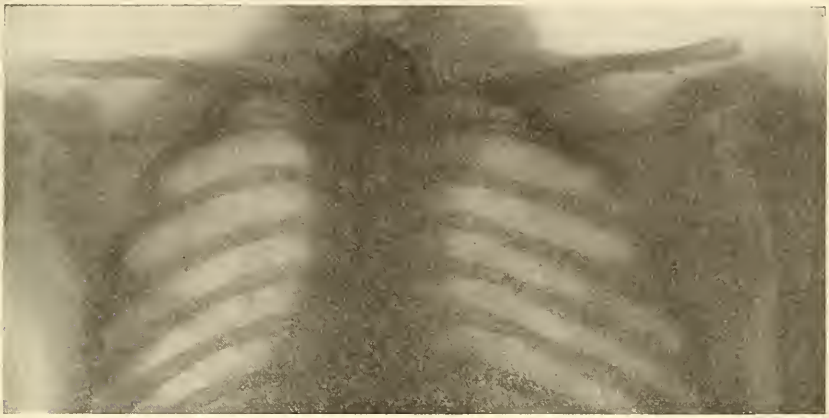


Fig. 595.—Author's case of whistle in esophagus, removed by external esophagotomy.

The *symptoms* are variable; if the body is large, there will be pain and difficulty in swallowing, and, in many cases, dyspnea from pressure upon the trachea or bronchus. Occasionally the dyspnea is such a prominent feature that it misleads the physician into the belief that the foreign body is lodged in the air-passages. Death may actually result from asphyxia. In some other cases the symptoms are very slight. If the body is sharp, there will be hemorrhage and severe pain. The blood may be hawked up, or may

Fig. 596.—Author's case of jackstone in esophagus, removed by external esophagotomy.

be swallowed and vomited. In rare cases a patient grows accustomed to a foreign body and ceases to notice it; but, more often, the foreign body produces inflammation. It may even ulcerate into the wind-pipe, the pleura, the pericardium, or the aorta. In many cases of impaction a patient makes violent efforts to hawk up the foreign body and so produces aphonia. There may be violent retching. Even after a foreign body has been removed by swallowing, by vomiting, or by surgical extraction a sensation is apt to remain as

if the body were still lodged. The diagnosis is made by the history, the detection of the body by external manipulation, by feeling it with an esophageal bougie, by esophagoscopy, and, if bone or metal, seeing it with the fluoroscope or obtaining a skiagraph.

*Treatment.*—The surgeon should learn, if possible, the size, shape, weight, and nature of the foreign body, and should locate its point of impaction. The exact point of lodgment of bone or a metallic body is determined by the *x*-rays. An anesthetic is given before manipulating a child, a nervous woman, or a lunatic, and is *sometimes* necessary for a man. If the foreign body is soft, external manipulation may succeed in altering its shape, so that it may be swallowed or ejected. If the foreign body is hard, external manipulation may shift its position. It is usually impossible to reach the foreign body through the mouth by means of the fingers (when the body is in the rear of the pharynx it may be pulled forward or pushed down). Sharp foreign bodies may be entangled and carried down when the patient eats mush, bread, or boiled potatoes. The administration of emetics is an old plan which occasionally succeeds, but which is too unsafe to be employed. The esophagoscope is of immense value in many cases. By its aid foreign bodies may frequently be removed which without it could only be removed by the performance of a grave operation (see page 943). Nevertheless I believe, with E. Fletcher Ingalls, that the esophagoscope "cannot entirely supplant the older methods" ("Amer. Jour. of Surg.," Jan., 1912). The instrument is not always at hand, and to use it successfully and safely one must be well trained in its management. Maylard says that when a mass of food is impacted it is occasionally possible to soften and disintegrate the mass by administering a mixture containing pepsin. The bristle probang (Fig. 597, C) is a very useful instrument for the removal of fish-bones, pins, and other small objects. It may be used to push a body downward into the stomach, or to catch the body and pull it up. When this instrument is withdrawn it opens like an umbrella. It has been shown that in an adult the cardiac opening is from 14 to 16 inches from the incisor teeth, a point to be remembered in deciding whether to push down or pull up the impacted article. Esophageal forceps (Fig. 597 A, B) are valuable in some cases. The coin-catcher (Fig. 597, D) is a useful instrument. Créquy's plan of removal is to take a tangled mass of threads, tie a stout piece of string about the middle of it, coat it with sugar, and have the patient swallow it. It may pass the foreign body; if it does so, on withdrawal it may entangle the object and extract it. To remove a fish-hook with line attached the following plan may prove successful; stick the line which projects from the mouth into a metal catheter, carry the catheter down to the hook, and push the hook out. It is not proper to allow a foreign body to remain in the

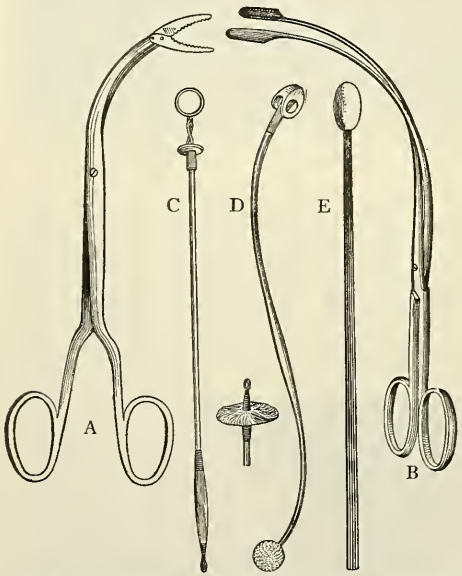



Fig. 597.—Esophageal instruments: A, B, Forceps; C, Gross's bristle probang; D, coin-catcher; E, bulbous esophageal sound.

esophagus until it causes ulceration. Neither is it proper to make prolonged efforts to extract it through the mouth. Such efforts may do great harm, and if one careful and consistent effort fails, and the esophagoscope is not suited to the case, fails, or is unobtainable, or if there is no skilled man to use it, an operation should be performed. If the body is lodged anywhere above the lower third of the esophagus, *external esophagotomy* is performed, and usually on the left side. Through this wound the foreign body is extracted. The cut is made on the left side, between the trachea and larynx in front and the carotid sheath behind, the center of the incision being opposite the cricoid cartilage. After the foreign body has been extracted the mucous membrane is sutured with chromicized catgut, and the superficial structures are closed with silkworm-gut after a drainage-tube has been inserted. The patient is fed by the rectum for eight or ten days. When a foreign body is lodged in the lower portion of the tube the stomach is opened and the body extracted by this route. In White's case of jackstone in the gullet gastrotomy was performed. A string was tied about some rolls of gauze, the string was passed by means of a whalebone from the stomach into the mouth, and the body was entangled and drawn out.

**Surgical Invasion of the Mediastinum.**—The posterior mediastinum has been entered in order to remove a foreign body from the bronchus and to extract a set of false teeth wedged in the esophagus. The same method can be followed to reach suppurative processes in the mediastinum, abscesses of the lung otherwise inaccessible, and diverticula and carcinomata of the lower end of the gullet. Nassilov resects ribs close to the spine. The portion of the esophagus above the aortic arch can be reached after partial resection of the third, fourth, fifth, and sixth ribs of the left side. The inferior portion of the esophagus can be reached after resecting portions of the lower third or fourth ribs on the right side (Binnie's "Operative Surgery"). Willy Meyer has made four heroic attempts to resect cancers of the esophagus. All the patients perished. He does it under positive pressure ("Surg., Gynec., and Obstet.," Dec., 1912). Out of 35 known operations 1 recovered and 1 lived two weeks after operation. The anterior mediastinum may be entered to remove a bullet, to drain an abscess, to reach a wound of the heart or lung, and to explore for the cause of symptoms. I explored the anterior mediastinum after rib resection, found a bullet embedded in the aorta, and allowed it to remain. The patient recovered. M. H. Milton[1] splits the sternum and separates the two pieces.

Invasion of the mediastinum is much safer if the operation is performed in the Sauerbruch chamber or with the aid of positive pressure within the bronchi. Either of these plans will prevent pulmonary collapse if the pleura is opened.

## XXVIII. DISEASES AND INJURIES OF THE ABDOMEN

**Diagnosis of Intra-abdominal Emergencies.**—The exact diagnosis is always difficult and in many cases is impossible. What a surgeon must try to determine, and what he usually can determine, is whether he is dealing with a trivial and temporary derangement for the relief of which an operation is entirely unnecessary, or whether he is confronted by a grave calamity which imperatively demands immediate surgical aid. We can decide that a calamity exists, but the exact nature of the lesion is often doubtful until operation is performed. Every operation in such a case is exploratory. Before the diagnosis of a calamity is made morphin should not be given, because it allays the pain, relieves the anxiety, causes the disappearance of rigidity, lowers the pulse, abates mental shock, and hence veils the real situation, so that the most discerning surgeon will probably be misled. If

[1] "Lancet," March 27, 1897.

shock is profound, diagnosis is usually impossible, unless shock is due to hemorrhage, and immediate operation during shock is not to be thought of except for a perforation or to arrest bleeding. If excessive and continued hemorrhage is suspected, immediate operation is indicated. If it is not suspected, the patient should be covered with blankets and surrounded with hot-water bags, atropin should be given hypodermatically, and hot salt solution should be administered by rectum, subcutaneously, or intravenously. When the patient reacts, and he usually will react, an attempt is made to make a diagnosis. If a patient must be moved to a hospital it is perfectly proper to give a single hypodermatic injection of morphin ($\frac{1}{3}$ gr.) after the effort has been made to diagnosticate the condition. The danger of deluding the surgeon is past, and the drug abates pain, lessens peristalsis, relieves mental anxiety, and is distinctly beneficial. Before the morphin was given the surgeon came to a conclusion as to the necessity for operation. After the morphin has been given, if an operation is indicated, it is performed as promptly as circumstances admit. Whenever it is consistent with safety, the patient ought to be removed to a hospital for operation.

**Foreign Bodies in the Abdomen.**—Now and then a sponge, a pad, or an instrument is left in the abdominal cavity during an operation—left, not because of the surgeon's carelessness, but in spite of the most painstaking precautions. Instruments and sponges are counted before and after the operation. A miscount means calamity. The surgeon does not and cannot make the count himself. He has not time. He is doing a more difficult thing which he is trained to do and which the patient expects him to do, that is, operating, closing the wound, and dressing it. The surgeon must delegate this duty, and he delegates it to assistants or nurses, who do it with as much certainty of accuracy as he could have were he to do it. At least *two* people count sponges, pads, and instruments at least *twice* before and *twice* after the operation. If the count is short, and the missing instrument or material is not discovered on the floor, in a bucket, on or under the table, the abdomen must be opened and the lost object sought for and found.

A great safeguard is to use no small sponge or pad unless it has a bit of tape sewed to it. The tapes stick out of the wound and on each one a forceps is clamped. The safest way of all is to use rolls of gauze (Halsted's packs). To do so means a long broad end sticking out of the wound so that it cannot be lost in the belly.

If a septic body of any sort is left in the abdomen it at once produces acute disturbances.

If an aseptic gauze pad or sponge is left it may become encapsuled and cause no trouble for months or years. Sooner or later it will be apt to cause trouble or even death. The sufferer may become a chronic invalid. The object may make its way into the intestine or into the bladder. It may even pass out through the rectum. Usually a piece of retained aseptic gauze or an aseptic instrument produces abdominal pain and a tendency to intestinal obstruction. Sooner or later an abscess forms and the symptoms of an acute septic condition arise.

Schachner says the mortality in reported cases is about 50 per cent. Many fatal cases have not been reported. If we suspect the presence of a metal instrument the *x*-rays will show it.

**Treatment.**—Laparotomy (which is often exploratory) and removal of the offending material.

**Simple Contusion of the Abdominal Wall Without Injury of Viscera.**—In some cases of contusion of the abdominal wall only the parietes are damaged; in other cases the viscera or the abdominal tissues are injured. Contusion may involve the skin alone, or may involve the skin, muscles, and peritoneum. In *simple contusion* there is considerable shock if the injury

is severe. There is pain, increased by respiration, motion, pressure, and attempts at urination or defecation. When tenderness appears some days after the accident there is usually deep-seated injury. Extensive ecchymosis may appear. Even after a severe contusing force has been applied there may be no discoloration, and it may happen that after a slight force there is much discoloration. There is great ecchymosis in anemic persons, victims of hemiplegia, obese individuals, opium-eaters, and drunkards. In severe cases the tissues are pulpified and sloughing inevitably ensues. Abscess occasionally follows contusion. The prognosis after abdominal contusion is always uncertain.

**Treatment of Simple Contusion.**—In treating simple contusion place the patient at rest in a supine position, with the thighs flexed over a pillow. Obtain reaction from the shock. If pain is severe, and we are certain there is no visceral injury and no internal hemorrhage, give morphin. After shock has passed off it is advisable to place an ice-bag over the seat of injury. If much blood is extravasated into the abdominal wall, aspirate and apply a binder. After twenty-four hours apply local heat by means of the hot-water bag, employ an ointment of ichthyol, and move the bowels, if necessary, by salines. Regard every contusion as serious, and watch carefully for the development of signs of internal hemorrhage or visceral injury.

**Muscular Rupture from Contusion.**—In this injury there are severe shock, and pain (increased by respiration and movement). Separation between the fibers of the muscle is distinct at first, but it is soon masked by effusion of blood. Such injuries may cause death or may lead to hernia. The rectus is the muscle most apt to rupture. The rupture is due to sudden contraction rather than to the direct effect of a blow.

The *treatment* is the same as for simple contusion. Always apply a binder. If a hernia exists it is returned and a compress is applied over the opening through which it emerged. Later operation is performed. If strangulation occurs, operate at once.

**Injuries With Damage to the Peritoneum or the Viscera.—Rupture of the Peritoneum.**—The peritoneum may be involved in an abdominal contusion. It may rupture even when there is no visceral injury or muscular contusion. The uterine peritoneum, the parietal peritoneum, the visceral peritoneum, or the mesentery may rupture. Rupture of the peritoneum causes intra-abdominal hemorrhage.

The *treatment* consists in opening the abdomen, arresting the hemorrhage, and bringing about reaction.

An injury to the peritoneum creates a point of least resistance, and at such a point peritonitis may develop. The peritonitis is usually local, but may become general. After any severe intra-abdominal injury the symptoms of *peritoneal shock* appear (*peritonism*), and the patient may rapidly die. In the condition of peritoneal shock the temperature is subnormal; the extremities are cold; the face is pallid and sunken; the pulse is small, weak, and very frequent; the respiration is shallow and sighing; there is great thirst; the patient is restless and turns uneasily, and there is rigidity and distention. Vomiting almost always occurs. In some cases there is regurgitation rather than vomiting. The abdomen is the seat of a violent, persistent pain. The patient is fearful of impending death. As the symptoms develop in a grave case they will point to one of two conditions—hemorrhage or peritonitis.

In *intra-abdominal hemorrhage* the subnormal temperature and other evidences of shock persist. Vomiting ceases, but nausea exists. The patient is uncontrollably restless and tosses about in bed. The thirst is great. The abdomen is rarely rigid. Fainting-spells occur. Blood examination shows a marked fall in the percentage of hemoglobin. Percussion demonstrates the

existence of an effusion which alters its position as the patient's position is altered, and which gradually increases in amount. Dulness is first met with in the loins. Digital examination of the rectum or vagina may aid in diagnosis, because in hemorrhage blood gathers in the rectovesical pouch. If peritonitis develops, the vomiting is aggravated, the pain is intensified, and the abdomen grows rigid and distended.

**Rupture of the Stomach Without External Wound.**—The usual cause of rupture is a violent blow, although the accident may happen while washing out the stomach. Rupture is more apt to occur when the stomach is distended with food than when it is empty. The rupture may be partial, the peritoneal coat not being torn. The rupture may be complete. Either the anterior or the posterior wall may suffer. The region of the pylorus is most apt to be lacerated. The symptoms of rupture are collapse, severe pain over the entire abdomen, great thirst, excessive tenderness, especially over the epigastric region, occasionally vomiting, the vomited matter being usually, but not invariably, bloody; tympanitic distention and muscular rigidity coming on after a few hours. Austin Flint pointed out years ago that after complete rupture or perforation gas may enter the abdominal cavity and cause the diminution or disappearance of liver-dulness, but the area of liver-dulness can be lessened by great intestinal distention, and I have seen cases of perforation of the stomach and intestine in which it was not lessened at all. (See article on Perforating Ulcer of the Stomach.) After *incomplete* rupture local peritonitis is frequent; in *complete* rupture the escape of stomach contents into the peritoneal cavity causes general peritonitis. The contents of the stomach are not so liable to escape after rupture of that viscus as are the contents of the intestine after rupture of the gut, because of the thickness of the stomach wall and the tendency of the mucous membrane to evert and block the opening. Perforations of the anterior wall are most apt to lead to extravasation and general peritonitis. Posterior laceration may cause subphrenic abscess. To diagnosticate between complete and incomplete rupture, Senn endeavors to distend the viscus with hydrogen gas; in incomplete rupture the contour of the dilated stomach can be made out upon the surface; in complete rupture the viscus cannot be distended, and the gas passes into the peritoneal cavity, producing the physical signs of tympanites. This maneuver is open to the objection that it may increase extravasation in a complete rupture.

The **treatment** for complete rupture is immediate operation. Treatment for shock is at once instituted and an intravenous infusion of salt solution is given before or during operation. In doubtful cases endeavor to bring about reaction and explore. Open the abdomen. Note if gas emerges from the wound or if stomach fluid appears. Search for the rupture in the same manner as we would search for the opening of a perforated ulcer. When the rupture is discovered, flush out the stomach and the peritoneal cavity with hot salt solution; sew up the stomach wound with a double row of silk sutures, the first row being buried and including the muscular coat and mucous coat, the second row being Halsted sutures; drain the abdomen; close the wound in the parietes; place the patient in Fowler's position; let salt water at low pressure flow continuously into the rectum; feed by the rectum for four days, and then begin the administration of a very little food by the mouth. In incomplete rupture the danger is perforation. The patient is put to bed, and after reaction has taken place is fed by the rectum for several days. Cases of complete rupture not operated upon occasionally recover, adhesions arising and perigastric suppuration taking place. The mortality is extremely large. In 1896 Petry collected 23 cases in which operation was not performed. The mortality was 59 per cent. This mortality is not so large as one would

anticipate. It is quite possible that some of the cases were not genuine instances of rupture. Many fatal cases have not been reported. Nevertheless, the lesion, for reasons previously stated, is not nearly so dangerous as rupture of the intestine. Another reason for the greater danger of intestinal rupture is that fecal matter is much more poisonous than the gastric contents. Laparotomy has lessened the mortality of rupture of the stomach. Petry and also Eisendrath mass together operations for rupture of the stomach and rupture of the intestine. Petry finds the group mortality to be 52.3 per cent., and Eisendrath finds it to be 52.5 per cent. Statistics referring to the stomach alone should show a much lower death-rate.

**Rupture of the Intestine Without External Wound.**—In the great majority of cases the damage is produced by direct violence. In some few cases the force is indirect (falls on the feet or buttocks, blows on the back or loin). The injury may result from *oscillation* or from *compression* (the younger Senn). The common cause is undoubtedly compression of the gut against the pelvis or vertebral column, but it is certain that a gut containing fluid may be ruptured purely by violent shaking or oscillation. If oscillation produces the damage, the rupture is on the portion of gut furthest from the mesentery; if compression is the cause, any part of the bowel may suffer. Rupture is most apt to occur if the belly is relaxed. It is predisposed to by adhesions, disease of the wall of the bowel, and irreducible hernia. Most ruptures are complete. In a very few cases the tear extends only through one or two of the coats and the rupture is incomplete. A contusion of the gut may be followed by rupture several days after the injury. A complete rupture usually permits leaking of feces, but in very rare cases a small opening is closely plugged by pouting mucous membrane. Leaking from a rupture may be delayed because intra-abdominal pressure may for a time keep the opening pressed against a section of sound gut (the younger Senn). The amount of damage to the belly wall does not convey any notion of the extent of visceral injury. The belly wall may be severely injured and the viscera escape. With only slight contusion of the wall there may be extensive visceral injury. Homer Gage[1] collected 85 cases; in 75 the injury was due to direct force, and in 32 of these the force was inflicted by the kick of a horse or of a man. In 1 of my cases it was due to the kick of a horse, in 1 to the kick of a man, and in 1 to a crush inflicted by a cart-wheel. The victims in the majority of reported cases were young men, probably because young men are most apt to be exposed to violence. In 78 collected cases (Gage) the situation of the injury was specified: The duodenum, 10; jejunum, 20; ileum, 42; large intestine, 6. Curtis found the large intestine injured in 4 cases out of 113, and Poland, in 5 cases out of 64. In many cases there is more than one tear, and sometimes many tears exist. Both the large and small intestines may suffer. Chavasse collected 106 cases in which the ileum or jejunum suffered, 19 in which the large intestine did, 7 in which the duodenum did, 7 in which both the large and small intestine were involved, and 1 case in which the rectum was ruptured (quoted by the younger Senn, in "Am. Jour. Med. Sciences," June, 1904). As Makins points out, the portion of gut most apt to be injured is a portion hanging low in the pelvis, because a loop in this situation is most easily squeezed against bone by a blow on the belly. The mesentery may be lacerated (it is in 7 per cent. of cases, according to Gage; in 16 per cent., according to Curtis). The symptoms of rupture of the intestine are profound shock, tympanites, abdominal pain, and rigidity, rapidly followed by peritonitis if the patient survives. In some cases pain is referred to the back. Vomiting comes on soon after the accident, the vomited matter being possibly at first bloody and later ster-

[1] "Annals of Surgery," March, 1902.

coraceous. The respiration is thoracic, the tongue is dry, and great thirst exists. The pulse, which may be slow at first, soon becomes small, rapid, and of high tension. Blood in the stools rarely appears early enough to be of diagnostic value, and there may be diarrhea or constipation. The respiration is costal. Dyspnea exists. There may be no marked symptoms for an hour or two or for many hours. Cases are on record of people with ruptured intestine returning to work perhaps for hours. Holland's patient had no symptoms for twenty-four hours, although the jejunum was ruptured. Poland's patient ruptured the duodenum, but walked one mile in spite of it. The escape of gas into the peritoneal cavity may cause the diminution or disappearance of liver dulness. After anesthetizing the patient, hydrogen gas insufflated into the rectum will come from the mouth if there is no perforation in the stomach or the intestine; if a perforation exists, tympanites is much increased and the area of liver dulness may disappear. To apply *rectal insufflation of hydrogen*, generate the gas in a bottle by means of zinc and sulphuric acid, catch the gas in a large rubber bag, and attach the tube from the gas reservoir to a tip which is inserted in the rectum. Give the patient ether to relax the abdominal muscles, direct an assistant to press the anal margins against the rectal tip, and when the patient is unconscious turn on the stopcock and press upon the reservoir (the elder Senn).

It has been suggested that ether vapor, mixed with air, can be used instead of hydrogen gas.[1] In this method a little ether is poured into the bottle of an aspirator, the valves are opened, one tube is carried into the rectum, the other tube is attached to a bicycle pump, and by working the pump the ether vapor is driven into the bowel. If there is perforation, tympanites is notably increased. Most surgeons regard the rectal insufflation test as unsatisfactory and often dangerous. Personally, I am not inclined to use it. Its application requires considerable time; it must, of necessity, increase fecal extravasation. If we operate after insufflation the gaseous distention is an embarrassment to the surgeon; as Le Conte[2] says, it "so distends the intestines that it may be impossible to return them to the abdominal cavity until they have been emptied of gas."

**Treatment for Rupture of Intestine.**—After an abdominal injury, if symptoms point to dangerous hemorrhage, and in any case in which the patient does not seem to be reacting, but is rather getting worse, operate at once. If in doubt as to whether or not rupture exists, make every endeavor to bring about reaction and explore. Reaction is brought about as previously directed. Asepticize and anesthetize. Perform a laparotomy, making the incision in the middle line and below the umbilicus; observe if gas escapes when the peritoneum is opened or if fecal material or an inflammatory exudate flows out; check hemorrhage; start at a fixed point and conduct a careful search to find the rent. When the rent is found, it should be closed by Halsted sutures if possible, but only a small rupture can be so treated. A large tear makes resection necessary. Because of the frequency of multiple lesions the surgeon must not be sure he has finished his work when he finds and closes one tear, but he must determine by careful search that no other tears exist. The surgeon notes if there is injury of the mesentery and if the circulation of any portion of the bowel is interfered with. If there is serious impairment of circulation in any part of the bowel wall, perform intestinal resection, followed by end-to-end approximation or lateral anastomosis. In some cases of rupture the patient is so severely shocked that it is impossible to do a resection with any hope of his living. In such a case stitch the ruptured portion of gut to the belly wall. The opening in the gut be-

[1] Emerson M. Sutton, of Geneva, in "Jour. Am. Med. Assoc.," July 23, 1898.
[2] "Jour. Am. Med. Sciences," Dec., 1901.

comes a fecal fistula, and if the patient survives, can be subsequently closed. The same procedure is proper if the bowel is distended and paralyzed. After closing the opening in the bowel or resecting, flush the abdominal cavity with hot saline solution, and wipe the peritoneal fossæ and the space between the liver and diaphragm with gauze. Finney eviscerates, wipes out the abdominal cavity, and wipes the intestines as he restores them. This is justifiable if the operation is done soon after the rupture, but not in later cases, in which the lymph has gathered on the bowel. Whatever method is used to cleanse the abdomen, remember that infectious material is apt to accumulate between the liver and diaphragm and in Douglas's pouch. Drainage is to be used. Suprapubic drainage is most advantageous. Place the patient semi-erect and employ continuous proctoclysis of normal salt solution as directed for peritonitis. The value of operation for intestinal rupture is conclusively demonstrated. Curtis collected 116 cases which occurred before 1887. Not a case was operated upon, and every patient died. Homer Gage collected 85 cases since 1887: 45 were not operated upon and every one died; 40 were operated upon and 17 recovered. Eisendrath collected 40 cases operated upon: 19 recovered and 21 died (52.5 per cent.). The mortality of cases not operated upon is, according to Eisendrath, at least 93 per cent. The sooner after the injury operation is performed, the greater the chance for success. The younger Senn points out that in operations done within four hours the mortality is 15.2 per cent.; in those done between five and eight hours it is 44.4 per cent.; in those done between nine and twelve hours it is 63.6 per cent., and in those done later it is 70 per cent.

**Identification of the Small Intestine and of the Large Intestine.**—"In abdominal operations it is frequently imperatively necessary that the large intestine be recognized with certainty or the small bowel be positively identified. The size of the tube will not always aid in this recognition, as a small intestine may be distended enormously, and a large intestine may be contracted to the size of a finger because of obstruction above. The longitudinal muscular fibers of the large bowel are accentuated in three portions; these accentuations constitute the three longitudinal bands which begin at the cecum and terminate at the end of the sigmoid flexure of the colon. Each band is composed of a number of shorter bands, the shortness of these constituent bands permitting the sacculation of the large intestine. Longitudinal bands and sacculation are not met with in the small gut, their presence or absence being a means of identification in many cases; but when the colon is much distended the bands cannot be seen distinctly and the sacculation disappears. From the large intestine only spring the appendices epiploicæ (small overgrowths of fat in pouches of peritoneum), but they are sometimes not well marked except upon the transverse colon, and when emaciation exists they may almost entirely disappear. The relatively fixed position of the large intestine and the free mobility of the small bowel are important points of distinction. The foregoing indicates that it is not always easy to distinguish between colon and small gut, and that, according to old rules, it may be often necessary to make large incisions, to see as well as feel, and to handle a large extent of the bowel. Any scrap of knowledge that will shorten an abdominal operation, that will permit of as certain work through a smaller incision, and that will diminish handling of intraperitoneal structures tends to increase the chances of recovery. For these reasons the writer suggests a method of bowel identification which rests upon the facts that each bowel has a posterior attachment, that the origin of the attachment differs according to the bowel it supports, that a single finger can detect the origin of the peritoneal support of any section of the bowel, and, this origin being known, the portion of the bowel it supports is with certainty deducible. In an exploratory operation, for instance,

the finger comes in contact with the bowel: to determine whether it is a large or a small bowel, note first if the structure is movable or is firmly fixed; next,

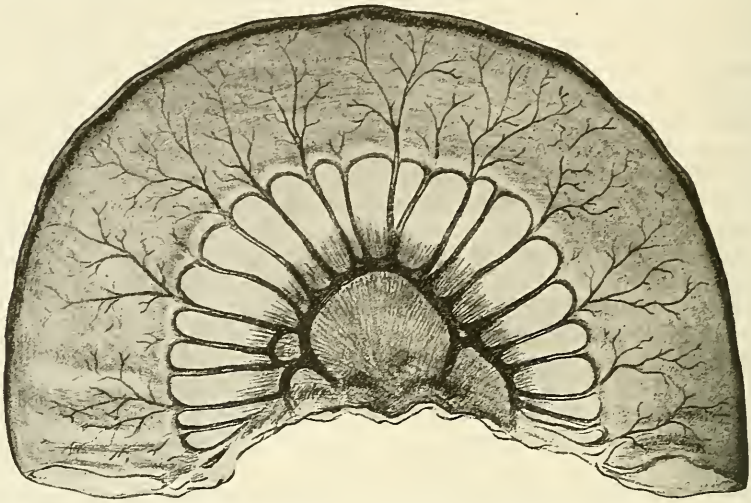


Fig. 598.—A loop of intestine, the middle of which is exactly 3 feet from the end of the duodenum. The gut is of large size. The mesenteric loops are primary, and the vasa recta large, long, and regular in distribution. The translucent spaces (lunettes) between the vessels are extensive. Below, the mesentery is streaked with fat. The veins, which had a distribution similar to the arteries, are for simplicity omitted from this and from the subsequent drawings. The subject from which the specimen was taken was a male of forty years, with rather less than the usual amount of fat. The entire length of the intestine was 23 feet (Monks).

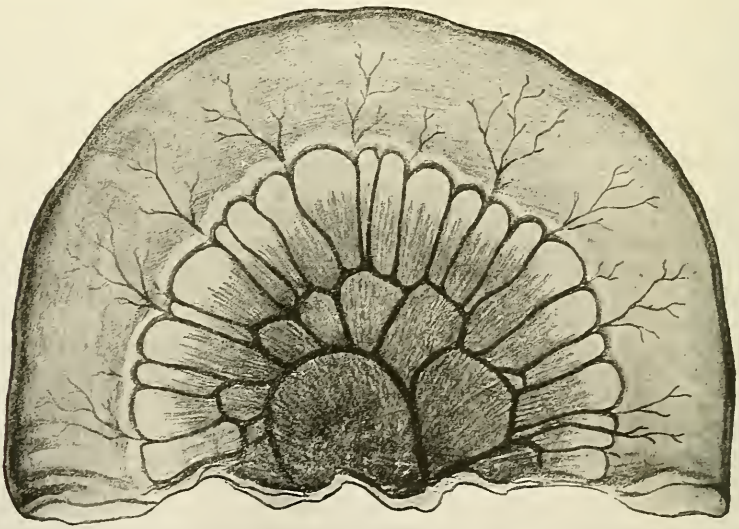


Fig. 599.—A loop of intestine at 6 feet. As compared with Fig. 598 the gut is somewhat smaller. The vascularity of the intestine and mesentery is less. Secondary loops are a prominent feature. The vasa recta are smaller. The lunettes are also present, but are not so large as in Fig. 598. The subject was a male of about thirty-five years, with an average amount of fat. The entire length of the intestine was 20 feet (Monks).

pass the finger over the bowel and let it find its way posteriorly. If dealing with a small bowel, the finger will reach the origin of the mesentery between the left side of the second lumbar vertebra and the right sacro-iliac joint; if dealing

with the large bowel, the finger will reach the origin of the mesocolon, or the point where the colon is fixed posteriorly and to the side."[1]

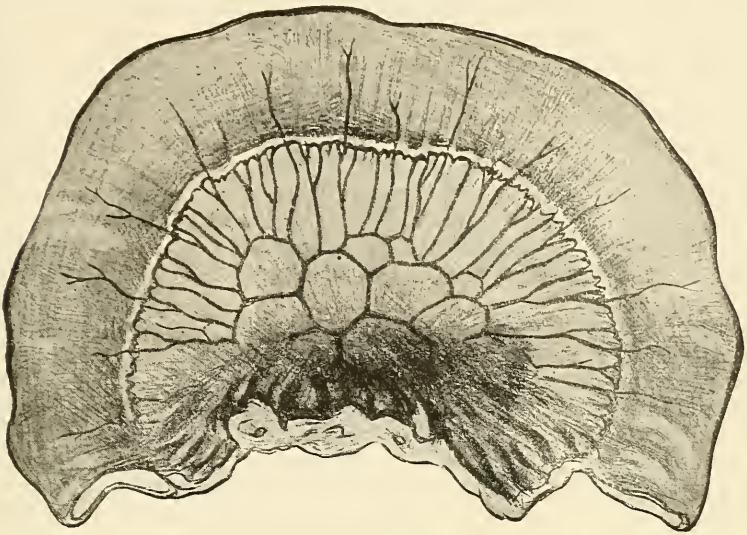


Fig. 600.—A loop of intestine at 12 feet. The vessels are smaller. The primary loops are lost in the fat, but secondary and even tertiary loops are visible. The vasa recta are shorter, more irregular, and branching. The specimen came from the same subject which furnished Figs. 598 and 599 (Monks).

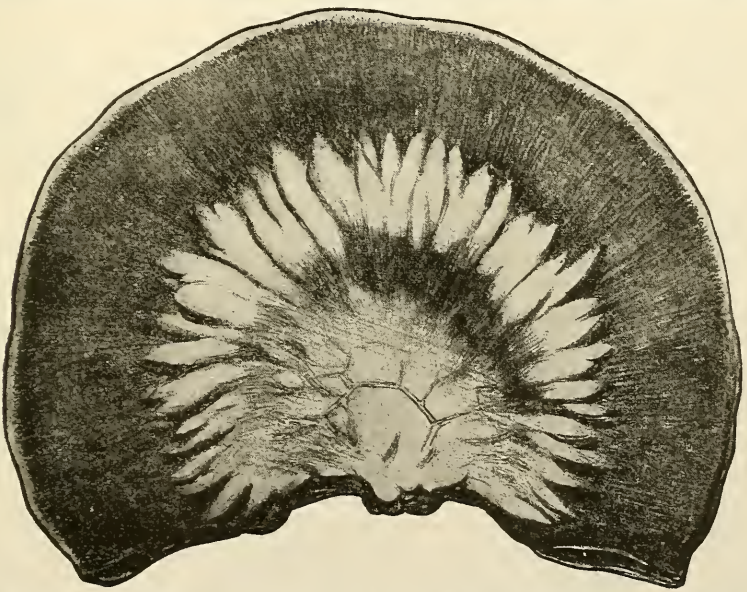


Fig. 601.—A loop of intestine at 20 feet. The gut appears to be thick and large. The mesentery is quite fat and opaque, and large and numerous fat tabs are present. The vessels, which are complicated, are seen with difficulty, and are represented by mere grooves in the fat. The subject was a stout woman, and the entire length of the gut was 21 feet (Monks).

**Location of a Loop of Small Intestine** (Figs. 598–601).—Monks points out a plan by which, in most instances, we can learn with approximate accuracy what portion of the small intestine we may have hold of ("Annals

[1] The author, in "Medical News," June 9, 1894.

of Surg.," Oct., 1903). He learns first by observation of the mesenteric vessels. Opposite the upper portion of the bowel there are primary vascular loops only with perhaps an occasional small secondary loop. As we descend "secondary loops become more numerous, larger, and approach nearer to the bowel than the primary loops in the upper part," and about the fourth foot these secondary loops first become a "prominent feature." As we descend primary loops become smaller, secondary loops become more numerous and nearer the bowel, and possibly tertiary loops appear. Opposite the lower portion of the ileum the loops are not definite in arrangement, but are simply a network. Monks points out that opposite the upper bowel the vasa recta, when put gently on the stretch, "are straight, large, and regular, and rarely give off branches to the mesentery," and are about 5 cm. long. In the lower third they are usually less than 1 cm. long, are smaller, are not quite so straight, are not so regular, and give off numerous mesenteric branches. Monks further shows that fat impairs the translucency of the mesentery. The thinnest mesentery is that connected with the upper gut. As we descend the mesentery becomes thicker and thicker because of fibrous tissue, unstriated muscle, and fat. Translucency varies greatly. If a loop of upper intestine is raised against the light, one notices close to the gut and between the vasa recta transparent lunettes. The lunettes become smaller and fatty as we descend, and disappear at the eighth foot. In an incision in the median line, if the loop of intestine is pulled downward, we can determine if "the line of resistance from above is from the median line of the body or from the left or right of it." This resistance of the mesentery indicates to which point the loop is attached, and hence what portion of bowel the loop comprises. I have used these observations of Monks repeatedly to great advantage.

**Rupture of the Liver.**—(See page 1032.)

**Rupture of the gall-bladder and the bile-ducts** (see page 1032) is most apt to happen from injury when gall-stones exist. Peritonitis, general or local, is almost certain to follow such a rupture. Besides those symptoms common to all severe abdominal injuries there may be intense jaundice.

**Treatment.**—Suture the laceration or make a biliary fistula.

**Rupture of the Pancreas.**—(See page 1054.)

**Rupture of the Spleen.**—(See page 1063.)

**Rupture of Mesenteric Arteries.**—The symptoms are those of hemorrhage. The superior mesenteric artery, the inferior mesenteric artery, or branches of either or both may be damaged. If branches of the superior mesenteric artery are divided near the bowel, gangrene of the bowel will result, but wound of a branch far from the intestine does not cause gangrene. The branches near the gut are terminal arteries, hence the gangrene. The branches of the artery divide and form arcades arranged concentrically and the terminal arteries come from the peripheral arcades. In wounds of the vessels far removed from the gut anastomosis prevents gangrene (Labastie, in "Archives Générales de Chirurgie," Jan. 25, 1908). In most cases in which there has been rupture of mesenteric arteries death from hemorrhage rapidly occurs. If the victim should not die of hemorrhage he is in danger of gangrene and peritonitis. Aldrich[1] reported a case in which death was deferred until the seventh day. The treatment is immediate laparotomy. If the wound is found close to the gut, the portion of gut supplied by the cut vessel should be resected. If well removed from the gut, simply ligate the vessel (Labastie, Ibid.). In a wound near the root of the mesentery perform extensive resection of the gut and remove also a portion of the mesentery.

**Rupture of the Kidney.**—(See page 1280.)

**Rupture of the Ureter.**—(See page 1282.)

[1] "Annals of Surgery," March, 1902.

**Wounds of the Abdominal Wall.**—**Non-penetrating wounds** are to be treated on general principles. They are sutured with great care and are firmly supported externally. Ventral hernia may follow a large wound.

**Penetrating Wounds.**—The *symptoms* of penetrating wounds of the abdominal wall are usually those of shock and hemorrhage, and later of septic peritonitis. Emphysema is apt to occur and viscera may protrude, and often do in the case of a large incised or lacerated wound. Extravasation of contents of intra-abdominal viscera is very apt to occur, and is sure to occur if the viscus was distended when injured. Normal urine and normal bile may do little harm, but if either excretion is septic, disastrous consequences are certain to ensue. If intestinal contents escape, septic peritonitis is sure to arise. Bleeding is usually profuse and prolonged, because spontaneous arrest of hemorrhage from any vessel of considerable size will seldom take place within an unopened abdomen.

*Treatment.*—The surgeon endeavors to promptly discover if a wound of the abdominal wall is or is not penetrating in character. This fact may be proved by protrusion of viscera, by the appearance of stomach contents in the wound, or by a flow of bile, urine, or feces from the wound. If none of the above indications exists, and if there are no signs of serious hemorrhage, the wound should be irrigated with hot salt solution, and should be dressed with gauze, and every effort should be made to bring about reaction; otherwise operation should be immediate.

When reaction is obtained, the wound should be enlarged layer by layer until it becomes obvious whether or not the peritoneum is open. Madelung,[1] of Strassburg, points out that incision layer by layer will be of no use in settling the question of penetration if the wound is in the chest, the buttock, the perineum, or the back of a fat individual. If after incision layer by layer it becomes evident that penetration has not occurred, the wound should be closed and treated on general principles. If it becomes evident that it has occurred, the abdomen should be opened at the point of penetration, and a thorough exploration of intra-abdominal structures should be made in order to locate injury and be able to treat it properly.

In a case still doubtful after incision by layers, do an exploratory laparotomy in the middle line. It is impossible from the appearance of the wound and it may be impossible from the symptoms to affirm that visceral injury has not occurred; hence in every case in civil practice in which it is evident that penetration has occurred, laparotomy is necessary in order to detect and correct intra-abdominal injury, and to clean the peritoneum by flushing with hot salt solution. If viscera protrude, they must be washed off with hot salt solution and covered with hot sterile pads, and after the patient has reacted the wound should be enlarged, the condition of the contents of the abdomen investigated, hemorrhage arrested, wounds properly treated, and the viscera returned.

It is customary to flush the belly with hot salt solution, some of the fluid being allowed to remain. This procedure mechanically cleanses the peritoneum, removes blood-clots, strongly combats shock, and antagonizes infection. It is not absolutely necessary to flush out the belly unless a considerable hemorrhage has occurred or feces or stomach contents have been extravasated. If extravasation of stomach contents or feces has occurred, not only should flushing be practised, but evisceration should be carried out; the fouled intestine should be wiped off with gauze pads wet with hot salt solution, and while extruded should be kept wrapped in hot moist towels; the peritoneal fossæ should be rubbed with gauze pads and the space between the liver and the diaphragm should be carefully wiped.

[1] "Annals of Surgery," Sept., 1897.

A wound of the stomach should be sutured; a wound of the bowel may be sutured, or resection and anastomosis or resection and end-to-end suturing may be required. Visceral injuries are treated by appropriate means. In a punctured wound or a gunshot-wound of the intestine rectal insufflation of hydrogen gas when the abdomen is open may disclose the situation of the injury, but evisceration is usually practised instead and is preferable.

After the completion of intra-abdominal manipulations the surgeon restores any protruding bowel.

Drainage is required if the contents of the stomach or the intestines escaped, if hemorrhage was severe, or if the liver, pancreas, kidney, or spleen were found to have been damaged. The peritoneum may be sutured with a continuous suture of catgut; the muscles and fascia, with continuous or interrupted sutures of catgut, and the skin, with interrupted sutures of fine silk or of sikworm-gut. If there is need of haste, through-and-through sutures of silkworm-gut may be used. Active stimulation and artificial heat are needed immediately after the operation to combat shock. In many cases intravenous infusion of hot normal salt solution is given. It is of great value. It may be given both during and after operation. Enteroclysis, or high rectal injection of hot saline fluid, is useful. So is hypodermoclysis, or the subcutaneous injection of hot salt solution. The usual after-treatment consists of the semi-erect position, continuous proctoclysis of salt solution, avoidance of food by the stomach for forty-eight hours, and the administration of brandy and water from time to time. For two days the patient should be fed by the rectum. On the appearance of flatulent distention, forty-eight hours or more after the operation, give a saline cathartic. It is not wise to purge during the first forty-eight hours after the operation unless a Murphy button was used. When there is no sign of peritonitis a purge should not be given until the fourth day. After forty-eight hours liquid food can usually be given by the stomach. Solid food may be given after seven or eight days, but the patient must not leave his bed until the abdominal wound is firmly united because of the danger of ventral hernia. A support should be worn for a long time. E. D. Fenner[1] reports 39 stab-wounds of the abdomen operated upon in the Charity Hospital of New Orleans. There were 9 deaths (23.07 per cent.).

**Gunshot-wounds of the Abdomen.**—The bullet may penetrate from the front, the side, the back, the chest, or the perineum. If a bullet has penetrated, it may or it may not have produced visceral damage; a pistol-bullet or the bullet of a sporting-rifle almost invariably does. A projectile of a modern military rifle may not or may produce wounds which can be recovered from without operation. A urinary examination should be made promptly to see if blood is present.

In gunshot-wounds of the belly shock is usually greatly added to by hemorrhage, and in civil practice prompt operation is certainly indicated. The incision is made through the belly even when the shot entered the back. In some cases the opening is made through the wound; in others it is not; but in every case the wound made by the bullet is explored and disinfected. The incision should be long enough to permit of thorough work. After opening the abdomen our first duty is to arrest hemorrhage, our next is to look for perforations of the viscera and mesentery and close them. If the anterior wall of the stomach is perforated, close the opening and examine the posterior wall through an opening made in the gastrocolic omentum. If a posterior perforation is found, close it, and insert posterior drainage into the lesser peritoneal cavity. As a rule, an intestinal perforation can be closed, but occasionally a portion of the intestine

[1] "Annals of Surgery," January, 1902.

requires resection. If the bullet is encountered it is removed, but a prolonged search for it should never be made. Finally, the abdominal cavity is cleansed, drainage is provided for, and the abdominal wound is closed. In one of my fatal cases the bullet entered the rectum low down and was not found. In a case of mine with 6 perforations of the small intestine recovery followed operation.

E. D. Fenner[1] reports 113 gunshot-wounds of the abdomen operated upon in the Charity Hospital of New Orleans; there were 78 deaths (69 per cent.). In a series of 14 cases operated upon by Vaughan the mortality was 64 per cent. ("Am. Jour. Med. Sciences," Feb., 1906).

Military surgeons have shown that wounds inflicted by the modern hard-jacketed projectile are not so apt to involve fatal hemorrhage and disastrous complications; in fact, such wounds are often recovered from without operation, and sometimes with an entire absence of serious symptoms. Again, it is difficult or impossible to treat such cases as in civil practice, even were it desirable. In fact, in military practice the results are slightly better from expectant treatment, whereas in civil practice expectant treatment is a ghastly failure. Still, even in war, if conditions permit, operation should be performed if there is hemorrhage or obvious visceral injury, or if septic peritonitis develops. Treves says that in the Boer War only 40 per cent. of cases of gunshot-wounds of the abdomen not operated upon died, but, as pointed out by Hildebrandt, many cases die on the battlefield and while being taken to the hospital, hence the real mortality of these injuries is much more than 40 per cent. In the war between China and Japan the mortality from gunshot-wounds of the abdomen is said to have been about 77 per cent.

**Gunshot-wounds of the Pregnant Uterus.**—It is rarely that both walls are perforated, as the force of the bullet is greatly lessened by the uterine contents. There are severe shock and hemorrhage, and occasionally amniotic fluid flows from the wound of entrance. The intestine may also be injured. As a rule, labor-pains come on soon after the injury. The proper treatment early in pregnancy, if the wound is small, consists in emptying the uterus and closing the wound. In Albarran's case the fetus was expelled forty-eight hours after operation. A large wound, or any wound late in pregnancy, demands the Porro operation—removal of the uterus, ovaries, and tubes. Gellhorn[2] has collected 18 cases. In this series there were 12 recoveries. Russell Fowler reports a case of a patient eight months pregnant who was shot twice in the abdomen. There were two wounds in the uterus. Fowler did a Cesarean operation. The cut in the uterus was carried through one of the bullet wounds. The other bullet wound was sutured. The baby was wounded on the fingers of the right hand. Both mother and baby recovered ("New York State Jour. of Med.," Nov., 1911). In Judge's case ("Jour. Am. Med. Assoc.," vol. i, 1912), a girl of sixteen, nearly at full term, was shot with a Winchester rifle. The child was in the abdominal cavity and dead. The mother recovered. Tucker, of Shanghai (Ibid.), did a Cesarean operation for gunshot-wound of the uterus. The mother died and the child survived.

**Omental Cysts.**—Cysts may spring from the outer surface of the omentum (escaped ovarian cysts, lymphatic cysts, dermoids).

True omental cysts arise within the cavity of the great omentum. In 50 per cent. of cases the patient is an adult, but in half of the reported cases the patients were under ten years of age. Some of the cysts are probably of embryonic origin. Dowd is inclined to think that the cyst he removed resulted from omental hematoma ("Annals of Surgery," Nov., 1911). In this case there was torsion of the pedicle of the cyst. Dowd (Ibid.) reports 1 case of omental cyst and collected 37 from literature. Hasbrouck ("Annals of Surgery," August,

[1] "Annals of Surgery," January, 1902.
[2] "St. Louis Med. Review," Dec. 2 and 9, 1901.

1908) points out that the condition is much more common in females than in males, that there are no characteristic symptoms, that the condition begins as omental endothelioma between the two surfaces of omentum which fuse because of inflammation and form a closed sac, that hemorrhages are apt to occur into the cyst, and that operation shows a mortality of 6 per cent. The operation performed is extirpation.

**Mesenteric Cysts.**—These rare cysts are divided into (1) embryonic, (2) hydatid, (3) cystic malignant disease (Dowd, "Annals of Surgery," 1910). A great majority of mesenteric cysts are embryonic. Moynihan believes that embryonic cysts arise in "rests" from "the Müllerian or Wolfflan organs or ducts, or from the ovary," which are included between the folds of mesentery. These rests undergo cystic degeneration. Mesenteric cysts grow and pass along between the layers of mesentery until they reach the gut and then compress the gut and push its wall ahead of them.

Such a cyst may contain embryonal material, bloody fluid, serous fluid, chylous fluid, or lymph. In Harry C. Deaver's report of a case ("Annals of Surgery," May, 1909) it is shown that in 40 cases there were 25 females and 15 males; that the size varies from that of a split pea to enormous dimensions; that there may be a pedicle form in front of the vertebral column or from the intestinal wall; that the sac wall may be exceedingly thin or as much as 1 cm. in thickness; that in some cases large veins are present on the surface of the cyst; that adhesions to the abdominal viscera are common; that intestinal obstruction may occur (kinks, volvulus, intussusception); that cysts are most commonly found in relation with the lower end of the ileum; and that there are no characteristic symptoms except perhaps the rapid body wasting mentioned by Moynihan. Moynihan also points out that the cyst fluctuates and is most prominent toward the navel, that it is very mobile, especially transversely (we should add if not anchored by adhesions), and that the cyst is surrounded by a resonant zone and crossed by a resonant band.

**Treatment.**—Incision and drainage if there are numerous and firm adhesions. Enucleation whenever possible. Resection of the involved portion of gut with the cyst in some cases of multiple cyst (Harry C. Deaver, Ibid.).

**Torsion of the great omentum** is usually caused by a fall or strain or attempts to reduce a hernia. In nearly all cases a hernia is present. Hedley ("Brit. Med. Jour.," Nov. 11, 1911) studied records of 73 cases. In 60 there was or had been a hernia. In 5 cases without a hernia there were adhesions. In 48 of the hernia cases the omentum was attached in the sac, in 4 it was anchored near the sac, in 3 to the bowel, and in 1 to the Fallopian tube.

**Symptoms** are like those of a strangulated omental hernia or of appendicitis.

The **treatment** is by laparotomy.

## Stomach and Intestines

**Foreign Bodies in the Stomach and Intestines.**—Foreign bodies of considerable size are rarely taken into the alimentary canal except by children, insane people, or drunkards. Small bodies (bits of straw, fragments of bone, etc.) are frequently swallowed. Most foreign bodies swallowed are passed with the feces, but some lodge. Any body which can pass the esophagus is not too large to pass through the intestines. Lodgment is an accident, not an inevitable consequence—an accident which is due to the shape and size of the body. A foreign body may lodge in the stomach. In some cases there are no symptoms. In other cases symptoms are violent. The severity of the symptoms depends upon the shape and character of the body.

In some cases it is possible to feel the body from without. A metal body in the stomach will deflect a magnetic needle held over the viscus (Polaillon). Many foreign materials can be skiagraphed. A body of small size may pass through the entire canal and emerge without having done any harm, but it may lodge and may cause perforation. If perforation occurs, the foreign matter may become encysted, for instance, in the mesentery; may cause an abscess or may cause general peritonitis. A fish-bone may cause an anal abscess. An epiploic appendix may cause sacculation of the bowel, perforation by a foreign body may take place in this sac, an *epiploic abscess* resulting, which may attain considerable size and may be mistaken for carcinoma (Sir J. Bland-Sutton, in "Lancet," Oct. 24, 1903). It is not wise to attempt to recover a foreign body from the stomach by inducing vomiting. In some cases gastrotomy is necessary. When a small or sharp foreign body has been swallowed and has not caused perforation, abscess, or obstruction, the usual treatment is as follows: a purgative should *never* be given to expedite the passage of a foreign body, because increased peristalsis increases the danger of impaction or of perforation. Endeavor to encrust the foreign body, and thus lessen the danger of perforation, by feeding with bread and milk only for several days, and at the end of this period give a mild laxative. An exclusive diet of mush or of mashed potatoes has been suggested. Suet dumplings may be given. Pain is relieved by opium. A foreign body rarely lodges in the duodenum, but may lodge lower down, and may cause ulceration, perforation, abscess, or intestinal obstruction. Operation is necessary in such cases.

**Volvulus of the Stomach.**—This condition is very unusual. Ten cases are on record (Streit, in "Am. Jour. Med. Sciences," June, 1906). One-third of the cases are associated with diaphragmatic hernia. The symptoms come on suddenly. There is violent abdominal pain. Distention and collapse are early. There is nausea, but the patient can neither vomit nor belch. In the upper left abdomen there is a tender, tense, and tympanitic area. The rotation of the stomach may be on its vertical or on its longitudinal axis. An hour-glass stomach may undergo twisting on its vertical or longitudinal axis. Berg operated successfully for volvulus of the stomach. He opened the abdomen, relieved distention by tapping the stomach with a trocar, and then easily corrected the twist. The gastrohepatic omentum should be shortened by Beyea's operation (see page 1104).

**Fibromatosis of the Stomach.**—This condition was long ago described by Brinton as cirrhosis of the stomach. Alexis Thomson has recently published an admirable description containing many new things ("Annals of Surgery," July, 1913). He shows that ulceration of some form is the cause (simple ulceration or cancerous ulceration). The condition may be and commonly is localized, but it may involve the entire stomach ("leather-bottle stomach"). There may be glandular enlargement. The local form starts at the pylorus and resembles cancer, but is not nodular. There may or may not be adhesions. There is no "cicatricial stenosis of the pylorus," but the canal of the pylorus is narrowed by submucous thickening. The thickening is not scar-tissue, but is more like a fibroma (Ibid.). It is probably a reaction against infection. Thomson proves that there is an innocent form of fibromatosis, and thinks it probable that there may even be an innocent form of leather-bottle stomach (Ibid.).

The **symptoms** suggest ulcer, cancer, or some sequel of ulcer. It is often possible to be mistaken as to the nature of the mass even when it is exposed by exploratory incision. The cases of "pyloric cancer" which have recovered after gastro-enterostomy and some of the cures of gastric cancer by gastrectomy are explained when we know how easy it is to mistake innocent fibromatosis for cancer.

**Treatment.**—Thomson always insists on an immediate microscopical examination of a portion of the tumor or, better, of an enlarged lymph-gland. He advocates resection for local fibromatosis because he believes cancer is liable to arise. In some cases gastro-enterostomy is performed; in some, jejunostomy.

**Carcinoma of the Stomach.**—Innocent tumors and sarcomata occasionally attack the stomach, but they are infinitely rare in comparison with primary cancer. This disease is unusual before the age of forty, and is very seldom seen before the age of thirty. I operated upon a man of twenty-three for gastric cancer. It is more common in men than in women, the proportion being as 5 to 4. Beyond question, in some cases cancer arises from the margins of an ulcer. The forms of cancer which may arise in the stomach are the spheroidal cell growth (either the hard form known as scirrhus or the soft form known as medullary or encephaloid), the cylindrical cell growth or adenocarcinoma, and colloid (due to the myxomatous degeneration of either a spheroidal cell or a cylindrical cell carcinoma). Scirrhus more than any other form produces constriction of the pylorus and more seldom than any other form produces hemorrhage. It may spread for a considerable distance along the submucous coat, muscular coat, and subserous coat without apparent involvement of the mucous membrane wide of the primary focus of disease. Fibromatosis may develop about it. In some cases scirrhus is limited to the pyloric region, in others it is a limited tumor of some other part of the wall of the stomach. In the condition known as malignant "leather-bottle stomach" a scirrhus has invaded the entire stomach wall, or fibromatosis has developed with the cancer and the wall of the viscus is thick and rigid.

Medullary cancer and adenocarcinoma produce hemorrhage. Both of these forms most often arise in the pyloric region. Medullary cancer may remain limited, but, as a rule, a cauliflower growth arises and eventually fills the stomach and portions of the mass may slough away.

Adenocarcinoma is apt to spread along the mucous membrane instead of "infiltrating the deeper layers," as other forms are prone to do ("Cancer and Tumors of the Stomach," by Samuel Fenwick and W. Soltau Fenwick). Spheroidal celled carcinomata are twice as common as cylindrical celled, and medullary cancer is about as common as scirrhus. Any cancer may be accompanied by fibromatosis.

Cancer may be limited to the body of the stomach (either curvature or either wall), the pyloric end or the cardiac end, but it may involve two of these regions or almost the entire stomach, or, being multiple, may be found in many parts. Sometimes there is a primary cancer on one wall, and another growth, due to contact, on a corresponding point of the opposite wall (*contact cancer*). All forms of carcinoma most frequently begin in the region of the pylorus. In many cases of gastric carcinoma adhesions form between the stomach and the liver, colon, or diaphragm. Even in cancer of the pylorus the duodenum is seldom invaded. Medullary cancer not unusually passes into the esophagus. Gastric cancer is usually fatal in from four months to two years, and most patients die within one year. In 60 per cent. of cases the pylorus is involved. In over half of the cases of cancer of the pylorus there is no important lymphatic involvement (McArdle). In investigating any gastric disorder follow the advice of the Mayo brothers and study the history of the case, the size and situation of the stomach, the existence and situation of pain and tenderness, the presence of a tumor, and if the passage of food is interfered with.

**Symptoms.**—Examine with care a patient in whom cancer is suspected. In unusual cases it produces no symptoms until it has lasted for some time and has attained a large size (latent cancer). In nearly all cases it does produce symptoms. The disease comes on gradually, usually with indigestion and physi-

cal weakness. The patient has persistent dragging pain, which is increased by eating and pressure, and attacks of vomiting are frequent. After a short time he becomes very weak and exceedingly anemic, and it is often possible to feel a tumor in the stomach. Blood examination shows diminution of red corpuscles and hemoglobin and perhaps absence of any increase of leukocytes after a full meal. The vomiting of a patient with gastric cancer is at first only occasional, but as the case progresses it becomes more and more frequent. Vomiting soon after eating occurs when the cardiac region is involved; vomiting an hour or so after eating occurs when the pyloric end is involved. When the body of the organ is the seat of disease, vomiting may be absent. The vomited matter is often mixed with a small amount of altered blood (*coffee-ground vomit*). In most cases occult blood is found in the feces, especially if hydrochloric acid is absent from the stomach contents. These symptoms above cited associated with "coffee-ground vomit" and lactic acid in the stomach contents are strongly indicative of cancer (Wm. J. Mayo, in "Surg., Gynec., and Obstet.," May, 1908). A *test-meal* is given, and important conclusions are sometimes derived from the presence or absence of hydrochloric acid and lactic acid. It is my custom to have the stomach washed out and then have Ewald's test-breakfast given. This consists of one roll of white bread (35 gm.), 400 gm. of $H_2O$, and 400 gm. of tea without milk or sugar. In one hour the stomach is emptied by means of a tube and a pump or a tube and abdominal compression, and the material is examined. If the result of the test seems out of accord with the other symptoms, repeat the process (L. Boas, in "Berlin. klin. Woch.," No. 440, 1905). In most cases free hydrochloric acid is not found in the stomach contents, but lactic acid is found and Oppler's bacillus can often be detected. There may be red blood-corpuscles in the fluid. If the cancer is not ulcerated, free hydrochloric acid will probably be found; if it is ulcerated, it will usually be absent.[1] Free hydrochloric acid may be absent from the stomach because of atrophy of glands, cessation of secretion, or neutralization by the products of the cancerous area. Free hydrochloric acid may be absent in individuals in whom cancer does not exist. I have noted its absence in several cases of cicatricial stenosis of the pylorus.

It may be absent in cancer of the esophagus, advanced Bright's disease, cancer of the duodenum, distant cancer, febrile conditions, and amyloid disease. The constant presence of considerable quantities of hydrochloric acid is strong evidence against the existence of cancer of the stomach. If cancer arises from ulcer, free hydrochloric acid is apt to be present for a considerable time after the cancer has begun.

Distend the stomach with gas or fluid and map out its outlines. Feel for a tumor. A tumor can usually be felt if it involves the greater curvature or anterior wall, and a large tumor of the pylorus can be palpated, but in other regions the tumor can rarely be felt.

Cancer of the cardiac end interferes with the entrance of food into the stomach, and in such a case the stomach is shrunken and the esophagus is dilated immediately above the growth. In cancer of the pylorus the food is partially or completely arrested as it passes to emerge from the stomach, and the stomach becomes much dilated. The vomited matter in a case of cancer rarely contains recognizable fragments of the growth, but fluid with which the stomach has been irrigated may contain pieces which can be identified as cancer (Rosenbach).

In cancer of the stomach the general course of the temperature is normal, but there are occasional deviations to below or above normal. In many cases the urine contains albumin, indican, acetone, and casts. Occasionally cancer of the stomach produces spasm of the esophagus. I have seen this in several cases.

[1] Reissner, in "München. med. Woch.," Dec. 3, 1901.

Cancer of the stomach is apt to involve secondarily adjacent lymph-glands, or organs or other structures, especially the liver; in fact, the liver is involved in 30 per cent. of the cases (Welch). Occasionally there is enlargement of the supraclavicular glands of the left side. Metastases are usual and early, but in cancer of the pylorus 60 per cent. of the cases show no distinct lymphatic involvement. In many doubtful cases exploratory incision is imperatively required.

**Treatment.**—The *medical* treatment consists in milk-diet, the use of morphin, and of *lavage* if the pylorus or body of the stomach is diseased. Perform lavage as follows: The tube for lavage should be long enough to extend about 3 feet out of the mouth when the other end is in the stomach, it should be flexible, should have an opening in the stomach end and another opening on the side about 1 inch above the stomach end. The tube should be greased with glycerin. The patient sits down, throws the head back, opens the mouth widely, and is directed to take deep breaths, at regular intervals. The tube is carried into the pharynx, the patient is ordered to make efforts to swallow it, and the tube is thus taken into the stomach. About 1 quart of fluid is poured into the funnel-like end of the tube, and just before the tube empties itself of the last of the water the funnel is lowered and the fluid runs out. This proceeding is repeated until the fluid becomes clear. The best fluid to use is a solution of bicarbonate of sodium, a teaspoonful of the salt to a quart of warm water. Lavage should be practised before breakfast, and sometimes also at bed-time.

The *indications for operation* are well set forth by Macdonald[1]: They are progressive aggravation of symptoms in spite of a rigid diet and medical treatment, loss of gastric mobility, progressive diminution of gastric peristalsis, progressive diminution of free hydrochloric acid, emaciation even under forced feeding, progressive reduction of hemoglobin to 65 per cent. or under, and moderate leukocytosis.

*Surgical* treatment aims to remove the growth or to obviate the effect of obstruction at one of the orifices of the stomach.

In cancer of the body of the stomach, if the growth is not extensive, excision of the growth may be performed; if it is extensive, it is useless to attempt it unless the growth is absolutely non-adherent. Conner, of Cincinnati, attempted total excision of the stomach in 1883, but the patient died on the table. In 1897 Schlatter, of Zurich, successfully removed the entire stomach. Brigham, Richardson, Macdonald, Boeckel, De Carvalho, Bardeleben, Haine, Gallet, Dollinger, Ferry, Ribera, and others have successfully removed the entire stomach and attached the esophagus to the small intestine (*complete* or *total gastrectomy*). In the successful cases digestion was satisfactorily performed after removal of the stomach. Very rarely will cases be found suitable for such a radical proceeding. The case suitable for this treatment is one in which the entire stomach is involved in the growth, in which there is no obvious glandular involvement, and in which the stomach is not adherent, but is freely movable. Herbert J. Paterson ("The Hunterian Lectures for 1906") collected 27 cases of total gastrectomy for cancer: 10 died and 17 recovered. If a small portion of the fundus is left the operation is called *subtotal gastrectomy*. H. J. Paterson (Ibid.) collected 20 cases of subtotal gastrectomy for cancer with 6 deaths. In limited cancer of the body of the stomach perform *partial gastrectomy*. In cancer of the cardiac orifice of the stomach the surgeon usually keeps the passage open as long as possible by the frequent passage of a tube, and through this tube introduces liquid food. Sometimes a small tube is introduced and permanently retained. When it becomes difficult to introduce a tube, *gastrostomy*, *duodenostomy*, or *jejunostomy* may be performed. As a matter of fact, in most

[1] John B. Murphy, in "Chicago Med. Recorder," June 15, 1902.

cases gastrostomy is done as a last resort, and it is scarcely worth doing in cancer of the cardiac end of the stomach. It is far more useful in cancer of the esophagus. In cancer of the pylorus, limited in extent and without lymphatic involvement, *pylorectomy* may be performed; but in cancer which has widely infiltrated the coats of the stomach and has involved the lymphatic glands, *gastro-enterostomy* is performed as a palliative measure, the patient during the rest of his life subsisting upon liquid or semiliquid foods and submitting to frequent irrigation of the stomach to remove food residue. In cases of irremovable and far-advanced cancer it is often best to refuse to operate and to deliberately create the opium-habit in the patient, although, in some cases, duodenostomy or jejunostomy may be performed.

The most successful of all the above operations are pylorectomy and partial gastrectomy. The mortality is large. In H. J. Paterson's series of cases the mortality was 28 per cent. There are in literature many cases which have survived three years or over. Mayo reported 21 gastro-enterostomies for cancer, with 4 deaths. The greatest prolongation of life was nineteen months. His experience makes him question if the operation is worth doing in malignant disease.

**Sarcoma of the Stomach.**—Of recent years it has been proved that sarcoma is more common than was once supposed. There are over 60 cases on record. It can occur at any age, but is more usual in early life than carcinoma. It has been estimated by Wm. T. Howard[1] that 37.7 per cent. of cases are under the age of forty, and 11.44 per cent. are under the age of twenty. The pylorus is involved in about one-fourth of the cases. In most cases the posterior wall and greater curvature are involved. Howard says there is a diffuse growth in 21.31 per cent. of cases and that the cardiac end is involved in only 4.9 per cent. of cases. Sarcoma arises in the submucous coat. Any form of sarcoma may arise. It causes stenosis in less than one-tenth of the cases. There is no sex predisposition in sarcoma. The growth may attain a great size.

**Symptoms.**—A tumor forms, grows rapidly, and often attains a large size, and not unusually actually causes a projection of the abdominal wall. If it ulcerates there will be hematemesis, but it often does not ulcerate, and bleeding is much rarer than in carcinoma. Not unusually this growth arises in a person under forty, and sometimes in one of less than twenty years of age. Stenosis is uncommon. The liver is involved secondarily in only 11.47 per cent. of cases (Howard), metastases are more rare than in carcinoma, free hydrochloric acid is usually absent from the gastric contents, and microscopical examination of washings from the stomach may detect fragments of sarcoma. Certain diagnosis is impossible without exploratory incision. Howard estimates the average duration of life to be from nine to ten months.

**Treatment.**—If the liver is free and if there are no metastases, partial gastrectomy or complete gastrectomy may be advisable. If pyloric stenosis should arise, gastro-enterostomy may be performed. Scudder ("Annals of Surgery," August, 1913) reports a case. He performed jejunostomy. Six weeks later he removed the tumor by partial gastrectomy, closed the jejunal opening, and did anterior gastro-enterostomy. A year later the man remained well.

**Ulcer of the Stomach (Peptic Ulcer of the Stomach).**—Ulcer of the stomach is a condition due to digestion of a portion of the stomach wall by very acid gastric juice, the destroyed portion having been the seat of lowered vitality. The reason for the lowered vitality of the gastric mucous membrane is uncertain. Thrombosis has been suggested as a cause, but it is rare in gastric ulcer. Embolism is assigned by some as a cause, but emboli are seldom found on pathological examination. It has been asserted that

[1] "Jour. Am. Med. Assoc.," Feb. 8, 1902.

menstrual disorders may be responsible for ulcer, that tight lacing may be, and that habitually bending over (as in making shoes) may be a cause. The Mayos are of the opinion that the grinding action of the pyloric portion of the stomach may be a traumatic exciting cause of ulcer of that region. Some assert that mental anxiety, alcoholism, and syphilis may be causal (Alderson). Thirty-two per cent. of the cases in the Mayo Clinic used alcohol. Ulcers due to syphilis and tuberculosis are not peptic ulcers.

Robson believes that gastric ulcer is septic in origin, and that oral sepsis is responsible for its origin in most cases. "Mild sepsis leads to gastritis and hyperchlorhydria, which in its turn provokes and keeps up ulceration" (A. W. Mayo Robson, in "Keen's Surgery," vol. iii). In 140 cases studied by Smithies in the Mayo clinic ("Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912) 26 had had enteric fever, 6 pneumonia, 5 syphilis, 8 malaria, and 27 some other general infection. Twelve had previously been operated upon for appendicitis and 2 for gall-stones. Some observers blame direct damage to the mucous membrane by traumatism or the swallowing of corrosive liquid. The question of cause is involved in uncertainty. What does seem to be certain is that anemia predisposes to the formation of very acid gastric juice (*hyperchlorhydria*) and to ulceration. In some cases chlorosis is associated with ulcer. According to Wm. J. Mayo there are three known causal factors of the first importance, viz., anemia, hyperchlorhydria, and traumatism (April 16, 1904).

It used to be stated that ulcers are far more common in females than in males. This statement is not correct. It applies to acute ulcers, but not to chronic ulcers. Up to July 1, 1912, the Mayos had operated on 404 proved ulcers of the stomach, and over 70 per cent. of these patients were males (Smithies, in "Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912). The acute round ulcer is vastly more common in women, and in young women rather than in those of middle or advanced age. The chronic indurated ulcer is most frequent in men. Men about forty, and women between twenty and thirty are particularly liable. Between thirty and forty is the period of greatest liability. I have only once found ulcer in a person under twenty. There is usually a single ulcer, but in one-fifth of all cases there are two or more, and when there is an ulcer on the anterior wall, it is not uncommon to find one exactly opposite on the posterior wall (*a kissing ulcer* Moynihan calls it). The Mayos divide ulcers into two clinical forms—the *indurated* and the *non-indurated*. In the indurated ulcer all the coats of the stomach are involved, and the mass of scar tissue indicates an effort at repair. The most common situation for this form of ulcer is the region of the pylorus (Wm. J. Mayo, in "Jour. Am. Med. Assoc.," Oct. 21, 1905). The non-indurated ulcer involves the mucous coat only and may be of microscopical size, and even a microscopical ulcer may cause death from hemorrhage. These non-indurated ulcers exhibit no sign, or almost no sign, on the outer surface of the stomach, and may not be detected even when the stomach is opened by the surgeon. The non-indurated ulcers are divided into the *mucous erosions* of Dieulafoy, in which the superficial epithelium only is involved, and the true round fissured *peptic ulcers* (Wm. J. Mayo, in "Jour. Am. Med. Assoc.," Oct. 21, 1905). Both conditions are rare. Ulcers are also divided into *acute ulcers*, which progress rapidly and produce definite symptoms, and *chronic ulcers*, which are usually chronic from the beginning, but which may exhibit acute exacerbations, and may have periods of great relief or apparent cure (Wm. J. Mayo, in "Med. Record," August 6, 1904). The most common seats of ulcers are the posterior wall and lesser curvature, especially in the pyloric region; in fact, 80 per cent. occur in the pyloric region. An ulcer may heal or may perforate. Only 1 or 2 per cent. of ulcers on the posterior wall perforate, as they tend to form adhesions to adjacent structures. Ulcers on

the anterior wall are unusual, do not tend to form adhesions, and are apt to perforate. It is not uncommon to have ulcer of the first portion of the duodenum associated with gastric ulcer. An ulcer may be accompanied or followed by fibromatosis which gives a deceptive likeness of cancer (see page 957). Gastric ulcer is at least four times as frequent in England as in the United States. In 2830 autopsies made in the Philadelphia Hospital there were 40 gastric ulcers, and in 3763 autopsies made in four Philadelphia institutions there were 51 gastric ulcers—a percentage of 1.35. (See A. P. Francine, in "Proceedings Phila. County Med. Soc.," March 31, 1905.)

**Symptoms.**—In an acute ulcer the symptoms are often typical; there are pain, tenderness on pressure, slight or distinct unilateral muscular rigidity, vomiting, hemorrhage, and hyperchlorhydria. In a chronic ulcer the symptoms may be clear, may be misleading, may be variable, and in some cases even absent (*latent ulcer*). In ulcer dyspepsia usually exists. It is usually but not always acid dyspepsia, and is associated with much flatulence. In most cases, though not in all, food promptly causes pain. There is a gnawing sensation (*hunger-pain*) when the stomach is empty, and may be actual pain. The taking of food may *temporarily* relieve pain, but as gastric peristalsis arises and perhaps as quantities of gastric juice are poured out to digest the food the pain increases. If hyperchlorhydria disappears (as it may do from chronic gastritis and does from gastric dilatation following pyloric obstruction), pain may not be increased during digestion. In ulcers of the cardiac end and lesser curvature gnawing uneasiness is but briefly or not at all relieved by taking food and pain develops immediately or almost at once after eating. In ulcer of the pyloric region the gnawing uneasiness may be distinctly relieved by taking food, but in an hour or two hours pain is apt to become severe. The time after eating when pain occurs may not be constant in a case. In ulcer the pain is paroxysmal. It is at times very violent in the epigastric region, and may pass to the back, being located between the eighth and ninth dorsal vertebræ to one side of the back (the right most often), into the esophagus, into the chest, or to the top of the ensiform process.

In gastric ulcer it is usual to find distinct or severe tenderness developed by epigastric pressure, and tenderness is associated with more or less rigidity. In ulcer of the lesser curvature pain and tenderness are in the neighborhood of the left costal margin. In ulcer of the pylorus they are above the umbilicus in or to the right of the midline. Vomiting usually relieves the pain, so does lavage, so does the administration of an alkali. In ulcers of the anterior wall tenderness is most acute. In many of these patients vomiting occurs about two hours after eating. The vomited matter, as a rule, contains much hydrochloric acid and the vomiting usually relieves the pain. Examination of the gastric contents after the administration of a test-meal shows in about 80 per cent. of the cases hyperacidity. Obvious hemorrhage from the stomach occurs in less than one-half of the cases, and from 3 to 8 per cent. of cases actually die of hemorrhage. Wm. J. Mayo states that "more than 90 per cent. of hemorrhages from the stomach are from chronic ulcers with a well-marked ulcer history" ("Surg., Gynec., and Obstet.," May, 1908). The blood may be brought up with food, and is then black and clotted, or may be vomited clear and in large amount. Blood may be present in vomited matter or stools in such small amount that its presence is observed only by the microscope. The demonstration in the feces of minute quantities of blood (occult blood) is important diagnostically. It may be demonstrated by the guaiacum test or the aloin test. For two days before this test the patient must not eat rare meat, sausages, or fish. Blood in the stools does not prove the existence of gastric ulcer. The blood may have come from any spot from the mouth to the anus.

As Wm. J. Mayo ("Surg. Gynec., and Obstet.," May, 1908) says: "Visible or occult blood in the stool affords proof as to the fact that there is blood, but it should never be lost sight of that it bears with it no evidence as to its exact gastro-intestinal origin. The patient may have bleeding gums or hemorrhage from some slight abrasion in any part of the many feet of mucous membrane which exist between the lips and the anus. If occult blood is found by one chemical test, it must be corroborated by others, as some unsuspected food or drug may give rise to the reaction.

"As a matter of fact, hemorrhage from ulcer is by no means of frequent occurrence. The base of the ulcer is clean and free from granulation tissue, so that bleeding may be infrequent. Careful examination of the stools for many days may be necessary to detect its presence."

In hemorrhage from an acute ulcer a pint or two may be ejected in a few minutes, and such a patient presents all the general symptoms of dangerous hemorrhage. When an ulcer bleeds the blood is far more apt to be vomited than passed by the bowels, but in some cases blood from the stomach is passed by the bowels in part or wholly. A very large hemorrhage may occur, and yet the bleeding never be repeated, or a large hemorrhage may be followed by another or be the first of three or of a series. In a great many cases after a large hemorrhage there is no further bleeding or there are subsequently a few small hemorrhages. Small hemorrhages may occur indefinitely, and may after a time eventuate in a large hemorrhage. In chronic ulcer in which small hemorrhages recur over a long period the condition is due to bleeding from congested mucosa, dilated veins, or to the erosion of small vessels which cannot contract or retract because they are embedded in fibrous tissue. A large hemorrhage may be due to the erosion of a large vessel, but is often produced by the existence of a great number of erosions of the mucous membrane, erosions perhaps so numerous that blood seems to pour from every portion of the mucous surface. It is usually stated that in a sudden acute violent hemorrhage there will probably be no history of antecedent stomach trouble, but Wm. J. Mayo is of the opinion that "a single hemorrhage from a patient who has not had previous gastric symptoms is probably not due to ulcer" (Ibid.). It may arise from rupture of veins about the cardia or from blood from hemoptysis being swallowed.

In a chronic ulcer it is sometimes, though seldom, possible to palpate the indurated area.

Constipation exists in at least 90 per cent. of cases. There is often very marked anemia, aggravated and, some think, occasionally caused by continued loss of blood. Indigestion aggravates anemia and also may cause it. Most cases complain of prolonged indigestion. There is often a tender area in the back a little to the side of the eighth and ninth dorsal spines (usually the right side). A triangular area of hyperesthesia may be found in the left epigastric region (Head).

If the ulcer does not cicatrize, but progresses, causing pain and hemorrhage, the patient usually becomes thinner, more anemic, weaker, and even exhausted. In 140 cases in the Mayo clinic studied by Smithies ("Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912) 107 showed loss of weight. The average loss of weight was 20 pounds. The maximum loss observed was 65 pounds in six months. The average hemoglobin finding was 72 per cent.

It is certain that many cases of gastric ulcer are unrecognized; in fact, as Habershon says, diagnosis is rarely made unless hemorrhage exists, and in certain latent cases both vomiting and bleeding are absent. It is believed by some that latent ulcers are even more common than are ulcers causing symptoms. Hall ("Am. Jour. of Med. Sciences," May, 1909) says: "Rather than look too narrowly for exactly this or that evidence, we should take the broader ground that ulcer probably exists in most patients complaining of persistent

indigestion, even though not of an acid character, if pain, tenderness, vomiting and rigidity, or two or three of these phenomena be present, and even though hyperacidity be not proved." A bleeding ulcer with palpable thickening of the pylorus, especially if there is anemia and loss of weight, is frequently mistaken for cancer. *Fibromatosis* of the stomach with or without ulcer is usually diagnosticated as cancer. This condition was fully described by Alexis Thomson at the 1913 meeting of the American Surgical Association. The entire stomach may be indurated. It is cases like the above that may get well after gastro-enterostomy and thus furnish the first obtained proof that the condition was not cancerous (see page 957). The diagnosis of ulcer is far less difficult when there is food retention than when there is not.

The fluoroscope may aid in the diagnosis. Skiagraphs after a bismuth meal give highly important information as to the existence of scars, contraction of the pylorus, dilated stomach, and hour-glass stomach. In many cases it is impossible to make a differential diagnosis between pyloric ulcer and duodenal ulcer. In duodenal ulcer the pain and tenderness are above the umbilicus and in the midline or to the right, and if the duodenal ulcer bleeds the blood is most apt to pass by the bowel, but vomiting of blood is not unusual. A small percentage of duodenal ulcers also involve the pylorus.

A gastric ulcer may cicatrize and thus be cured, but the cure of the ulcer may prove the ruin of the stomach by producing stenosis of one of the stomach orifices or hour-glass contraction of the body of the stomach. An ulcer may perforate. Perforation occurs in about 15 per cent. of cases (Robson). A *perforation* may be *acute;* that is, the ulcer suddenly breaks open when the stomach contains food or liquid, and the contents of the stomach are poured into the free peritoneal cavity. A *subacute* perforation occurs when the stomach is empty or nearly empty. The opening is small in size, there is no escape of stomach contents or the escape of only a small amount, and the opening may be quickly closed by adhesion to an adjacent surface of peritoneum or a piece of omentum. If a certain amount of stomach contents is extravasated, it is usually surrounded by adhesions or tracks slowly toward the pelvis. In what is known as a *chronic* perforation the break takes place usually in the posterior wall into a box of preformed adhesions, the extruded gastric contents are circumscribed by these adhesions, the general peritoneal cavity is not invaded, but circumscribed suppuration is inaugurated.[1] This condition is known as *perigastric abscess*, and the subphrenic form is the commonest. In such a case the abscess may break into the pleural cavity or even into the lung. I operated on a girl of sixteen and found a perigastric abscess and a perforation of the anterior wall near the pylorus, and this condition was tuberculous. A fistula persisted for months, but finally healed.

*Perforation* is generally preceded by a history of indigestion, but it may come on without a suggestion of antecedent stomach trouble, is usually brought about by muscular effort, and is most common after a full meal, but it may occur when the patient is perfectly quiet and has not eaten for some time. The real cause is spasm of the pylorus, which causes tension of the stomach walls and keeps the viscus from emptying. Pyloric spasm is very common in sufferers from ulcers. In acute perforation food is the most active cause; in chronic perforation, muscular effort. "The severity of the symptoms depends upon several conditions: the previous state of health, the size and number of the perforations, the condition of the stomach, whether full or almost empty, the bacterial virulence of its contents, and the occurrence of vomiting."[2] The situation of the ulcer has some influence on the symptoms. "If in the fundus, at the cardiac end, or in the body of the stomach, an acute infection of the whole

[1] See paper by B. G. A. Moynihan, "Brit. Med. Jour.," Jan. 31, 1903.
[2] Moynihan, in "Brit. Med. Jour.," Jan. 31, 1903.

peritoneal cavity rapidly follows; if the ulcer be at the pylorus or in the first portion of the duodenum, the fluid is directed down the right side of the abdomen, owing to the hillock formed by the transverse mesocolon at the pyloric end of the stomach".[1] In such a case the fluid may gravitate toward the right iliac region and the condition may be mistaken for appendicitis. In a case of subacute perforation I operated, believing that appendicitis existed. Alderson calls attention to the fact that the sudden perforation of an ulcer may be mistaken for poisoning, and he cites the death of Henrietta, Duchess of Orleans, in 1670.

*Acute perforation* can be certainly diagnosticated if the case is seen early. Such an emergency has usually, but not invariably, been preceded by positive and prolonged symptoms of gastric disorder. It causes sudden and intensely violent epigastric pain, greatly increased by swallowing fluids, by vomiting, by turning the body, by cough, by inspiration, and by pressure. This pain may radiate throughout the abdomen, but the chief tenderness is in the region of the stomach. The seat of the pain after perforation does not, of necessity, correspond to the seat of perforation. Vomiting occurs in about half the cases after rupture. When it does occur it comes on soon after the pain, may recur again and again, and does much harm by increasing shock and by ejecting gastric contents into the peritoneal cavity. Vomiting of blood is very unusual. In many cases there is, singularly, little shock. Even when severe shock exists its duration is usually temporary. This important fact is insisted on by Eliot ("Annals of Surgery," May, 1912). Board-like rigidity exists, and it is most marked in the upper portion of the abdomen. The area of liver dulness is in some cases diminished and in exceptional cases obliterated. This symptom is due to gas passing into the peritoneal cavity and getting between the liver and the parietal peritoneum. It is seldom present after pyloric perforation or when the stomach at the time of the perforation contained very little food. It is when perforation is far from the pylorus and when the stomach contains fermenting food that enough gas escapes to diminish liver dulness (Eliot, Ibid.). There may be dulness in one flank or both flanks due to fluid. Eliot (Ibid.) lauds auscultation as an aid to detecting small amounts of fluid, and calls attention to *Shoemaker's symptom*, that is, a dull note on light percussion giving way to a tympanitic note when the percussed finger is pressed firmly against the abdominal wall, thus coming nearer to the intestine by pushing fluid away. The pulse may be very rapid, but often shows curiously little disturbance. Some few cases die rapidly in shock, but, as a rule, reaction occurs and, if operation is delayed, peritonitis arises. Acute perforation of the stomach may be in certain cases mistaken for appendicitis, cholecystitis, or hemorrhagic pancreatitis. If a patient with acute perforation is not promptly operated upon, he will soon exhibit the symptoms of general peritonitis. Subacute perforation causes less violent symptoms and they come on more gradually. There is in the beginning severe but not agonizing pain, which gradually abates. Moynihan points out that there is gastric uneasiness for several days before the perforation. Peritonitis develops slowly and the chief symptoms are often pelvic. *Chronic perforation* gives the signs and symptoms of perigastric abscess.

**Treatment.**—*Medical Treatment of Non-perforated Ulcer.*—Rest in bed. It is necessary to abandon stomach feeding for a time. For seven to ten days give nothing whatever by the mouth and give an enema of 10 oz. of normal salt solution every sixth hour. This is preferable to a nutritive enema because every time a nutritive enema is given a flow of gastric juice takes place into the stomach. (See W. Pasteur, in "Lancet," May 21, 1904; Seymour J. Sharkey, in "Lancet," Nov. 10, 1906.) During this treatment the patient is usually comfortable and is not unbearably disturbed by hunger and thirst. At the

[1] See paper by B. G. A. Moynihan, in "Brit. Med. Jour.," Jan. 31, 1903.

end of a week or ten days pancreatinized or peptonized milk is cautiously given by the mouth. According to some, nutritive enemata should now be substituted for saline enemata and be given for a few days before stomach feeding is instituted. After rectal enemata (saline and nutritive) have been abandoned the patient is placed on a very bland diet, preferably pancreatinized milk, and lavage is given twice a day. The value of introducing food into the rectum is, to say the least, doubtful. Saline fluid and certain drugs are absorbed from the rectum, but little if any protein matter is absorbed. Investigators are now seeking for some form of digested protein matter that will be absorbed. Protein material when in the rectum is not acted upon by the enzymes necessary for its absorption, it undergoes putrefaction, causes irritation, and sets free toxic alkaloids which are absorbed. It is suggested that digested albumin and fat and grape-sugar may be absorbed. In some cases of ulcer Carlsbad salts are given by the mouth (Ziemssen); in others, silver nitrate with extract of belladonna, bismuth subnitrate, or oxalate of cerium. If pain is severe, opium may be required. Many cases are apparently cured by medical treatment. Russel's statistics show that 40 per cent. of cases were reported cured under medical treatment, but no one knows how many of those reported cured again gave evidence of the disease or later perished because of hemorrhage or perforation. Further, 18 per cent. of the 500 London Hospital cases under medical treatment died and 42 per cent. were not cured when discharged. Out of the supposed 40 per cent. of cures many later undoubtedly developed or will develop renewed symptoms and perhaps fatal conditions.

*Surgical.*—The exact curative value of operation is not settled. Kronlein's clinic claims 85 per cent. cures, Von Eiselsberg's clinic but 52 per cent. of cures and 15 per cent. of improvements. The nearer the ulcer is to the pylorus, the better the chance of cure. Following the Mayos, we would not advise surgical treatment in acute ulcer unless complicated by hemorrhage, perforation, or obstruction; or in chronic ulcer, until careful medical treatment has failed. Operation is indicated for chronic ulcer when a mechanical cause is responsible for retention and stagnation of stomach contents, and in certain cases of hemorrhage. Operation is also indicated in chronic ulcer with frequent exacerbations, but the surgeon should be very chary of operating upon neurotic women with gastroptosis unless, of course, there is a positive indication (Wm. J. Mayo, in "Jour. Am. Med. Assoc.," Oct. 21, 1905).

In a chronic ulcer if the patient grows worse in spite of careful dietetic and medical treatment, if hemorrhage has been profuse or if there have been frequent distinct hemorrhages, if the pain is violent, or if tenderness is marked, open the abdomen and inspect the stomach. An ulcer with indurated edges is easily found. The form, called by the Mayos the non-indurated ulcer, gives no evidence or little evidence of its existence when the outer coat of the stomach is felt and inspected (Wm. J. Mayo, Ibid.). Even when the stomach is opened, no ulcer may be found. According to Mikulicz, in some mucous ulcers there is a very little thickening, and, according to Moynihan, the mucous coat may be a little adherent to the muscular coat, so that it does not slide easily. An enlarged gland in a portion of the omentum may be a sign of ulcer (Lund). An indurated ulcer may be removed by an elliptical incision in the long axis of the stomach, the coats being sutured by the usual method, and gastro-enterostomy being also performed. In ulcer of the pylorus with great thickening we may excise the pylorus, close both the duodenal and stomach openings, and perform posterior gastro-enterostomy. In some cases gastro-enterostomy alone leads to the cure of chronic ulcer. The Heineke-Mikulicz operation is not satisfactory in ulcer. Finney's gastroduodenostomy is not advisable if there is an unhealed ulcer, because food still passes over the ulcer after its performance.

Wm. J. Mayo ("Annals of Surgery," 1910) has described a transgastric method for excising ulcers of the posterior wall which are adherent to the pancreas. The excision passes through pancreatic tissue. Bleeding is arrested by suture-ligatures. The wound in the pancreas is not sutured, but is closed by a mobilized portion of gastrohepatic or of gastrocolic omentum.

*Operation for Gastrorrhagia (Hemorrhage from the Stomach).*—Rydygier proposed in 1882 to operate for hemorrhage. The first operation was done by Mikulicz in 1887, and the first successful operation was reported by Roux in 1893.

In acute and violent hemorrhage threatening life the proper course to pursue is somewhat uncertain. It is not proper to operate if there has been but one hemorrhage, because the chances are that the bleeding will not be repeated. Again, the chance of arresting such a hemorrhage by operation is, on the whole, poor. The danger of waiting after one hemorrhage is not so great as the danger of immediately operating, because collapse antagonizes renewed hemorrhage, but adds enormously to the risk of an operation. In over 90 per cent. of cases the hemorrhage ceases spontaneously. In over 18 per cent. of those dying of hemorrhage death is so rapid that operation is impossible (Savariaud). If the bleeding is from a distinct ulcer, we may succeed in excising the ulcer or in ligating the bleeding-point. Roux, of Lausanne, saved a patient by excising an ulcer and ligating the bleeding coronary artery on each side of it. As a rule, however, the bleeding is not from a distinct point, but from a multitude of excoriations. In the light of our present knowledge we may lay down the following rule: Do not operate for one acute hemorrhage. Simply bring about reaction by gentle means, let the patient take bits of ice, and give suprarenal extract by the stomach. If the bleeding recurs once or twice in comparatively trivial amounts, do not operate; but if it recurs violently, we should advise operation. In cases of ulcer in which bleeding in small amounts persists, operation is indicated. In operating for a severe hemorrhage the surgeon opens the abdomen while hot salt solution is being thrown into a vein, The stomach is opened, the clots washed out, and a search made for the source of the blood. If it is found that the blood comes from an area of ulceration, this area may be extirpated, ligated, or cauterized with the thermocautery. Some advise surrounding it with a purse-string suture. Others, notably Moynihan, simply perform gastro-enterostomy, which is of service by draining and giving rest to the dilated stomach, the hemorrhage being perhaps arrested by contraction of the gastric walls and the rest secured preventing the detachment of hemostatic clot. Gastro-enterostomy is of most service in ulcer near the pylorus and in duodenal ulcer. If the ulcer is well above the pylorus it should be excised if possible. As a rule, it will be found that the vessels entering the ulcer are varicose. Excision is indicated because of this varicosity. If excision is impossible "the main blood-vessels leading into the ulcer should be ligated and the peritoneum and muscular coats drawn over it" (Wm. J. Mayo, in "Surg., Gynec., and Obstet.," May, 1908). If it is found that the bleeding comes from a multitude of excoriations and that the stomach is, as Moynihan expresses it, "weeping blood," we can do nothing but gastro-enterostomy, which in such a condition is of uncertain value.

*Operation for Perforation.*—In acute and subacute perforation operate at once, having all proper means taken to bring about reaction from shock, while the abdomen is being sterilized and while ether is being administered (hot saline enemata, external heat, atropin hypodermatically, etc.). As a matter of fact, shock is seldom so profound as to cause us to hesitate about operating. I formerly advised to wait until reaction was established before operating. I now believe such advice erroneous. To delay after an acute perforation is to wait for what may never come. Open the abdomen at the point of greatest

tenderness, or, if there is no such point, open it in the epigastric region, a little to the right of the midline. When the abdomen is opened there may be an escape of odorless gas, and food or fluid may be discovered in the peritoneal cavity. The perforation is sought for and is usually found in the anterior wall. When found, it should be buried and overlaid by stomach wall, a portion of which must be inverted by two layers of Halsted sutures. I do not believe that excision or paring the edges is necessary or desirable in a case of perforated ulcer. If it is too large to close, stitch a plug of omentum into the opening or insert a tube and create a temporary gastrostomy. If no perforation is found on the anterior wall, make an opening into the lesser peritoneal cavity through the gastrocolic omentum, explore the posterior wall, and close and cover any perforation found. In addition to closing the perforation, gastro-enterostomy is theoretically indicated in order to drain the viscus, give it rest, and lessen the tendency to recurrence of ulceration. But, as a matter of fact, such ulcers seldom return. By the time the perforation has been closed the patient is perhaps too severely shocked to render such an additional operation justifiable, and I agree with Gibbon that such an operation should be performed only when there are multiple ulcers or when there is pyloric constriction (John H. Gibbon, in paper before the Tri-State Med. Assoc. of Virginia and the Carolinas, Feb. 23, 24, 1904). After closing the perforation the abdominal cavity is irrigated with hot salt solution, and the space between the liver and diaphragm is sponged out with a gauze pad wet with hot salt solution. If the case is operated on many hours after the perforation, or if the peritoneum was badly soiled, drainage *must* be used, but even in other cases it is safest to use it. Drainage is obtained by means of a cigarette drain or a piece of gauze passed to the suture line in the stomach. In cases with much extravasation, especially if the extravasation has reached the pelvis, a suprapubic opening is made and a tube inserted. After the patient has reacted from the shock of the operation he should be placed in a semi-erect position to direct the flow of infective material to the pelvis, and continuous proctoclysis should be employed as in peritonitis (see page 1024). The treatment of chronic perforation is the treatment of perigastric abscess, and consists of incision and drainage. Of late, a number of cases of acute and subacute perforation have been successfully operated upon. Moynihan estimates that 35–40 per cent. of acute perforations recover after operation. T. Crisp English ("Lancet," Nov. 28, 1903) reported 42 consecutive gastric perforations operated on in St. George's Hospital; 22 recovered.

**Cicatricial stenosis of the orifices of the stomach** results from the healing of an ulcer, the swallowing of a corrosive substance, or traumatism from a foreign body. Constriction of the *cardiac orifice* is indicated by gradually increasing difficulty in swallowing. After a time the esophagus above the stricture dilates or pouches; the fluid food passes into the stomach, but the solid food lodges in the esophageal pouch and is soon regurgitated. The site of the stricture is located by a bougie, and by having the patient swallow while auscultating over the esophagus and cardiac end of the stomach. If the constriction be malignant, the patient will be found to be beyond middle life, the vomit is occasionally bloody, emaciation is rapid and decided, and occasionally the supraclavicular glands are enlarged. A tumor of the cardiac end of the stomach can seldom be palpated. If the constriction be cicatricial, the history will indicate the cause. Constriction of the *pyloric orifice* causes retention of food and dilatation of the stomach. Dyspeptic symptoms will be found to have been long present. A tube passed into the stomach permits of the injection of fluid so as to fill the stomach. When the fluid runs out it contains portions of undigested food, which was perhaps eaten days before, and measurement of the liquid shows that the capacity of the stomach is enormously in-

creased. If hydrogen be forced through the tube, the outline of the distended stomach is at once made clear. The usual method of distending the stomach is by a Seidlitz powder: two solutions are made; the bicarbonate solution is swallowed at once, and the tartaric solution is taken afterward in small amounts at a time. Percussion over the distended stomach indicates the size of the viscus. It is well to remember that when gastric ulcer exists dilatation of the stomach can occur without cicatricial stenosis. The cause in such a case is pyloric spasm, or perhaps the atonic condition which may result from anemia and neurasthenia.

In malignant disease of the pylorus a tumor may often be palpated; there are tenderness and considerable persistent pain, great cachexia and emaciation, absence of free hydrochloric acid from the gastric juice, diminution of red corpuscles and hemoglobin, and perhaps no increase of white corpuscles after a full meal. There is sometimes enlargement of the supraclavicular glands. Vomiting of bloody fluid occurs in 40 per cent. of the malignant cases. The use of the *x*-rays after a bismuth meal is a valuable aid in diagnosticating pyloric constriction. The diagnosis of cardiac constriction is discussed in the section on Stricture of the Esophagus. In cicatricial stenosis of the pylorus there may be paroxysms of pain, there is no tenderness, emaciation is not so early in onset or so rapid in progress, and the supraclavicular glands are never enlarged. Vomiting occurs, but the ejected matter is not bloody.

**Treatment.**—Cicatricial cardiac stenosis requires dilatation with bougies and the maintenance of the restored caliber. If dilatation from above is unsatisfactory, perform gastrotomy, push a small bougie from the mouth into the stomach, tie a string to the bougie, draw the string through the stricture, use the string as a saw to cut the fibrous bands, pass a full-sized bougie, close the wound in the stomach, and maintain the caliber of the cardiac orifice by the repeated passage of dilating instruments. If no instrument can be passed through the stricture from above, perform a gastrotomy, introduce an instrument from below and pass it into the mouth, tie a string to it, draw the string into the stomach, and use Abbe's string-saw (see page 936). If no instrument can be passed from below, convert the gastrotomy into a gastrostomy. In malignant stenosis of the cardia, gastrostomy, if performed at all, should be performed early. Jejunostomy is a better operation. Cicatricial pyloric stenosis was formerly treated by gastrotomy and digital divulsion of the stricture (*Loreta's operation*); but this operation is obsolete, experience having shown that recontraction is inevitable. Pyloroplasty was until recently advocated by many surgeons. This is known as the Heineke-Mikulicz operation. In 30 per cent. of the cases the symptoms are not relieved by pyloroplasty, a condition which renders gastro-enterostomy necessary. Mayo points out that in such cases pyloroplasty fails because the pylorus is on a higher level than the gastric pouch, the degenerated muscle of the stomach is unable to lift the food from the pouch to the pylorus, and the symptoms of gastric dilatation and retardation of the passage of food into the duodenum are not relieved. The operation has been generally abandoned. Finney's method of gastroduodenostomy (Figs. 645–648) is a great improvement on pyloroplasty. The opening is large and in a proper position to afford satisfactory drainage. Gastro-enterostomy is the most satisfactory operation in most cases and usually effects a cure. Malignant stenosis is treated by pylorectomy or gastro-enterostomy. (See under these heads respectively.)

**Congenital or Infantile Hypertrophic Stenosis of the Pylorus.**—Osler tells us that the first case was published by an American, Hezekiah Beardsley, in 1778. Hirschsprung, of Denmark, in 1887 published the first modern case (Bunts, "Am. Jour. Med. Sciences," Jan., 1912). Stenosis in adults is almost invariably due to cancer or to ulcer, but in very young children one occa-

sionally meets with a form that is congenital. The history of such a case is that during the first two or three days after birth the child seems in every way normal, but that after several or a number of days or perhaps weeks vomiting suddenly begins—vomiting for which no dietary cause seems responsible, and which persists irrespective of medication. After the stomach has been emptied by vomiting the child seems much relieved, but when, after a time, food is administered, vomiting will begin again, either in a very short time or after an hour or so. It has been noted that the vomited matter in congenital stenosis of the pylorus never contains any bile whatever, for obvious reasons—the pylorus is shut and the bile cannot enter the stomach. A child in this condition receives little or no nourishment, becomes quickly emaciated, and soon dies. Some of these children die in a month; others, in several months, and a few may live for five or six months. It may be possible, in these cases, to palpate a thickened pylorus, and the outlines of the dilated stomach may be made out. Regurgitant vomiting by keeping the stomach empty may prevent dilatation. The most common symptom is gastric peristalsis. In Bunts's table gastric peristalsis was present in 84 per cent., and tumor in 69 per cent. of cases. In true congenital stenosis there is hypertrophy. The circular muscular fiber undergoes great increase from hypertrophy, perhaps with some fibrosis. The mucous mmbrane is hypertrophied and thrown into folds. The opening into the duodenum may be no larger in diameter than a pin and may be totally blocked by folds of mucous membrane. Even when there is comparatively little or no hypertrophy the lumen of the pylorus may be closed by spasm. It is these latter cases which are benefited by medical treatment. Such cases are not instances of congenital hypertrophy and should be classified as pyloric spasm. In congenital hypertrophic stenosis the intestines are very much collapsed, and the child is, of course, much constipated. Cases of pyloric closure have recovered after lavage carried out daily for some weeks and careful breast feeding. Such cases are probably instances of spasm. This plan is only permissible if there is no palpable thickening at the pylorus. A trial should be given this method unless the condition of the patient demands immediate relief. If there is palpable thickening of the pylorus, operation is called for imperatively. The delay in employing surgery in hopes of lavage succeeding must never be so long that the patient emaciates. If the condition does not soon show signs of improvement, operation is indicated. Cases reported cured by medical means may have been instances of pyloric spasm. The operation for this condition is usually gastro-enterostomy. The mortality after the operation is apparently over 50 per cent. It is superior to pyloroplasty because it enables us to at once feed the exhausted child. Bunts ("Amer. Jour. Med. Sci.," Jan., 1912) operated on 7 cases and 4 recovered. Gastro-enterostomy saves the child from starvation and restores the function of the intestinal canal, but the pyloric tumor remains permanently (Scudder, in "Surg., Gynec., and Obstet.," Sept., 1910).

**Perigastric Adhesions.**—That perigastric adhesions are frequently responsible for stomach pain and digestive difficulty is undoubted. Such adhesions often arise in cases of protracted ulceration of the stomach or duodenum. A common cause of perigastric adhesions is gall-stone disease. Tuberculous peritonitis causes dense adhesions. In some cases adhesions are traumatic, in some are due to syphilis, in many the cause is uncertain (Fred. D. Bird, "Intercolonial Med. Jour. of Australia," Dec. 20, 1900). Adhesions may cause blocking or kinking of the pylorus, or may glue the stomach to the parietal peritoneum or to some adjacent viscus. In Fenwick's table of 123 cases he finds that the adhesions usually cause the stomach to adhere to the pancreas or to the liver. The formation of adhesions in cases of gastric ulcer is, in many instances, conservative, serv-

ing to prevent perforation or to limit extravasation if perforation of the stomach wall occurs.

The **symptoms** are variable. In some cases the adhesions produce little or no trouble; but in the majority of cases they cause definite symptoms, and sometimes the condition becomes one of absolute disablement. The symptoms may be due to blocking of the pylorus, a condition that is followed by gastric dilatation. They may be due to dragging upon the adhesions when the stomach contracts during digestion, or when peristalsis occurs in an adherent piece of intestine.

The usual symptom is pain, frequently of a violent character. The pain comes on in paroxysms, and recurs over and over again, it may be during years. H. Hale White[1] points out that in these cases there is usually some pain persisting, which is now and then increased into violent paroxysms; and that the only other condition that produces persistent pain with violent exacerbations is cancer. In *adhesion-dyspepsia*, however, there is no distinct loss of weight; the condition may exist in youth, as well as in middle age or old age; it is not always increased by taking food, and it very rarely causes death. If there is a history of antecedent gall-stone disease or of ulcer of the stomach, it is possible to make the diagnosis without exploratory operation. Even in other cases the condition may sometimes be diagnosticated, because, although there are these attacks of violent pain, there is no tenderness. In rare cases the adhering and matting together with inflammatory exudate produces a palpable mass. In doubtful cases of chronic and disabling stomach disease an exploratory operation should be performed; if adhesions exist, they will then become manifest.

**Treatment.**—In some cases simply dividing an adhesion effects a cure; in other cases it is necessary to make extensive separation of adherent structures, covering the raw surfaces with omental grafts. In serious adhesions about the pylorus gastro-enterostomy is usually the proper operation.

**Bilocular Stomach (Hour-glass Stomach).**—It is usually stated that some cases are congenital, but the writings of Mayo Robson, Moynihan, and H. L. Paterson cause us to doubt if the condition is ever congenital. Even in the so-called congenital cases ulcers are found, or ulcer scars exist, or ulcer adhesions are demonstrable. The advocates of a congenital origin say that the ulcers are secondary to the narrowing, and that ulceration tends to occur, particularly at the seat of constriction. Beyond doubt, a very great majority, at least, of cases of bilocular stomach result from adhesions produced by the healing of an ulcer. In hour-glass stomach with a large opening between the two sacs there may be no symptoms. When the opening is small the symptoms resemble those of pyloric stenosis. The sac toward the cardia is frequently much dilated.

**Symptoms.**—The diagnosis of cancer is often made. The protracted gastritis may have caused free hydrochloric acid to disappear and acids of fermentation are usually found. The patient vomits from time to time, bringing up food which was eaten a day or two before, proof that food is retained in the stomach and not digested. Occasionally, perhaps, blood is vomited. There is pain and the patient is harassed by foul-smelling eructations. Emaciation becomes pronounced. Cumstom[2] points out that in a thin belly distention of the stomach may make the condition evident; further, that if water is thrown into the stomach, only a part returns, and when the stomach is emptied as much as possible by a tube, a splashing sound can still be elicited in the stomach because the pyloric pouch is not empty. One cause of death is torsion on the axis. A skiagraph taken after a bismuth meal gives diagnostic information of the first importance.

[1] "Lancet," Nov. 30, 1901.

[2] "Med. News," Dec., 1901.

**Treatment.**—The diagnosis becomes certain after exploratory operation, and exploration also enables the surgeon to decide with certainty as to what operation should be performed. Cumstom[1] gives us the following suggestions:

1. In rare cases resect the stricture and suture the pouches.
2. If there is trivial ulceration or a slight scar, do gastroplasty, an operation upon the constriction exactly similar to pyloroplasty.
3. The best operation in most cases is gastrogastrostomy—that is, anastomosis of the cardiac pouch to the pyloric pouch; but this cannot be done if the pyloric pouch is small. Then do gastro-enterostomy.

Other operations are:

4. Gastroduodenostomy.
5. Gastrojejunostomy.
6. Gastrolysis.

In malignant disease resection (partial gastrectomy) is indicated. After gastroplasty recontraction is common, and I do not believe in the operation. Gastro-enterostomy is unsatisfactory. The ordinary operation drains but one pouch. Weir and Foote advised a double gastro-enterostomy, tapping each sac. In most cases gastrogastrostomy followed by gastro-enterostomy is the best procedure.

**Chronic Dilatation of the Stomach.**—A dilated stomach, roughly speaking, is one which can contain more than 1.5 quarts (Ewald). Some few cases of dilatation result directly from atrophy of the muscular coat brought about by drinking quantities of liquid, especially beer; chronic catarrh of the stomach; and conditions such as cancer, tuberculosis, diabetes, etc. The common cause of dilatation is constriction of the pylorus. In order to force food through the pyloric narrowing more force is necessary than is required in a normal state of affairs to cause the food to enter the duodenum, hence the stomach muscle hypertrophies. This muscular hypertrophy is compensatory, and dilatation does not occur so long as the muscle is efficient. But finally the pyloric opening becomes so narrow that compensation fails, the stomach contents accumulate, and the stomach dilates.

**Symptoms of Dilated Stomach.**—There is annoying hunger unless cancer exists. Thirst is complained of. At intervals of a day or two the patient vomits enormous quantities, and portions of food may be identified which were eaten one or more days before. The vomited matter is sour and foul smelling, contains numbers of yeasts, and much fermentative acid. Free hydrochloric acid is often absent. In some cases vomiting occurs two or three hours after each meal. The patient suffers from foul gaseous eructations. There are progressive emaciation, constipation, scantiness of urine; sometimes cramp in the legs, belly, and arms; tetany may occur (see Parathyroid Tetany, page 1244) insomnia is the rule; cardiac palpitation occurs, and there is dyspnea, particularly at night.

**Physical Signs of Dilated Stomach.**—The epigastric region is hollow and the left side of the abdomen is more prominent than the right. The outline of the greater curvature of the stomach can be distinguished. If the stomach contains air, percussion gives a tympanitic note; if it contains fluid, a dull note. When it is partly full of fluid, by altering the position of the patient we can show by percussion that the fluid changes its position. In a doubtful case give a light meal in the evening, and in the morning, before the patient has eaten, introduce a tube and remove any material contained in the stomach. The presence of undigested food points to dilatation.

**To Test the Motor Power of the Stomach.**—*Klemperer's Test.*—Wash out the stomach. Introduce 100 c.c. of olive oil by means of the tube. After two hours withdraw the oil. The stomach cannot absorb oil, and if the

[1] "Med. News," Dec. 7, 1902.

amount withdrawn is subtracted from the amount introduced the difference is the amount which passed the pylorus. If the condition is normal, not more than from 20 to 40 c.c. should be found in the stomach after two hours.

*The Salol Test of Ewald.*—Salol is not decomposed in the stomach, but in the intestine is broken up into phenol and salicylic acid. Salicylic acid is absorbed and salicyluric acid soon appears in the urine. If salol cannot reach the intestine, salicyluric acid will not appear in the urine. If salol reaches the intestine more slowly than normal, salicyluric acid will appear after a longer interval than when there is no pyloric block to retard the emptying of the stomach. In a normal person salicyluric acid is found in the urine in from three-fourths of an hour to an hour after swallowing a dose of salol. In stenosis of the pylorus it appears much later. The test is made as follows: The bladder is emptied and the patient is given three capsules, each containing 5 gr. of salol. The patient is directed to pass water every half-hour, until he has done so four times. Each sample voided is examined for salicyluric acid by adding neutral chlorid of iron. If salicyluric acid is present, a violet color is noted.

**To Test the Absorptive Power of the Stomach.**—The absorptive power of the stomach can be tested by giving the patient a capsule containing 1½ gr. of iodid of potassium. Normally the drug should be found in the saliva in from ten to fifteen minutes. When absorption is deficient, it may not appear for an hour or longer. In order to test for it, moisten starch paper with the saliva and touch the moist paper with a drop of fuming nitric acid. If iodin is present, a blue color develops.

While the diagnosis of dilatation of the stomach can be certainly made, the determination of the cause may require an exploratory operation.

**Treatment.**—Cases not due to pyloric obstruction are much improved by lavage, regulated diet, use of an abdominal belt, electricity, aperients, and other agents called for by symptoms.

In all cases in which there is pyloric obstruction, in many doubtful cases, and in cases in which medical treatment fails, exploratory operation is indicated. In dilatation without pyloric obstruction some surgeons advocate gastroplication. If pyloric obstruction exists, the surgeon may elect to do pylorectomy, pyloroplasty, or gastro-enterostomy, the method selected depending on the condition discovered. If gastroptosis exists, gastropexy or Beyea's operation may be performed.

**Acute Dilatation of the Stomach.**[1]—This condition may suddenly arise in the course of chronic dilatation or when no previous dilatation existed. Its clinical features were described by Brinton in 1859. Hilton Fagge in 1873 furnished us with the first comprehensive description of the signs and symptoms. The cause is uncertain, and is a subject of active investigation at the present time. It is said to be due to degeneration of the gastric muscle in the course of specific fevers, to paresis arising in the course of chronic gastritis, and to the drinking of a quantity of effervescing liquid. If is occasionally a fatal sequence of abdominal operations, particularly operations upon the gall-bladder and bile-ducts. The surgeon sees it in the course of sepsis and during shock from operations in which a general anesthetic was used, and occasionally in cases of spinal curvature.

One set of observers maintains that the condition is brought about by actual constriction of the duodenum, the constricting cause being the root of the mesentery and the superior mesenteric artery and the duodenum being squeezed against the vertebral column (Rokitansky, Albrecht, Robinson, and Kundrat).

[1] See Kelling, in "Archiv. f. klin. Chir.," 1901, lxiv; Albrecht, in "Virchow's Archiv.," 1899, clvi; Conner, in "Am. Jour. Med. Sciences," March, 1907.

Codman believes that normally in man there is more or less tendency to such constriction on standing erect or lying down, and that a trivial increase of the constriction which may be brought about by various causes may completely obstruct the duodenum ("Boston Med. and Surg. Jour.," 1908). Lewis A. Conner collected 18 fatal cases shown by necropsy to be due to mesenteric obstruction ("Am. Jour. Med. Sciences," March, 1907).

Another set of observers asserts that acute dilatation is due to lesion of the nerve-trunks or nerve-centers, resulting in paresis of the muscle of the stomach wall and spasm of the pylorus. The cause of the nerve lesion is variously held to be hyperacidity of gastric secretion, the absorption of the toxins of fermentation, the secretion of chloroform into the stomach, the overdistention of the viscus with ether vapor, or an enormously great secretion of fluid into the stomach.

As Conner (Ibid.) says, the theory of pyloric spasm being causative is untenable because in most cases vomited matter contains bile, and in two-thirds of the cases the duodenum is involved in dilatation.

It seems certain that some cases of gastric dilatation are associated with mesenteric constriction of the duodenum, brought about by the intestines descending into the pelvis. The constriction inaugurates the dilatation and is aggravated by the dilatation. Conner points out that the intestines enter the pelvis as a result of dorsal decubitus, a long mesentery, and a gut nearly empty of gas and feces, and is favored by relaxation of the belly wall ("Jour. Am. Med. Assoc.," March, 1907).

**Symptoms.**—The most frequent and prominent is violent vomiting, usually inaugurating the symptoms and continuing throughout the illness, although occasionally it ceases for some time before death. The amounts vomited are always large and often enormous. The vomitus is thin and of a green or black hue, usually contains bile and sometimes a little blood, but very seldom feces.

In most cases, but not in all, there is epigastric or umbilical pain and tenderness. Distention is the rule. Rigidity is rare. In many cases there is no passage of gas or feces, but in some diarrhea exists. There is great thirst, there may be hiccup, and delirium may arise before death. The temperature is nearly always normal or below normal. There is seldom visible gastric peristalsis (Conner, Ibid.), and splashing sounds are obtainable over the stomach.

Collapse arises early and quickly becomes profound. Tetany occasionally occurs. A case of my own died of acute gastric dilatation after an operation for stone in the kidney. He suddenly developed attacks of violent and profuse vomiting, rapidly went into collapse, lividity developed and hiccup arose, and he died in forty-eight hours.

A case may die in less than twenty-four hours or may die after ten days or more. Conner's group of cases shows a mortality of 72.5 per cent. Fluid cannot reach the small intestine, and as none is absorbed from the stomach and little from the duodenum, the tissues starve for the want of it (Laffer, in "Annals of Surgery," April, 1908). The condition is frequently diagnosticated acute intestinal obstruction or peritonitis from perforation.

**Treatment.**—When the stomach has dilated greatly and when collapse is profound, treatment is usually of no avail. When an early diagnosis is made treatment is often of the greatest value. The stomach must be at once emptied by the use of a tube and the treatment be repeated at intervals of a few hours. Neither food nor drink should be given by the mouth. Saline enemata and perhaps nutritive and stimulating enemata should be given. The patient should at once be placed upon the belly and kept there until the condition abates, in the hope that this posture will relieve duodenal constriction.

Gastro-enterostomy has been employed, and is advocated by Mayo Robson. Byron Robinson had a successful case. I would be disposed to employ it in spite of the reported failures. In a number of cases the stomach has been opened and washed out without benefit. Petit opened the abdomen and discovered a kink at the junction of the duodenum and jejunum; he raised the jejunum and sutured it to the transverse colon and the patient recovered (Conner, "Jour. Amer. Med. Assoc.," March, 1907).

Not until we know definitely the cause or causes of acute dilatation of the stomach will we be able to lay down with precision and accuracy the treatment which is indicated. There is much difference of opinion as to the causation of the condition, and widely different methods of treatment are advocated by various surgeons.

Fig. 602.—Gastroptosis (shown by a skiagraph).

**Gastroptosis** (Fig. 602).—In this condition the stomach has undergone displacement downward, the greater curvature in many cases being but little above the pubic symphysis and the lesser curvature being between the ensiform cartilage and the umbilicus. This condition is far more common in women than in men, and is especially common in women who have had many children. It may be produced by tight lacing and may follow movability of the right kidney, of the liver, or of the spleen. It is often associated with enteroptosis, mobile cecum, and prolapse of the colon, and is particularly prone to arise in the anemic and tuberculous.

**Symptoms.**—There may be no symptoms for a long time, but sooner or later dyspepsia arises because the stomach cannot empty itself. The stomach becomes atonic, its secretions are scanty and altered, and while the viscus may be normal in size or even shrunken, it is usually dilated. The malposition can be made out by percussion when the stomach is distended with air or with fluid, and by the *x*-rays after the patient has drunk a pint of a solution of mucilage of acacia containing subnitrate of bismuth. The bismuth lines the stomach and intercepts the *x*-rays and a radiograph shows the outlines of the stomach and hence its size and position. The pylorus descends to the umbilical region, which it does not do in plain dilatation. In dilatation the pylorus is but slightly lower than normal, but the lower border of the stomach

is notably depressed. In gastroptosis there is often a constriction a short distance from the pylorus due to a kink produced by the sagging.

When a patient with gastroptosis stands erect the bulging is most prominent in the region of the umbilicus and the epigastrium is deepened.

Gastroptosis is not infrequently associated with chlorosis and commonly with neurasthenia.

**Treatment.**—Lavage, regulation of diet, improvement of the general health, the wearing of an abdominal binder, and placing the patient supine for a time after each meal. If medical treatment fails and the condition is producing grave impairment of the general health, it may be necessary to perform a surgical operation. Gastro-enterostomy is advocated by some on the ground that the unpleasant symptoms result from stagnation of gastric contents. Good results have been reported by this plan. The operation of Depage is unphilosophical. Duret's operation is objectionable (see page 1104). Beyea's operation or Ransohoff's method are preferred (see page 1104).

**Chronic Intestinal Stasis.**—This is a term employed by Mr. Arbuthnot Lane to designate such a delay in the passage of material along the gastro-intestinal tract as to permit of the absorption of so much toxin that the body cannot successfully deal with it (Lane's paper before the Derby Med. Soc., Oct. 17, 1911). Defective drainage permits absorption of poison, and absorption of poison is responsible for ill health and the lowering of vital resistance to various bacteria. Lane and his followers are of the opinion that toxemia so induced may cause chronic mastitis, rheumatoid arthritis, gastric ulcer, duodenal ulcer, appendicitis, and, by lowering resistance, may be responsible for progressive tuberculous disease. They believe that the block to drainage is brought about by kinks, and that the kinks, which tend to form in certain situations, are due to bands; in other words, the condition is due to a mechanical cause (Lane, Ibid.). Mayo regards such bands as congenital. Lane holds that faulty feeding in early life causes abnormal distention and pull on the gut, that when the erect posture is assumed the condition is exaggerated by the formation of new peritoneal bands which are formed to resist the dropping of the intestine.

In 1909 Jabez Jackson described a pseudomembrane sometimes seen over the peritoneum of the lower ileum and colon and loosely attached to it. Sometimes it is also attached to parietal peritoneum and thus limits movements of the gut. This thin membrane is known as *Jackson's veil*, and it is probably identical with Lane's bands. Some observers regard Jackson's veil as the result of pericolitis, some as due to infantile colitis, some to chronic colitis, some to appenditits. Isaacs ("New York Med. Jour.," Oct. 26, 1912) points out that this pseudomembrane may be found in other regions than over the colon, and suggests calling the condition *membranous perienteritis*. He believes the membrane is formed because of ulceration or inflammation of the gastro-intestinal tract.

The large intestine plays such a prominent part in toxin absorption that Barclay Smith (quoted in "Brit. Med. Jour.," Dec. 7, 1912), Metchnikoff, Lane, and others put under ban that portion of the gut, regard it as a useless and dangerous encumberance, and would take it out and cast it away.

**Treatment.**—If a band or membrane exists, remove it, and if a raw surface is left, cover it over with peritoneum.

In partial blocking of the beginning of the large intestine or of the lower ileum some perform ileocolostomy. Lane extirpates the colon and fastens the upper end of the divided ileum to the rectum. He does the same operation for rheumatoid arthritis, tuberculous joints, and other conditions—radical steps on a road where so far few have gone more than a short distance.

**Intestinal obstruction** (**ileus** or **enterostenosis**) is a condition in which fecal movement is mechanically impeded or prevented. It may be

either *partial* or *complete*, *acute* or *gradual*. *Acute obstruction* is due to a sudden narrowing or occlusion of the lumen of a portion of the intestine. *Chronic obstruction* is due to a gradual narrowing of the lumen of a portion of the intestine, and it may at any time become acute. If there is not only interference with the passage of the fecal current, but is also obstruction to the blood-current in the wall of the bowel, the condition becomes *strangulation*. The primal cause is mechanical in nature, in most cases a mechanical block. In paralysis of the bowel from peritonitis there is inability of the bowel wall to contract and force the feces onward. In all cases of unrelieved obstruction the nerves are sooner or later damaged and paralysis occurs. In acute obstruction the stagnated intestinal contents become charged with powerful poisons which act most harmfully on the musculature of the intestinal wall, and, after absorption, attack the heart and nervous system. The inaugural shock is due to the production of the block; later the increasing depression is due to absorption of poisons. Gas forms in great amount in the intestine and produces distention. Distention impairs the circulation in the wall of the intestine and embarrasses respiration by pushing up the diaphragm.

**Acute Obstruction.**—The worst forms of this grave condition are due to sudden and absolute blocking of the bowel with strangulation, as when a portion of bowel is caught under a band or in a hernial aperture.

As soon as strangulation occurs there is violent peristalsis. The bowel above the strangulation for a short time vainly lashes itself into effort to force intestinal material by the obstruction. The peristalsis below empties the bowel below the obstruction, leaving it empty and contracted, but not paralyzed.

Peristalsis above the obstruction soon ceases, and the bowel in this region becomes greatly distended with bloody fecal fluid and gas. The putrefaction of the intestinal contents forms great quantities of gas: none of it can pass the obstruction and none of it can be absorbed because of circulatory disturbance in the bowel wall. It is only early in an acute obstruction that the distended bowel above the block is thin. It soon becomes congested and edematous, bleeding may occur from the mucous membrane, and that structure desquamates and may be eroded. When the mucous membrane is desquamated, bacteria pass through the bowel wall and cause peritonitis, or erosions may perforate. Miles ("A System of Surgery," edited by C. C. Choyce) points out that swelling of the gut is greatest when obstruction is in the small intestine because "secretion of a considerable amount of fluid is reflexly stimulated." The distention may ascend all the way to the stomach.

In most cases strangulation at first blocks veins; later, in severe cases, it also blocks arteries. In a very sudden and complete strangulation both arteries and veins are shut off simultaneously. Early in the case, when the veins only are obstructed, the coil of gut is purple, edematous and distended, and bloody serum may pass into the lumen of the bowel and also into the cavity of the peritoneum. Later the arteries are blocked and then gangrene soon occurs. In very acute cases when the arteries and veins are blocked simultaneously gangrene arises promptly. In such a case the strangulated coil is not distended and is gray or greenish in color. When strangulation occurs bacteria soon begin to pass through the walls of the strangulated coil and cause peritonitis. Perforations may take place in the coil, at the seat of constriction, or even in the gut above the block.

**Chronic Obstruction.**—This comes on gradually. A tumor may slowly fill up the lumen of the bowel or a cicatrix or tumor in the bowel wall may constrict more and more. Pressure outside the bowel may be responsible. In regions where the feces are fluid great narrowing can occur without symptoms. When fecal passage is seriously hindered the bowel above the obstruction becomes distended and its muscular wall undergoes hypertrophy. The hyper-

trophy depends on the exercise of greater and greater effort to overcome the obstruction. Miles ("A System of Surgery," edited by C. C. Choyce) states that above the obstruction the bowel becomes thick, "elongated, and tortuous"; that in the small intestine hypertrophy exceeds dilatation, and in the large intestine dilatation exceeds hypertrophy.

The mucous membrane is inflamed because of irritation of retained decomposed material and ulceration may occur. Peri-intestinal suppuration may arise. Gas does not gather above the obstruction because the circulation is still active in the bowel wall and because some gas can still pass naturally. When the narrowed channel in a partially obstructed bowel suddenly and completely closes, acute obstruction arises. When it does the bowel above becomes congested and distended. An active purgative or indigestible food may be responsible for acute obstruction.

**Various Causes of Intestinal Obstruction.—Obstruction by Adhesions** (Fig. 603).—Adhesions result from previous peritonitis. There may be a few adhesions or a multitude of them. A portion of bowel may be bent or twisted by the traction of adhesions or gaseous movement may twist a coil or bend it above an adhesion. The obstruction may be acute or chronic. Even when acute, strangulation is unusual.

**Volvulus** (Fig. 604).—By this term we mean twisting of a loop of bowel upon its mesenteric axis. It is true that under peculiar circumstances the bowel may twist on its own long axis because of adhesions, but such a twist is not a true volvulus. Volvulus may occur in a hernial sac. In rare cases two coils of intestine twist together. Volvulus is not limited to the pelvic colon, but in a very large majority of cases it is that portion of bowel which suffers. The twist may be partial, a complete turn, or even two or three complete turns. This very dangerous condition occurs particularly in adults. A colon loaded with feces predisposes; so does a long mesocolon and a mesocolon with a narrow base, and so may adhesions. Rotation may be caused by straining at stool, by a sudden shift in position, by lifting, by a blow upon the abdomen, or by peristalsis induced by an active purgative. In most cases the twist is tight enough to occlude the blood-supply of the loop. The loop becomes plum colored and edematous, bloody serum flows into the bowel and into the peritoneal cavity, and immense distention of the loop occurs. The colon above the obstruction also distends. Peritonitis occurs early. Perforation may occur and is most apt to take place above the loop.

**Intussusception** (Figs. 605, 606).—By this term we mean the invagination of a portion of bowel wall into the lumen of an adjacent part of the gut. In nearly all cases an upper segment invaginates into the lower. One-third of all cases of obstruction are due to this cause (Treves). In young children it usually causes acute obstruction; in an adult, chronic obstruction, ending perhaps in an acute attack. Most cases of obstruction in children are due to intussusception. Pitt reports that in St. Thomas's Hospital, from 1875 to 1900 inclusive, there were 115 cases of intussusception, and every patient was under fifty years of age. Gibbon's patient was fifty-eight. Rutherford Morrison had a case due to polypus, and the patient was sixty-two years of age. Males are twice as liable as females. During the performance of peristalsis a localized circular constriction forms and the invagination takes place through the constricted area. The great relative frequency of intussusseption in childhood is due to the greater mobility and irritability of the child's bowel (Treves). The irregular and localized spasm is due to bulky or irritant material within the gut, and, according to Rushmore ("Annals of Surgery," August, 1907), the starting-point of invagination is obstruction. Were peristalsis alone the cause the condition would be far more common than it is in the diarrhea of children. There are four chief varieties: the *ileocecal,* in which the ileum and the ileocecal valve pass into the

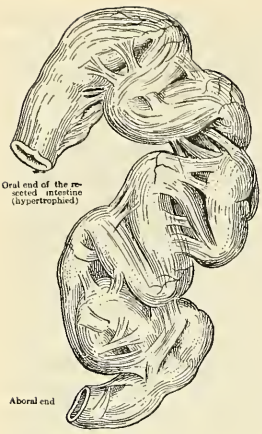



Fig. 603.

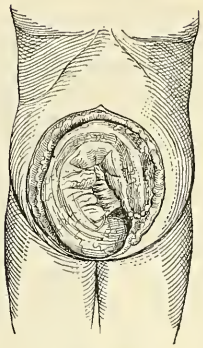
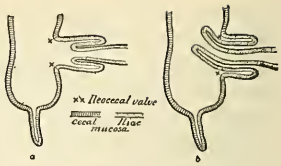


Fig. 604.

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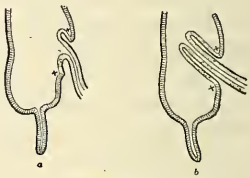

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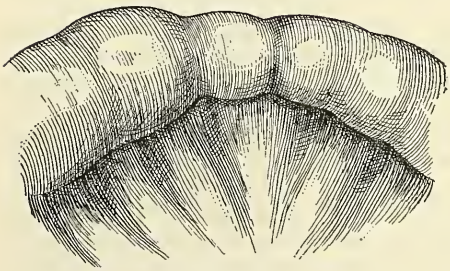


Fig. 607.

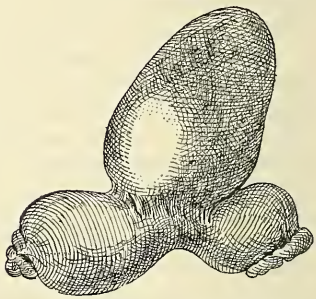


Fig. 608.

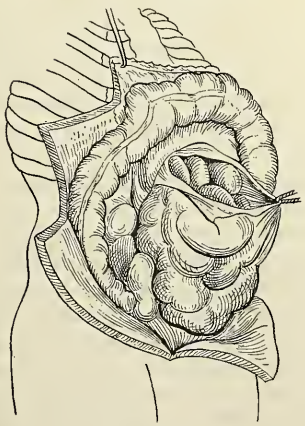


Fig. 609.

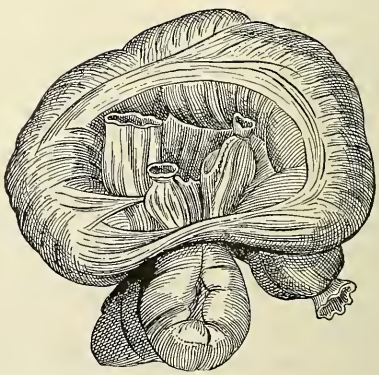


Fig. 610.

Figs. 603–610.—Forms of intestinal obstruction.

Fig. 603.—Stenosed ulcerated tumor of the pylorus; coils of intestine agglutinated by numerous adhesions. Resected intestinal coils (Payr).

Fig. 604.—Volvulus of the sigmoid flexure (Richardson's case).

Fig. 605.—*a*, Invaginatio iliaca; *b*, invaginatio ilia-ileocolica (H. Lorenz).

Fig. 606.—*a*, Prolapsus ilei; *b*, invaginatio ileocolica (H. Lorenz).

Fig. 607.—Obstruction of the jejunum due to gall-stone, showing the contraction of the muscular fibers of the intestine upon the stone, which is smaller in diameter than the lumen of the gut (Mixter's case).

Fig. 608.—Meckel's diverticulum (Bunts).

Fig. 609.—Hernia into the fossa duodenojejunalis (after Cooper).

Fig. 610.—Strangulation by a band. (Warren Museum.)

cecum and colon; the *colic*, in which the large intestine is prolapsed into itself; the *ileal*, in which the small intestine alone is involved; and the *ileocolic*, in which the ileum prolapses through the ileocecal valve. Other forms are *diverticular* (with a diverticulum), *retrograde* (due to reversed peristalsis), *of Meckel's diverticulum*, *ileo-appendiceal*, and *cecal*. The first variety is vastly the most common. In Rushmore's table ("Annals of Surgery," August, 1907) the location was definitely stated in 237 cases: 140 of these were ileocecal and 31 were ileocolic.

The *intussusceptum* consists of the entering tube and the returning layer.

The *intussuscipiens* is the sheath or receiving tube. As the intussusceptum grows longer and longer the mesentery is drawn between the entering tube and the sheath. The mesentery becomes curved and draws the involved intestine toward the back and left side. The dragging, twisting, and squeezing of the mesentery impairs the blood-supply of the intussusception. The invaginated portion, especially the returning tube and apex, becomes congested and swollen, the mucous membrane secretes profusely, and blood passes into the bowel. From this source come the blood and mucus passed by the bowel. An intussusception tends to become irreducible by adhesion of its walls and by engorgement, particularly of the apex. Any intussusception may eventually cause complete obstruction, strangulation may take place, gangrene may occur, peritonitis may arise.

**Foreign Bodies, Gall-stones, and Enteroliths** (Fig. 607).—Foreign bodies include, besides certain substances that have been swallowed, gall-stones and enteroliths or intestinal calculi. Foreign bodies are apt to lodge in the lower portion of the ileum or in the cecum, and they may cause ulceration at the seat of lodgment. If a gall-stone is sufficiently large to cause obstruction, it cannot have passed the duct, but must have ulcerated into the bowel from the gall-bladder. About three-fourths of the cases of gall-stone intestinal obstruction occur in women. The stone is arrested at some point because either local spasm or paralysis of the bowel has developed. It may be arrested high up, but is most apt to be in the lower ileum. A stone under 1 inch in diameter ought to pass. A. W. Mayo Robson ("Brit. Med. Jour.," May 1, 1909) points out that all cases of obstruction due to gall-stones are not the result of mechanical obstruction by a large stone which entered the intestinal canal by ulceration, but that there are three other possible conditions, viz.: Local peritonitis in the gall-bladder region causing paralysis of the bowel; volvulus of the small intestine due to biliary colic or to ulceration of a gall-stone into the gut; obstruction coming on "after the original cause has disappeared" and due to adhesions without or obstruction within the gut.

Enteroliths (fecal concretions) are usually deposits of salts from the feces, especially apt to be formed when the patient has long suffered from catarrhal inflammation of the bowel. The nucleus may be a hair-ball in woman who swallows hair, or the stone of a fruit. A gall-stone may have a concretion form about it. Food residues mixed with salts may constitute concretions. So may insoluble materials taken frequently as medicine or from habit (chalk, bismuth, magnesia). An enterolith increases in size up to a certain point, but seldom becomes enormous.

**Internal Herniæ** (Fig. 609).—These include: (1) retroperitoneal hernia, (2) diaphragmatic hernia.

A hernia may pass into one of the fossæ about the duodenum, one of the fossæ about the cecum, the intersigmoid fossæ, or through the foramen of Winslow into the lesser peritoneal cavity. The appendix may be strangulated in a pericecal fossa and cause obstruction. Diaphragmatic hernia is described on page 1164. An internal hernia may become strangulated just as does an external hernia.

**Obstruction by Bands and Abnormal Openings** (Fig. 610).—The band may be a portion of the great omentum which has taken on an unnatural attachment, a Meckel's diverticulum, an appendix attached at its tip, a broad band or cord-like peritoneal adhesion. The obstruction in these cases is apt to be acute and is commonly accompanied by strangulation. It usually involves the ileum, sometimes the colon. There may be an abnormal opening, congenital or acquired, in the omentum, the mesentery, or the mesocolon. The bowel may slip through such an opening and be caught and strangulated.

Obstruction may take place by *Meckel's diverticulum* (Fig 608) (see page 988), a structure due to persistence of the vitelline or omphalomesenteric duct, coming off from the ileum from 12 to 36 inches above the ileocecal valve, and present in about 2 per cent. of persons. The vitelline duct should be obliterated in the eighth week of fetal life. If it persists the individual possessing it is in constant and serious danger. The mortality of a series of cases of obstruction due to Meckel's diverticulum is enormous. A Meckel's diverticulum usually has no mesentery, is from 3 to 10 inches long, and arises from the convex side of the gut. It may hang free or may be attached to the umbilicus by its tip or by a fibrous cord formed by the obliterated tip. In some cases it remains open at the umbilicus (see page 402). In other cases a cord runs from the umbilicus to the gut or the tip of the diverticulum or is adherent to another portion of the intestine. The diverticulum may become strangulated, may enter a hernial sac, may ulcerate or perforate like an appendix. (W. Sheen, in "Bristol Medico-Chir. Jour.," Dec., 1901, gives an admirable account of "Some Surgical Aspects of Meckel's Diverticulum"; see also article on "Obstruction of the Bowels by Meckel's Diverticulum," by James E. Moore, in "Jour. Am. Med. Assoc.," Oct. 4, 1902, and on "Abdominal Crises Caused by Meckel's Diverticulum," by Miles F. Porter, in "Jour. Am. Med. Assoc.," Sept. 23, 1905.) Strangulation of the diverticulum may take place beneath an adherent appendix, a Fallopian tube, a portion of mesentery, or the pedicle of an ovarian tumor, or it may take place in an omental or a mesenteric aperture. Gangrene, inflammation, or twisting may occur. Obstruction may be due to invagination of the diverticulum into the bowel. H. Tyrrell Gray collected 39 cases of invagination and added 1 of his own, 40 in all ("Annals of Surgery," Dec., 1908).

**Cicatricial Stricture.**—This is not a common cause. I have operated on 1 case due to the scar of a typhoid ulcer. The obstruction occurred gradually and became acute years after the fever. Any healed ulcer may be responsible. Tuberculosis is the most common cause. Syphilis or dysentery may be responsible. A contusion or wound of the bowel may be causal.

**Tumors of the Bowel.**—They are uncommon in the small intestine. Adenoma, lipoma, fibroma, and myoma may occur. They may cause obstruction from blocking or may be responsible for intussusception. Sarcoma is seldom met with, but more often in the small bowel than in the large gut. The patient's general health is much impaired, and a tumor can be palpated before obstruction occurs, and obstruction may never occur. Cancer of the small bowel is more common than benign tumors and sarcoma, but less common than cancer of the large bowel. Cancer of the small intestine is most common in the lower ileum, and arises from columnar cells. It causes constriction and stenosis and usually ulcerates. In the large intestine fibroma, lipoma, adenoma, or myoma may arise. I removed an adenoma from the sigmoid which had produced obstruction. Apparently it has not recurred after four years. A great number of adenomata may be present (*multiple adenoma of Virchow*). Obstruction is rare from innocent tumors. The colon is a common site for cancer. It is usually primary, but may be secondary. It produces chronic

obstruction. The growth is columnar celled, and may be scirrhus, encephaloid, or colloid.

**Obstruction by Tumors, Etc., Outside the Bowel.**—Among the causes of such obstruction are retroflexion or retroversion of the womb, especially in pregnancy, cysts or tumors of the kidneys, ovaries, uterus, etc., movable kidney, and enlarged spleen. Obstruction from any of the above causes takes place in the rectum, the sigmoid flexure, or the colon above the sigmoid.

**Obstruction from Fecal Accumulation.**—Fecal impaction resulting from prolonged constipation is quite common. Obstruction sometimes, but seldom, occurs. When obstruction follows upon impaction it is usually brought about by rotation of a loop of bowel (volvulus) or angulation of a segment, but may be due to abolition of peristalsis because of paralysis. In impaction the fecal mass is usually soft, but may be hard. There may be one large mass or numerous smaller ones. The mass may reach from the rectum even into the transverse colon. It usually begins to form in the pelvic colon (Miles, "A System of Surgery," edited by C. C. Choyce). The weight of such a quantity of feces may cause a packed loop of gut to sink into the pelvis and may stretch and narrow the mesocolon. Ulcerations of the mucous membrane may form (*stercoral ulcers*).

**Postoperative Obstruction.**—Obstruction may come on a day or two after, several or many days after, or weeks or even months after an abdominal operation. We deal here with early obstruction.

Obstruction may be due to a mechanical cause at the seat of operation (adhesion of the bowel to a raw surface, volvulus, catching of the gut under a band, etc.). It may be due to a mechanical cause distant from the seat of operation (bands, adhesions, displacement of the intestine, etc.). It may be due to thrombosis of the mesenteric vessels. Although any one of the above conditions may arise after an operation, there is nothing special and peculiar in it when it does. Such conditions are considered under special headings (bands, adhesions, etc.). Again, postoperative obstruction may be due to the pressure upon the gut of gauze-packing or of a drainage-tube. It may be due to paralysis of a loop of gut because of local sepsis (as after an operation for appendicitis), or paralysis of the intestine from widespread sepsis (general peritonitis).

What we really mean by postoperative obstruction is a condition arising from gaseous distention. Gas begins to accumulate soon after the operation. The patient cannot expel it. He tries, but the straining efforts fail and may burst the belly wound. More and more gas gathers and annoyance becomes torture. The overdistended bowel finally becomes paralyzed and paralytic obstruction occurs. This true postoperative obstruction is particularly apt to arise if evisceration has been practised, if the intestines have been squeezed and handled and not protected from chill, and if the operation were prolonged.

**Embolism or Thrombosis of the Mesenteric Vessels.**—The arteries may be the seat of embolism or thrombosis; the veins, of thrombosis. The section of bowel, large or small, from which the blood is kept undergoes paralysis. Gangrene and peritonitis soon follow. This very fatal condition is one of suddenly arising paralytic obstruction in an individual who has cirrhosis of the liver or valvular disease of the heart, or who has had ulcerative endocarditis.

**Pseudo-obstruction or Spasm of the Intestine.**—In this condition a limited portion of the bowel undergoes spasmodic contraction, which lasts for several hours or even for a day and then passes quickly away. This contraction is usually in the pelvic colon. In many cases a swelling can be made out. It is due to a loop of distended gut and disappears when the patient is under ether. Such a swelling is known as a *phantom tumor*. These attacks occur in neurotic women, especially those with catarrh of the colon. Besides the causes of obstruction above mentioned we should refer to shrinking of the mesentery.

**Symptoms of Acute Obstruction.**—The onset is marked by pain, shock, and vomiting. The tighter the constriction, the more sudden is the attack; the more bowel there is involved, the greater the shock. This element of shock often makes the diagnosis uncertain, and we may suspect abdominal hemorrhage or perforation when the real lesion is strangulation. The pain is violent and continuous, with fierce exacerbations. The continuous pain is due to the constriction; the exacerbations, to the colic of peristalsis. If the small intestine is involved the continuous pain is about the umbilicus. If the large intestine or duodenum is the seat of trouble the continuous pain is located about the lesion. The pains of peristalsis are generalized throughout the abdomen. The higher up the constriction, the tighter it is, and the more bowel there is caught, the greater the pain. Later, when paralysis of the gut occurs or perforation takes place, the pain for a time abates, to recur again with peritonitis. Early in the case there is neither rigidity nor tenderness; as paralysis begins tenderness develops. Early in the case pressure may actually afford some relief. When tenderness exists tapping is more apt to cause pain than is pressure; in peritonitis pressure causes more pain than tapping (Battle). When peritonitis arises, of course tenderness becomes acute and rigidity develops.

Vomiting comes on soon after the development of pain, but does not give any relief. It is accompanied by nausea and violent retching, continues practically without cessation, and whether food and drink are taken or not.

The vomited matter consists first of the contents of the stomach, next of quantities of bilious matter, and finally of brown or yellow stinking fluid, which was long believed to be feces. Vomiting of this character is called *stercoraceous vomiting*. Vomiting of genuine feces may occur, but is rare. In stercoraceous vomiting the fluid gushes up in quantity and by regurgitation rather than efforts of vomiting. This sort of vomiting can occur even when the obstruction is in the upper jejunum or duodenum.

A notable characteristic of obstruction is total inability to pass gas or feces. It is necessary to remember that in the very beginning of the case there may have been a bowel movement, due to peristalsis emptying the intestine below the seat of lesion. A single movement early is no proof that the condition is not obstruction. It is only in cases of strangulation in the pelvic colon that the patient has a strong desire and makes frequent attempts to move his bowels. If an enema is given it is usually retained, or else leaks away without bringing fecal matter with it. Distention from gas (*tympanites*) is always soon present. The lower the obstruction, the more widespread is the distention. Early in the case the abdomen is flat and relaxed, but this condition is very temporary. We may be able to gain information by studying the distention. If both flanks bulge, the block is in the pelvic colon. If the left flank is flat and the right distended, the block is in the colon well above the pelvic colon. If only the small intestine is distended, the block is on the proximal side of the ileocecal valve. As the case progresses distention increases and causes great embarrassment to respiration by pushing up the diaphragm. In thin patients, early in the case, we may occasionally see waves of peristalsis above the obstruction. We can sometimes feel them even when they are unrecognizable by the eye. In many cases of intussusception and in a number of cases of chronic obstruction which have become acute they can be felt.

Cases of intestinal obstruction tend to pass into collapse. This condition is due to the absorption of poisons from the decomposing matter above the obstruction, and is aggravated by vomiting and sweating which abstract quantities of fluid from the blood. When collapse has come on the temperature is subnormal; the extremities cold; the pulse very rapid and weak, often a mere thread or trickle; the respirations are rapid and shallow, and the face *Hippo-*

*cratic* (eyes and temples sunken, nostrils thin and drawn, features pinched, lips blue, skin livid or deadly pale). The amount of urine passed is very small. The higher the obstruction, the less the amount passed. During or after the second day indican is usually present in the urine. It results from putrefaction of proteins and the formation of indol in the intestine. There is leukocytosis, the white count being from 15,000 to 30,000 (Bloodgood, in "Johns Hopkins Hospital Reports," vol. vii).

The mortality from acute obstruction is very high. According to Elsberg, gangrene occurs in 13 per cent. of cases. Seldom can a mass be felt. It can often be felt in intussusception; it can sometimes be palpated when there is a fecal concretion or other foreign body.

**Symptoms of Chronic Obstruction.**—The symptoms come on gradually. There are periods when great constipation exists, alternating with periods of comfort or perhaps with seizures of fluid diarrhea. After a time the attacks of constipation become painful and accompanied, it may be, by vomiting after eating. The patient feels abdominal discomfort most of the time and is much annoyed by gas in the intestine. It is very difficult to expel the gas from below. All the time the constipation is growing more obstinate and the patient is resorting to stronger and stronger purgatives and obtaining less and less response. Finally, a strong purgative may fail utterly, only serving to cause severe colic and perhaps vomiting. It may also cause pain at the seat of the lesion. Patients in this condition may develop attacks of diarrhea, small amounts of feces mixed with mucus being passed. This condition is due to colonic catarrh, is called the "diarrhea of constipation," and affords no mitigation to the discomfort and pain.

During the progress of the case the bowel above the block distends. Because of the hypertrophy of the muscular wall of the gut and of the loss of flesh painful peristalsis (which can be felt and seen) is often noted above the obstruction. This sign is detected far oftener in chronic than in acute obstruction. The distended intestine showing peristaltic contraction is a strong suggestion of chronic obstruction. The painful peristalsis is often productive of rumbling and gurgling noises (*borborygmi*). By noticing the portion of gut distended we may be able to locate the seat of stenosis (see page 984). This method is more valuable in chronic than in acute obstruction. On digital examination of the rectum the rugæ may no longer be felt and the rectum is greatly distended because of paralysis. This is known as *rectal ballooning*.

The above-described condition may cease to intermit or even remit, all the symptoms may grow progressively worse, constant nausea being present, vomiting ensuing on taking food, and the breath being horribly foul. Such a patient is being poisoned to death by putrefactive toxins. He may perish from exhaustion, from perforation, or from peritonitis. At any time during the progress of chronic obstruction acute obstruction may develop. The sudden complete block may be due to a plug of hardened feces or some foreign body. It not unusually follows the taking of a strong purgative. It may be due to volvulus or to bending.

In some cases of chronic obstruction a tumor may be palpated, in some intussusception can be made out, in some there is a history of antecedent peritonitis, in many there are evidences of malignant disease.

**Diagnosis.**—*The determination of the seat of lesion* requires abdominal and rectal examination. An intussusception may sometimes be felt by a finger in the rectum and can often be felt by palpation of the abdomen. Vaginal examination may be demanded. Pain is apt to arise at the seat of obstruction or to radiate from there. Abdominal palpation may detect a tumor. Rectal insufflation of hydrogen may locate the obstruction by causing great distention below it. Entire suppression of urine, early vomiting, absence of

abdominal distention, and rapid collapse mean obstruction in the duodenum or in the jejunum. Early vomiting, a rapidly progressive case, with great distention of the umbilical region, means obstruction of the ileum or the cecum. Distention of the entire abdomen and of the flanks, linked with tenesmus, with less violent symptoms, less rapidity of progress, and less diminution of urine than in the above-cited forms, means obstruction low down in the colon or in the rectum. An old test for obstruction in the adult large intestine is an injection by a fountain-syringe: if 6 quarts can be introduced, there is no obstruction in the large intestine; if less than 4 quarts can be introduced, there is probably obstruction in the large intestine. This test is unreliable. The passage of a sound in the rectum is generally useless and is often unsafe. In many cases the seat of the lesion and the cause of the obstruction can be determined only by exploratory laparotomy.

The *determination of the causative condition* is always difficult and is often impossible. *Intussusception* may arise in child or adult. It is the common cause in children. Intussusception may induce acute or chronic obstruction. In the child acute obstruction is far more common than chronic obstruction. In most cases the child was previously healthy except for previous constipation or diarrhea. The attack begins with a violent seizure of colicky pain and vomiting, but although the patient is pale, there is seldom shock at this stage. The vomiting may be occasionally repeated, but it is not forcible retching and very rarely becomes stercoraceous. The bowel below the obstruction is often emptied in this stage. After a time pain abates or disappears, to recur again and again. Between the seizures of pain the patient often appears to be in good condition, with a normal temperature and an almost normal pulse. Tenesmus soon arises in most cases, bloody mucus in small quantities being passed frequently. The source of the bloody mucus is the squeezed and congested intussusceptum. A sausage-shaped mass can usually soon be felt somewhere over the large intestine. It may be in the right iliac fossa or in the left iliac fossa, but usually over the region of the transverse colon. Osler reports that a mass was palpable in 66 of 93 cases, and in over one-third of these cases it was noted the first day. It has been felt as early as three hours after the onset of pain. It becomes rigid during a pain. It may shift its position hour by hour, and it may disappear after having been obvious. It may be detected only when an anesthetic is given. Rectal examination gives no information in early cases or cases involving only the small bowel. In late cases a mass may perhaps be felt in the rectum. The abdomen is rarely rigid, distended, or tender until late in the case, and at this period there may be shock or sepsis. In chronic intussusception in a child the condition may progress very slowly. The stools contain bloody mucus, there are attacks of abdominal pain, a lump may be felt along the colon or by the rectum, and peristalsis may be visible. If an abdominal lump is detectable it will harden during colic and will change its position from day to day.

Acute intussusception in the adult seldom presents characteristic features unless the sausage-shaped lump can be detected.

Chronic intussusception in an adult causes sudden attacks of vomiting and of abdominal colic which last a short time and pass away as suddenly as they arose. Later they may linger longer. A lump can usually be made out. There may be constipation or mucous diarrhea, and blood may be passed with the mucus. Acute obstruction usually arises.

*Obstruction from adhesions* is chronic obstruction which may become acute. There is a history of antecedent peritonitis. A local distention of the intestine is often noted. In *obstruction from bands* there is a record of antecedent peritonitis, of a traumatism, of a violent effort, or of pelvic pain. The attack is sudden in onset, is fierce in character, and is usually excited by violent

exercise or the taking of food. Vomiting is early and intractable, and it soon becomes stercoraceous; pain is violent; peristalsis above the obstruction is forcible for a time; tympanites and abdominal tenderness appear after the attack has lasted for some little time; obstruction is complete, not even gas being passed; collapse soon arises; no tumor can be detected, and rectal examination is negative. *Volvulus,* which is usually located in the pelvic colon, is far commoner in men than in women, in the middle aged than in those at the extremes of life. It is usually preceded by constipation. The symptoms come on with explosive suddenness and rapidly attain great severity. Obstruction is absolute; vomiting is late and is rarely stercoraceous; no tumor can be detected; rectal examination is negative; abdominal distention is early and pronounced, and may become enormous; peristalsis above the volvulus is seldom discovered; collapse is not so rapid nor so grave as in obstruction from bands and internal hernia. There is severe continuous pain with frequent exacerbations. The pain is about the umbilicus and in the left iliac region and left loin. Tenderness is soon manifest and is ominous, as it means peritonitis. If unrelieved, death will occur in from forty-eight to seventy-two hours. Cases of incomplete obstruction by volvulus are rarities. *Obstruction by a foreign body* may sometimes be inferred from the history of some such body having been swallowed. The obstructing body may occasionally be felt during palpation or may be discovered by the *x*-rays. Abdominal distress may exist for days or weeks before obstruction occurs. Vomiting is late and is rarely severe, but pain, tenderness, and distention are marked. In *obstruction from gall-stones* the victim is elderly and usually stout. There may be a history of one or more attacks of hepatic colic, but often the only history is of what was supposed to be painful chronic indigestion. When the stone is ulcerating into the bowel it causes localized pain and tenderness. As it slowly descends along the intestine it may be arrested again and again, causing attack after attack of blocking. When the gut becomes really blocked pain is early and acute, and vomiting is invariable and usually becomes stercoraceous. There is seldom much tenderness. Rigidity is not common. Shock is seldom present at the onset. Distention is not marked. In rare cases the stone can be palpated.

In *obstruction by an enterolith* the patient may give a history of many attacks of supposed enteritis or colitis, followed after some time by temporary attacks of obstruction. The calculus in rare cases can be palpated. The *x*-rays will show it.

In *obstruction from fecal impaction* the subject is usually of advanced years, and more often a woman than a man. There is a long history of constipation, flatulent indigestion, abdominal annoyance, painful straining at stool, and of repeated attacks of the diarrhea of constipation (small fluid movements, which bring no relief because the solid masses remain agglutinated to the wall of the bowel). The colon is distended by a mass of feces, usually dough-like, but sometimes hard, which is appreciable by palpation. Sometimes hardened or putty-like masses can be touched by a finger in the rectum. In such a subject acute obstruction may at any time arise. It is characterized by severe colic, great distention, and often by vomiting, but the vomiting is not violent and is seldom stercoraceous. Collapse soon begins. *Obstruction from stricture* or *from pressure* comes on acutely after a prolonged period of disturbance, during which period attack after attack of temporary obstruction, complete or partial, took place. A history of blood or pus in the stools suggests *tumor of the bowel;* a history of *blood* or pus having been absent would suggest pressure from without. In *pseudo-obstruction* or *spasm of the bowel* there is sudden apparent obstruction occurring in a neurotic subject, especially a female. It is most apt to arise in a victim of old colitis, or in one who has very recently been subjected to an abdominal operation. There is severe pain, vomiting, distention, and

inability to pass gas or fecal matter. The attack is transitory, lasting a few hours or, at most, a day or two, and ceasing as suddenly as it began. Sometimes a limited swelling can be detected. It is due to a loop of distended bowel and results from a contracted segment of gut. It disappears under ether, and is called a *phantom tumor*.

The presence of an *internal hernia* is not thought of until strangulation takes place and seldom then. In duodenal hernia the patient has long suffered from pain of a colicky character. There is "a circumscribed globular swelling, resembling a movable cyst, except that it is resonant on percussion and yields intestinal sounds on auscultation. Owing to the compression of the inferior mesenteric vein at the neck of the fossa, the patient usually suffers from piles which bleed freely" (Alexander Miles, in "A System of Surgery," edited by C. C. Choyce). When strangulation occurs shock is early and overwhelming, and vomiting is early, violent, and persistent. Obstruction from pericecal or intersigmoid hernia is not diagnosticated as to cause.

In hernia into the foramen of Winslow the pain is violent and epigastric, and in this region there is a swelling which gives a dull note on light and a tympanitic note on deep percussion. Diaphragmatic hernia is considered on page 1164.

*Obstruction due to embolism or thrombosis of the mesenteric vessels* is announced by colicky abdominal pain, but early in the case there is no tenderness, no rigidity, and no distention. Pain in the loin and back is often complained of (McArthur, "Annals of Surg.," vol. xxxiii). Moderate vomiting may occur and there may be blood in the vomitus. There is great restlessness, usually collapse; frequently bloody diarrhea or, at least, bowel washings may be bloody. Cardiac disease can usually be demonstrated and albuminuria is common.

In *true postoperative obstruction* (see page 983) as the pulse mounts the abdomen distends, usually vomiting comes on, and there is an almost continuous regurgitation of brownish putrid fluid. It may cease for an hour or two, when there will be a great gush of the fluid. The regurgitated matter may contain blood. There is seldom pain or rigidity, the temperature is subnormal, the extremities are cold, and the face is Hippocratic (see page 985). The condition resembles acute gastric dilatation. The cause is intestinal paralysis. The symptoms of *postoperative thrombosis of the mesenteric vessels*, according to A. E. Maylard,[1] are as follows: Abdominal pain, perhaps colicky in character, gradual or acute in onset, and, as a rule, constant. Early in the case there is no abdominal tenderness, no distention, and no rigidity. The pulse is rapid, the patient is extremely restless, there may be vomiting, but it is never violent, as in acute obstruction; often there is diarrhea, and sometimes bloody diarrhea. These symptoms become particularly significant if there is cardiac or vascular disease.

*Obstruction from Meckel's diverticulum* is usually acute, but is sometimes chronic, and occurs particularly in young adults and children. It has been stated that other and visible deformities are usually present, but in a study of 69 cases by A. E. Halstead[2] this was true of but 1 case, in which harelip existed. In obstruction from Meckel's diverticulum there is often a history of former mild attacks (Ibid.). Halstead sums up the symptoms as follows: As the obstruction is high up, the abdomen is the shape of an inverted cone; early in the attack there is often local meteorism, especially under the costal arch of the right side, but there is no distention in the flanks. Early, active peristalsis may be visible. The tenderness is just to the right of the umbilicus, on a level with it or below it. In most cases there is early fecal vomiting. Gray ("Annals of Surgery," Dec., 1908) describes invagination of the diverticulum into the bowel. The average age for those attacked is fifteen years. Such cases give a history of previous abdominal crises due to

[1] "Brit. Med. Jour.," Nov. 16, 1901. [2] "Annals of Surgery," April, 1902.

twisting, inflammation, or slight invagination. The acute attack is apt to begin while the patient is active. Gray says that about 30 per cent. of the patients pass blood; that the pain is umbilical and usually very violent; that tenderness is seldom acute unless there is peritonitis; that there may be an abnormal umbilical cicatrix; that in a minority of cases there is a palpable abdominal mass, and that the mortality after operation in acute cases is nearly 60 per cent.

*Differentiation of Intestinal Obstruction from Other Diseases.*—Always examine for a strangulated hernia at every hernial outlet. If obstruction is complicated with an irreducible hernia above the seat of lesion, the hernia, if it contains gut, will always enlarge and become tender because of accumulation of feces. Functional obstruction may attend peritonitis or may follow the reduction of a hernia. In this condition, if peritonitis is absent, there is no pain, no tenderness, no tumor, no tendency to collapse, but simply absolute constipation, distention, and perhaps non-stercoraceous vomiting. Appendicitis with peritonitis may cause symptoms similar to those of obstruction; but there is fever, is a history of pain in the right iliac fossa, and the vomiting is not stercoraceous. Acute pancreatitis produces symptoms so similar to those of intestinal obstruction that a diagnosis cannot always be made (see page 1059). We must consider in the diagnosis perforation of a gastric or duodenal ulcer, rupture of an extra-uterine pregnancy or a pus tube, strangulation of the pedicle of an ovarian tumor, renal colic, and hepatic colic. Poisoning by arsenic or by corrosive sublimate should not be confounded with intestinal obstruction.

**Prognosis.**—Without surgical interference most cases of acute intestinal obstruction die within ten days—usually within seven days. In volvulus death may occur in forty-eight or seventy-two hours. It may occur as early in obstruction by a band, in internal hernia, or mesenteric blocking. Death may be due to shock, to exhaustion, to perforation, to peritonitis, or to obstruction of respiration and circulation by tympanites. Recovery occasionally, but very rarely, happens by the formation of a fistula externally or into another portion of the bowel. In acute obstruction from foreign bodies the obstructing body occasionally passes. Volvulus and strangulation by bands are almost invariably fatal unless an operation is performed. In intussusception recovery occasionally follows the sloughing away of the prolapsed gut, but stricture almost inevitably results from this rare event. Functional obstruction gives a good prognosis. The prognosis of chronic obstruction depends upon the causative lesion. It does not threaten life immediately to anything like the degree that acute obstruction does.

**Treatment.**—In any abdominal case in which the diagnosis is uncertain and the patient is shocked give an enema of brandy and hot water, wrap the patient in blankets, surround him with hot-water bottles, and study the development of symptoms and signs. In half an hour, as a rule, reaction will be brought about and a probable diagnosis may be made (Greig Smith). In acute obstruction it is usually customary to empty the stomach by lavage and to evacuate the rectum by means of copious injections given while the patient is in the knee-chest position. The emptying of the stomach is imperative if stercoraceous vomiting has been going on, for vomiting of a quantity of such material while a patient is taking ether may cause death by drowning, the fluid flowing in enormous quantity into the bronchi. In very severe cases a general anesthetic cannot be given and the belly must be opened under cocain. Hutchinson's method of taxis and massage is uncertain, and is as liable to inflict harm as to confer benefit. Some surgeons apply constant compression to the abdomen by means of straps of adhesive plaster. Puncture of the intestine by an aseptic hypodermatic needle (introduced obliquely)

to relieve gaseous distention is a decidedly dangerous proceeding. The passage of a small tube from the anus as high as possible will empty the colon of gas if no obstruction intervenes. In intussusception it is the custom of some surgeons to give no food by the stomach; administer opium and belladonna to arrest peristalsis, wash out the rectum with copious injections, give an anesthetic, and insufflate hydrogen gas or carbonic-acid gas in order to distend the bowel. Other surgeons treat intussusception by forcing air into the rectum by means of an ordinary bellows, and others inject water by a fountain-syringe, the reservoir being at a height of 3 feet. D'Arcy Power believes in the value of hydrostatic pressure in intussusception in children. He states that the child should be anesthetized and the large intestine filled *gradually* with hot saline fluid, the reservoir not being raised more than 3 feet above the patient. The fluid should be retained for ten minutes. My own feeling is that whereas it may be justifiable to try to reduce by gaseous or hydrostatic pressure during the first twenty-four hours of the attack, early operation, except in newborn infants, gives a better prognosis and is safer and more certain. Without opening the abdomen it is impossible to know the condition of the intestine. Gangrene may occur early (it has been found in less than four hours after the onset of symptoms), and if gangrene exists gaseous distention or hydrostatic pressure will cause death. Rushmore's reported case shows that there may be "no systemic symptoms to indicate the presence of gangrene" ("Annals of Surgery," August, 1907). Another point against conservative treatment is the common uncertainty as to whether complete reduction has been accomplished. Vomiting may continue for hours after genuine reduction and the bowels may not move for some time. Waiting to be sure reduction was accomplished will probably be responsible for death if reduction was not obtained. After the first twenty-four hours it is never justifiable to use gaseous or hydrostatic pressure because of the great risk that ulcer or gangrene may exist. Pressure cannot be accurately regulated, and if the bowel is much damaged, may lead to rupture. If the case is not seen until after the first day, or if injections have been used and have failed, all surgeons believe that laparotomy should be performed.

Frederick Holme Wiggin has made a study of the reported cases of laparotomy for infantile intussusception, and considers that operation done within the first forty-eight hours will give a mortality of 22.2 per cent.[1] (see Operation for Intussusception). In very young infants the mortality of laparotomy is very high, and it is a fair question if on them immediate laparotomy should be advised without a trial of conservative methods if the case is seen during the first twelve or twenty-four hours. The mortality of operation is very large on those under eighteen months of age. But even in those under six months of age Wiggin thinks the mortality after operation is not above 22.2 per cent. if operation is done within forty-eight hours, and my personal feeling is that operation should be the method of treatment even in the case of a young baby.

I agree with Rushmore that operation done during the first twelve hours would greatly reduce the mortality, perhaps to the figures he expects would come from such a practice, viz., 12.5 per cent.

In obstruction of the main mesenteric vessels operation is of no avail. In obstruction of branches it may be possible to resect the involved region of bowel, a region which is found to be gangrenous or at least is becoming so.

In obstruction from fecal impaction use large rectal injections and give small repeated doses of salines or of castor oil. If there are signs of inflammation, do not give cathartics, even in small doses, but give opium and belladonna to arrest vomiting and to relax spasm. Impactions in the

[1] "Med. Record," Jan. 18, 1896.

rectum can be removed by the use of a spoon. In acute intestinal obstruction do not delay, but open the abdomen, if possible, before collapse comes on, and find the cause of the obstruction. If it is a gall-stone or enterolith, try to crush it without opening the intestine; if this fails, push it up a little distance, incise the bowel, remove the stone, and close the incision with Halsted sutures. Pilcher[1] collected 40 cases operated upon for gall-stone obstruction, with 21 deaths. If there is fecal obstruction, break up the masses by pressure and push the fecal plug down without opening the bowel. If there is intussusception, reduce the prolapse and shorten the mesentery; but if reduction is impossible, perform a resection and enterorrhaphy, or make an artificial anus. In volvulus untwist and shorten the mesentery; but if this is impossible, treat as an irreducible invagination. In obstruction from adhesions try to separate them and straighten out the bowel, stitching healthy peritoneum over each raw spot to prevent recurrence. In obstruction by a band, free the loop and, if it is gangrenous, resect or follow the plan of Mikulicz (see page 992). Anastomosis may be necessary. In flexion separate the intestines, remove the flexion by a V-shaped incision, and suture the wound in the bowel. In chronic obstruction it is often advisable to perform an exploratory laparotomy, discover the condition, and determine what is to be done to correct it. Some tumors external to the bowel may be removed. Growths in the bowel wall may be removed by resection of the involved portion of intestine, or an anastomosis may be performed, or it may be necessary to make an artificial anus. In obstruction from Meckel's diverticulum that structure may be found twisted, the gut near it may be kinked or twisted, or the diverticulum may act as a band, the bowel being caught under it or kinked over it. Intussusception of the gut below it sometimes occurs; so does invagination of the mucous membrane of the diverticulum; so does chronic inflammation and cicatricial narrowing of the diverticulum or gut (Halstead). The diverticulum may be gangrenous, perforated, or cystic. In internal hernia the constriction must be divided and the bowel removed from the fossa. Any gangrenous gut should be resected if the patient's condition justifies it, or Mikulicz's plan should be followed (see page 992).

After opening the abdomen the surgeon must be guided by conditions. The diverticulum should be removed, just as the appendix is removed in appendicitis, and complications relating to the gut must be dealt with.

The mortality after operations for acute intestinal obstruction is very high (from 60 to 75 per cent.). If the diagnosis were made earlier, operations would be done earlier and the mortality would be much less. Nine out of 10 of these cases that I see in hospital work are gravely shocked and practically dying on admission. If a patient with obstruction is very gravely shocked, I usually follow Moynihan's plan, of simply opening the bowel and draining it in its most distended coil without any search being made for the lesion. The object is to drain the poisons from the intestine, poisons which are the active agents in killing the patient. The abdomen is opened under cocain, the incision being small. A distended coil of intestine is sutured to the peritoneum about the abdominal incision, every care being taken that the stitches do not penetrate the mucous membrane of the gut (Moynihan). A purse-string suture is now inserted so as to enclose an area of the exposed gut; an incision is made into the gut in this enclosed area, and gas and feces flow out. Paul's glass tube (see Fig. 718) is passed into the gut and the purse-string suture is tied. Instead of Paul's tube we may use a rubber tube sutured as is the tube in Kader's gastrostomy (Elsberg, in "Annals of Surgery," May, 1908). The obstruction is thus temporarily relieved, and if the patient recovers, the causative lesion may be subsequently sought for and attacked. If a fecal fistula follows the enter-

[1] "Med. News," Feb. 8, 1902.

ostomy and refuses to close, it may be closed by operation. If Elsberg's plan is followed a persisting fecal fistula will be rare. My colleague, Professor Francis T. Stewart, has devised a method by which the bowel can be drained without any risk of infection of the peritoneal cavity, a risk which always exists in using Paul's tube. Stewart places a clamp at either extremity of the loop of bowel and surrounds it with gauze. One half of a Murphy button is inserted into the empty loop through a small incision. The other half of the button is squeezed into a rubber tube the diameter of which is somewhat smaller than the flange of the button. The two parts of the button are then clamped, and the clamps are removed from the loop of bowel. The intestine is sutured to the wound margins and the feces drain into a receptacle on the floor. Fig. 611 shows Stewart's operation. In any case of intestinal obstruction if gangrene exists the temptation to do immediate resection is strong. I have done it a number of times with a very large mortality. Of late I have been following the plan of Mikulicz and results have been far better. The gangrenous loop is brought outside of the abdomen, it is fixed parallel to the wound, and enterostomy is performed above it. The loop is dealt with later. If a fecal fistula forms it is subsequently closed by appropriate methods. Postoperative obstruction coming on soon after a surgical operation is often not recognized for a time, and the surgeon will be in doubt as to whether he is dealing with peritonitis or intestinal paresis. When distention becomes evident after an operation we should wash out the stomach with warm salt solution, administer salines in small doses frequently repeated, and employ enemata. Many surgeons give two or three doses of atropin hypodermatically at intervals of two hours. Each dose should be $\frac{1}{200}$ gr. Atropin is given with the idea that it increases peristalsis and contracts blood-vessels. It is probably merely sedative, relaxes spasm, and is useless if strangulation exists. Eserin stimulates the muscular coat of the intestines. There seems no doubt that eserin given soon after an operation tends to prevent distention. A better plan than the administration of atropin is to give hypodermatically $\frac{1}{30}$ gr. of eserin and $\frac{1}{40}$ gr. of strychnin every hour until four doses have been taken. During fifteen minutes of this period carry the heated Paquelin cautery to and fro over the abdomen, not touching the skin, but near enough to it to

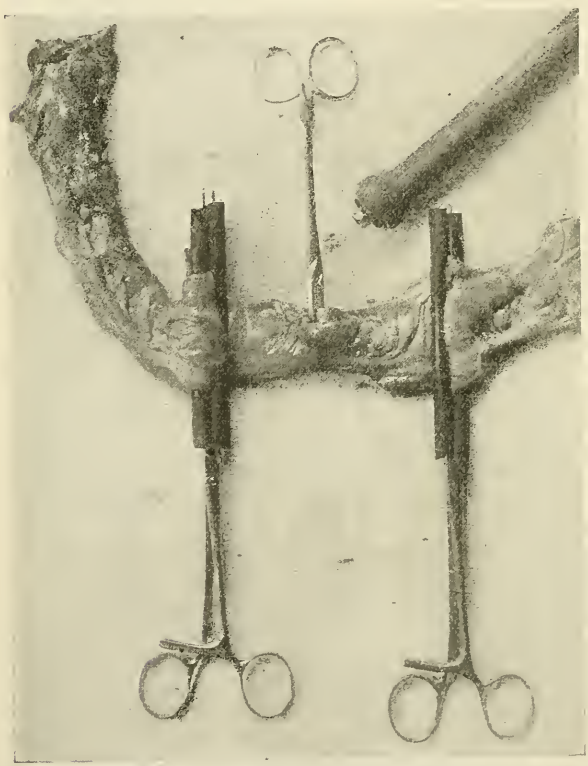


Fig. 611.—Stewart's method of enterostomy.

cause distinct reddening. The intestinal tract should be gone over systematically with the cautery, first, the stomach and small intestine, then the large intestine. Pituitrin is an extract of the posterior lobe of the pituitary gland. When given intravenously or subcutaneously it raises blood-pressure, is a diuretic, and stimulates involuntary muscular fiber. It is useful in cases of flatulent distention. It comes in ampoules each of which contains 1 c.c. The dose is one to two ampoules, repeated in two hours. If these measures are not soon followed by the passage of flatus or feces, open the abdomen; never wait for the advent of stercoraceous vomiting.

**Fecal Fistula and Artificial Anus.**—A fecal fistula is an abnormal opening in the intestine through which gas or a portion of the feces escape (Fig. 612). If all the intestinal contents escape through the opening, it is called an *artificial anus* (Fig. 613, Senn). A surgeon may make a fistula deliberately (*intentional fistula*). A fistula may be the product of disease or injury (*accidental fistula*). Senn enumerates the following causes of accidental fistula: wounds, injury of the intestines, intestinal ulceration, intestinal strangulation, foreign bodies in the intestinal canal, malignant tumors, actinomycosis, pelvic and abdominal abscess, appendicitis, injury of the bowel during an abdominal operation, the application of ligatures, catching by sutures, and the employment of drainage-tubes.

**Treatment.**—Many fistulæ close spontaneously. This can be hoped for only if the opening is quite small, if the general health of the patient is good, if the cause has passed away, if the fistula is not lined with mucous membrane, and if there is no spur (spur is shown at *a*, Fig. 613). In most cases

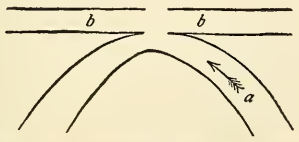



Fig. 612.—Fecal fistula: *a*, Direction of fecal flow; *b*, *b*, belly wall.

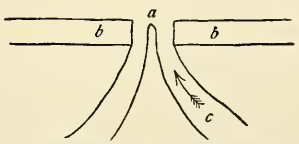



Fig. 613.—Artificial anus, showing spur: *a*, Spur; *b*, *b*, belly wall; *c*, direction of fecal flow.

of fistula not high up it is well to give Nature a chance to effect a cure, and not to be in a hurry to operate. Most fistulæ in the large bowel heal spontaneously. The part is cleansed frequently with peroxid of hydrogen, the patient is kept recumbent, food is given which does not leave much residue, pads of gauze are applied with pressure, and the bowels are kept regular.

If the track is lined with granulations, it may be touched with lunar caustic; if it is lined with mucous membrane, the actual cautery should be applied. Any collection of pus which exists should be drained. If these methods fail, an operation must be performed. The fistula may be sutured by extraperitoneal manipulation (Greig Smith); it may be covered with skin (Dieffenbach); the spur may be removed by means of a clamp, or resection may be performed. In most cases it is best to incise a button of skin around the opening, temporarily suture the fistula, open the peritoneal cavity, deliver the bowel, and suture carefully (Senn's method). In some cases partial exclusion of the fistulous part is necessary, the bowel being divided above the fistula, the end near the fistula sutured, and the other end anastomosed to the bowel below the fistula. In other cases complete exclusion may be performed (see page 1118).

**Ulcer of the Bowel.**—In typhoid fever and in dysentery ulceration occurs. In erysipelas, septicemia, pemphigus, melena neonatorum, and uremia ulceration may occur. An ulcer may be due to tuberculosis or cancer. Most ulcers of the duodenum (see below) are due to the same causes as ulcer of the stomach. In rare cases ulcer of the duodenum results from a sur-

face burn (Curling's ulcer, page 162). An ulcer of the jejunum sometimes develops after the performance of gastrojejunostomy for gastric ulcer (see page 1090). An ulcer may heal and, by causing thickening and constriction, produce chronic intestinal obstruction. It may perforate, causing collapse and subsequent peritonitis.

**Ulcer of the duodenum** may be due to tuberculosis. It may exist in septicemia, erysipelas, melena neonatorum, and uremia. It may be caused by burns. The last named and very rare condition is called *Curling's ulcer*, he having described it in 1841. Moynihan shows in his book on "Duodenal Ulcer" that James Long, of Liverpool, was really the first one to describe duodenal ulceration following burns. He did so in the "London Medical Gazette," 1840. A duodenal ulcer following a burn or scald is "a toxic ulcer, and, therefore, analogous to the ulcer which occurs in septicemia, uremia, typhoid fever, erysipelas, and pemphigus" (Moynihan, Ibid.). It occurs only when the burnt or scalded area has become septic, and septic emboli may be responsible for ulceration. Some believe that the irritant cause is in the bile. Ulcer from a burn is extremely rare to-day, rarer than formerly, if older reports are correct. This is probably due to the modern cleanly treatment of burns and scalds. When we use without qualification the term "ulcer of the duodenum" we mean an ulcer like the ordinary stomach ulcer and which may be regarded as a peptic ulcer of the duodenum.

Septic ulcers are acute. The peptic ulcer is nearly always chronic.

**Peptic Ulcer of the Duodenum.**—The condition was first described by Travers in 1817 ("Duodenal Ulcer," by Sir Berkley G. A. Moynihan). It occurs usually in that portion of the duodenum which is above the opening of the bile-duct; in other words, in the region acted on by the acid fluid from the stomach. In Perry and Shaw's list of 149 cases the first portion was involved in 123, the second portion in 16, and the third and fourth portions in 2; in 8 the ulcers were scattered. Moynihan (Ibid.) says the first portion is involved in 95 per cent. of the cases. The most common seat of ulcer is on the anterior wall of the duodenum, $\frac{1}{2}$ inch below the pylorus. The pylorus is definitely marked for the surgeon by the pyloric vein which runs upward from the greater curvature (Moynihan, Ibid.). The ulcer is often puckered like a scar. Any old ulcer is much indurated. In rare cases there is no induration. Sometimes an extensive area of the duodenum is indurated, sometimes a duodenal pouch or diverticulum is formed. In rare cases an ulcer completely encircles the bowel. Duodenal ulceration is much more common in males than in females in the proportion of 3 or 4 to 1. It may occur at any period of life, from early youth to extreme old age, but is most common between the twenty-fifth and forty-fifth years. Duodenal ulcers are usually single, but may be multiple. It was long taught that gastric ulceration is vastly more common than duodenal ulceration. Some recent observers regard duodenal ulcer as likely to be met with one-seventh as often as gastric ulcer. In this connection Codman's statistics should cause us to revise our estimates. In 3000 autopsies held in the Massachusetts General Hospital an open duodenal ulcer was found in 1 per cent. of the cases. The duodenal ulcers were found twice as often as gastric ulcers (quoted by E. R. McGuire, "Buffalo Med. Jour.," June, 1912). A gastric ulcer may exist with a duodenal ulcer. Murphy sums up the supposed causes into hyperchlorhydria, local infection, embolism and thrombosis, and disturbances of the organs of elimination. An indurated chronic ulcer may exist, and this may heal and produce cicatricial stenosis. The scars of two ulcers may cause double constriction or hour-glass duodenum.

An ulcer may heal and break down many times. A duodenal carcinoma seldom arises from ulcer. What Moynihan calls the "tucked back" ulcer becomes adherent to the liver or posterior wall of the abdomen. An ulcer adhe-

rent to the pancreas may eat into that organ. An ulcer scar may clutch the common bile-duct in the duodenal wall or block the outlet of the ducts in the ampulla of Vater. Perforation is more common, but walling off is more frequent than in gastric ulceration (Wm. J. Mayo, "Med. News," April 16, 1904). In the vast majority of cases the patient gives a history of having suffered at intervals for many years from attacks of flatulent or acid and painful indigestion. A man suffers thus for days or weeks, and then gets apparently perfectly well, but after a variable time the attack returns. The attack comes on from two to six hours after a heavy meal. If he eats his heavy meal at 7 P. M. his attack may usually be expected about 10 o'clock or later. These seizures of "indigestion" may be brought on by worry, overwork, wet feet or chilling of the body, or by eating some indigestible article of diet. They may be caused to disappear by rest, ease of mind, or change of climate. They have been called "hunger-pains" because food for a time distinctly relieves them. A patient learns this and is apt as soon as the annoyance begins to eat some crackers or drink a glass of milk. It was long believed that hunger pain was due to an open pylorus allowing acid material to run into the duodenum, and that food or an alkali caused the pylorus to close. Moynihan, Hertz, and others now reject this explanation because *x*-ray studies show that food begins to leave the stomach at once and that pain begins when about half the food has left the stomach (Moynihan, "Duodenal Ulcer"). It must be that the last portion of food in the stomach is much more acid than the portions which pass out earlier. When the stomach is entirely empty there is no pain; when half empty there is pain.

Arbuthnot Lane would explain the relief of pain by food by invoking a reflex contraction of the duodenum caused by food and serving to empty that portion of the gut. The condition above described may go on for many years, periods of relief alternating with periods of pain, water brash, flatulence, etc. Usually, but by no means always, there is hyperacidity. In some cases acidity is normal; in some, actually subnormal. It is probable that during painful attacks there is hyperacidity. We have stated that the pain occurs from two to six hours after a meal. If it occurs very late the ulcer is posterior; if it occurs earlier than two hours stenosis has begun or the ulcer is adherent to the liver or belly wall (Moynihan, Ibid.).

Hemorrhage occurs in about 40 per cent. of the cases. The blood may be passed by bowel only or it may be passed by bowel and also vomited. In most cases it is passed by bowel and not vomited. When there is a severe hemorrhage the patient first has a peculiarly severe attack of indigestion, he then grows deathly pale, presents the symptoms of internal hemorrhage, and passes a quantity of blood which is first tarry black and later red, and perhaps he also vomits blood. Just as chronic gastric ulcer may be latent, no symptoms ever being observed, so may chronic duodenal ulcer be latent. The pain is located in the epigastric or right hypochondriac region and may last until the next meal. If the digestion of the evening meal is delayed the pain rouses the patient from sleep, but a glass of milk will quiet it. The pain is less severe than is usual in gastric ulcer and in many cases does not radiate to the back, although in others it does radiate to the right scapular region. There may be tenderness on deep pressure in the right hypochondriac region. Symptoms of indigestion are not nearly so marked as in ulcer of the stomach. Vomiting is far less common than in gastric ulcer and it does not relieve pain. The hemorrhage from the bowels may be so profuse as to kill or almost kill the patient. It may be so frequently repeated as to make him profoundly anemic. Unlike the bleeding from gastric ulcer, bleeding from duodenal ulcer is often fatal. In some cases the man bleeds insidiously, perhaps even without knowing it, losing some blood daily, and finally becoming extremely anemic. Probably in every case of duodenal ulcer occult

blood is at times present in the stools. In many cases there is no visible blood in the feces, but the guaiac or aloin tests show occult blood. Vomiting of quantities of blood is much rarer than in gastric ulcer. In very rare cases of duodenal ulcer the first symptom is perforation. I have operated on one such case. In other cases the first symptom is hemorrhage, or is due to stenosis or some other complication. Examination by *x*-rays after the taking of a meal containing bismuth shows, if there is ulcer, greatly increased activity of the stomach. In some cases stenosis is demonstrated, in some a pseudodiverticulum. Moynihan[1] mentions the following complications: severe hemorrhage; perforation; periduodenitis; cancer, and cicatricial contraction involving the bile-duct.

*Acute perforation* is more common than we once thought. Perforation is usually on the anterior wall. It is much less common on the posterior wall. It is extremely rare on the superior wall and practically never occurs on the inferior wall. Moynihan gathered 49 cases from literature and added 2 of his own. In the great majority of cases perforation of the duodenum cannot be differentiated from perforation of the stomach by a study of the symptoms. In some cases the symptoms resemble appendicitis. In most cases there is a sudden onset of violent abdominal pain, followed by vomiting, shock, rapid pulse, tenderness of the epigastric or right hypochondriac region, and board-like rigidity of the upper abdomen. Profound shock is rare. Often shock is trivial. Even if shock is severe the patient usually reacts after a few hours. Sheild's case got better in four hours and walked some distance to the hospital.[2] Lucy's case got better a short time after the onset, walked home, and attended to a horse, but then became rapidly worse. The improvement is apparent, not real, and is only temporary. The symptoms quickly become worse, and when they become worse, besides the pain, tenderness and rapid pulse, there will be occasional vomiting, rigidity of the abdomen, usually an elevated or normal temperature, and possibly diminution of the area of liver-dulness. Just as in stomach ulcer, there may be acute, subacute, or chronic perforation.

**Treatment.**—In duodenal ulcer the risks of serious hemorrhage and of perforation are much greater than in gastric ulcer, and operation should always be recommended if the diagnosis is made. We used to say operate only if the symptoms are not amended by rigid diet and medication; if severe hemorrhage occurs or if cicatricial contraction interferes with the passage of food through the bowel or bile into the duodenum. Moynihan refers to 4 cases of chronic ulcer operated upon, and all recovered. In some cases excision is practised; in others, excision with gastrojejunostomy; in still others, gastrojejunostomy alone.

If grave hemorrhage occurs and is repeated the surgeon should open the abdomen, ligate the bleeding vessels, bring the outer coats of the bowel together over the indurated area, and perform posterior gastrojejunostomy (Wm. J. Mayo, in "Surg., Gynec., and Obstet.," May, 1908). In such a bleeding ulcer the vessels entering it are usually varicose.

In perforation operation is performed, as in gastric ulcer, as soon as possible. In these cases, as in perforated gastric ulcer, I believe operation should be immediate and that we should not wait for a possible reaction from shock. Personally, I do not practise excision of the ulcer, as I believe that closure is just as permanently useful and is safer. The ulcer is inverted by two rows of silk sutures applied in a vertical line or as purse-string sutures. If the gap of the perforation is huge it may be impossible to close it by inversion. It may then be closed by the gall-bladder (as was done by Downes) or by an omental plug. Failing in this, a gauze tampon may be used or a duodenal fistula can be formed, a rubber catheter being inserted in the duodenum and

[1] "Lancet," Dec. 14, 1901. [2] Ibid., March 29, 1902.

wrapped about with a plug of omentum (Eliot, Corscaden, and Jamieson, in "Annals of Surgery," May, 1912). Then gastrojejunostomy should be done. Gastrojejunostomy is imperative if the suturing has narrowed the duodenum. It is valuable in any case because it removes irritants from the scar, allows of early administration of food, favors healing, and antagonizes recurrence. Some surgeons do not drain, but I feel it safer to drain. Sir Berkeley G. A. Moynihan[1] gathered 49 operations for perforated ulcer, with 8 recoveries. Mr. T. Crisp English reports 8 operations for perforation of duodenal ulcers, with 2 recoveries ("Lancet," Nov. 28, 1903). During 1912 in the Mayo clinic 6 cases of acute perforating ulcer were treated by suture and gastro-enterostomy. There was 1 death. In perforated duodenal ulcer the extravasated fluid is apt to flow into the right iliac region. If an erroneous diagnosis of appendicitis was made, an opening in the right iliac region, by giving vent to this fluid, might for a time confirm the surgeon in error, but the character of the fluid should make evident the condition of affairs.

In subacute perforation we may separate adhesions, find and close the perforation, and perform gastrojejunostomy, or follow Lund's advice ("Boston Med. and Surg. Jour.," 1905) and do gastrojejunostomy alone. Lund maintains that as the perforation is walled off it is not necessary to open it up in order that we may close it. The treatment for chronic perforation is that for an abscess about the duodenum. In every operation for duodenal ulcer Moynihan examines the appendix and often finds it diseased ("Lancet," Jan. 6, 1912).

**Ulcer of the Jejunum After Gastro-enterostomy.**—(See page 1090.)

**Perforated Typhoid Ulcer.**—Perforation occurs in 2 or possibly 3 cases out of 100. About 70 per cent. of perforations occur in the second, third, or fourth week. Perforation in a typhoid ulcer is usually effected rapidly, a large opening is formed, and a considerable quantity of fecal matter is passed into the peritoneal cavity. In a few perforations very little fluid escapes. Severe pain and a nervous chill indicate that perforation is occurring or has occurred. Some maintain that the two above-named symptoms associated with marked leukocytosis indicate that perforation is about to occur, and they call this stage the *preperforative* stage. That distinct symptoms may in some cases point to impending perforation is, I believe, true, and in 1 case I operated on the conviction and found two areas almost perforated. In most cases, however, I do not believe that there is a distinct preperforative stage, but the perforation exists when the symptoms are first noted. The conviction that perforation was occurring would be strengthened by a progressive increase in the leukocyte count. It is to be remembered, however, that the leukocyte count is increased by sweating, cold bathing, vomiting, hemorrhage, severe diarrhea, or some positive complication. When perforation occurs, violent pain develops. As a rule, there are tenderness, rapid pulse, costal respiration, abdominal rigidity, vomiting, and shock. Usually there is temporary reaction from shock, the subnormal temperature giving way to a normal or to an elevated temperature. The vomiting in some cases becomes stercoraceous. There is constipation and sometimes dulness on percussing the flanks. The face is Hippocratic. The patient may die of the preliminary shock or may react and die subsequently of toxemia. In a few hours after perforation distinct leukocytosis may be observed, but it may never take place at all. Even when leukocytosis arises, it may disappear as peritoneal infection spreads and systemic poisoning deepens. Le Conte points out that rupture of a mesenteric gland simulates intestinal perforation.

**Treatment.**—In 1884 Leyden suggested operation, and in the same year Mikulicz obtained the first operative success. Before Mikulicz's paper was

[1] "Lancet," Dec. 14, 1901.

published my colleague, Dr. James C. Wilson, published a paper in which he advocated operation. Death is practically certain without operation. Operation should save at least one-fourth of the cases. It should be done at once, proper means being adopted to combat shock. In many cases a general anesthetic should not be given, but a local anesthetic should be employed. The incision should be made in the right iliac region and the colon should be first located and then the end of the ileum. By locating the colon we obtain a fixed point from which to begin our search for perforations, and by opening the abdomen in the right iliac region we come down at once on to the perforated gut in the vast majority of cases. When a perforation is found, it should be inverted by two layers of Halsted sutures. It is not wise to excise the ulcer. If the bowel is very badly damaged, resection can be considered, but it is usually wiser to make a temporary artificial anus. In some cases the perforation can be used as the anus, a tube being inserted, or the bowel being stitched to the skin. After finding a perforation and closing it, examine to see if there are others. Close every perforation, and if a point is found where the thinning of the bowel wall indicates that perforation is liable to occur, protect this point by inverting the area of ulceration by sutures. Cleanse the peritoneum by flushing with hot salt solution. Leave the wound open, insert strands of iodoform gauze, and establish tubular suprapubic drainage. Elevate the patient a little in bed and employ continuous proctoclysis of salt solution. I have operated 10 times for typhoid perforation, with 3 recoveries; 3 cases died of shock. In 1 case the perforation was not found, but was discovered postmortem in the hepatic flexure of the colon, the gall-bladder being responsible for the ulcer of the bowel. One case improved greatly, lived for eight days, developed another perforation, and died of shock. The necropsy showed that the sutured perforation was soundly closed. One case, a young man, brought to me by Dr. Godfrey, was operated upon twenty-four hours after perforation. There was one perforation in the ileum and considerable fecal extravasation. The opening was large and the stitches would not hold. The 6 inches of bowel between the ulcer and the ileocecal valve presented several ulcers almost perforated. The patient was too weak for a resection. After cleansing the abdomen an artificial anus was made proximal to the perforation. The patient recovered and subsequently the anus was successfully abolished by resection. In another case, that of a young woman, on opening the abdomen violent appendicitis was found, the appendix being swathed in lymph and gangrenous. The appendix was removed. Search showed a perforation in a loop of gut 2 feet from the ileocecal valve. There was considerable extravasation. The perforation was closed. The peritoneum was cleansed, drainage was inserted, and the patient recovered. Cultures from the appendix and from the peritoneal cavity showed only the colon bacillus. In a third case, that of a young woman, impending perforation was diagnosticated by Dr. Kalteyer because of pain, tenderness, some rigidity, and definite and increasing leukocytosis. Two ulcers almost but not quite perforated were found. They were covered over by the use of inversion sutures, the wound was closed without drainage, and recovery followed. Culture from the peritoneal cavity was negative. These 3 successful cases were operated upon in the Jefferson College Hospital. Harte and Ashhurst collected the cases operated upon up to January, 1903. There were 362 cases, with a mortality of 74.03 per cent. Dr. F. D. Patterson ("Am. Jour. Med. Sciences," May, 1909) collected 369 cases occurring since the paper of Harte and Ashhurst. Of these cases 242 died, a mortality of 65.58 per cent. The perforations were located as follows: stomach, 1; jejunum, 1; Meckel's diverticulum, 2; ileum, 279; cecum, 5; appendix, 15; colon, 12; not stated, 54.

**Primary Intestinal Tuberculosis.**—Although intestinal tuberculosis is common in patients with chronic pulmonary tuberculosis, primary intestinal tuberculosis is a rare condition. The exact propriety of rigidly regarding such cases as *primary* is doubtful. Kocher's cases (reported before the Swiss Medical Congress in 1892) came from tuberculous stock, and suffered in infancy from enlarged glands, pleurisy, or bronchitis, and that surgeon says that, in all probability, there had for some time been somewhere in the body a latent tuberculous focus, and from this focus came the bacteria which attacked the intestine. Intestinal tuberculosis, in the victims of phthisis, begins with the formation of multiple ulcers, due to swallowing tuberculous sputum. Primary intestinal tuberculosis usually begins as one ulcer or several, or even many ulcers in the ileum or perhaps in the cecum. These ulcers when they heal tend to form strictures of the small bowel, seldom of the large. Primary tuberculosis is most common in the ileocecal region. It may exist as an ulcerative process with inflammation and abscess about the cecum or as enormous tumor-like thickening of the cecum. The first form is called by Hartmann *enteroperitoneal tuberculosis*. The second form is called *hyperplastic tuberculosis*. The cecum may be involved alone, but usually a portion of the ileum also suffers. The ulcers cause extensive sloughing. The appendix may be involved. All about the cecum a multitude of adhesions form containing masses of tuberculous matter, which may break into the intestine, through the skin of the abdomen, or in both directions. (See Hartmann, in "Revue de Chirurgie," Feb., 1907.) Tuberculous areas suppurate. In tuberculosis of the small intestine there are bloody diarrhea, colicky pain, emaciation, and weakness. There are very active peristalsis and gurgling. The abdomen may be tender. The bacilli may be found in the stools. As ulcers heal strictures develop and produce the usual symptoms. Peritonitis may arise or death may be due to pulmonary tuberculosis. In primary intestinal tuberculosis the urine is apt to show the diazo-reaction (Kocher).

In hyperplastic (conglomerate) tuberculosis the cecum and lower end of the ileum suffer as a rule. In some cases the colon participates in the process. Great fibromatosis with adiposis occurs simulating tumor and due to reaction of tissue *against infection*. Tuberculosis of the ileocecal region is most common between the ages of twenty and forty. There is seldom demonstrable phthisis.

The enteroperitoneal form of ileocecal tuberculosis simulates appendicitis. At first there may be diarrhea, the liquid stools containing blood. Pain in the right iliac fossa may be the first symptom. In any case pain becomes the permanent symptom. A lump can be palpated. After acute pain subsides the lump persists and increases. There may be attack after attack of pain. The symptoms of abscess arise. Spontaneous opening finally occurs, usually about Poupart's ligament. Several fistulæ may form. Each fistula discharges pus with fecal matter. An abscess may open into the intestine. When it does, pus appears in the stools. Bacilli may be found in the stools. In enteroperitoneal tuberculosis death occurs because of the development of pulmonary tuberculosis or, in a small number of cases, by peritonitis due to rupture of an abscess into the peritoneal cavity (Hartmann, Ibid.). The hyperplastic form develops slowly, the individual complaining of indigestion and annoying feelings in the right iliac fossa. During many months the patient feels at times better and at times worse. Then food begins to disagree radically. Several hours after a meal the patient suffers from colicky pain in the right iliac region and distention of the abdomen. Severe pain simulating appendicitis may occur intermittently. Constipation may be severe or may alternate with diarrhea. Gradually a tumor mass forms, evidences of obstruction become obvious, the patient becomes weak and emaciated. The lungs are involved very late, if at all. In some cases ulceration occurs. Con-

rath's study of 77 cases led him to believe that death occurs in from two and one-half to three years.

**Treatment of Primary Intestinal Tuberculosis.**—In the first stage the proper treatment is excision of ulcerated areas, possibly excision of the cecum. Later, if stricture is causing chronic obstruction, an operation may be performed to give relief. Laparotomy, careful separation of adhesions which are not fused with the gut, and the introduction of iodoform into the peritoneal cavity may prove of value. Hartmann ("Revue de Chirurgie," Feb., 1907) has collected 229 operations for ileocecal tuberculosis, viz., partial excisions of the cecum, resections (with end-to-end anastomosis, side-to-side anastomosis, or end-to-side implantation), resections in two séances, ileocolostomies, unilateral exclusions, bilateral exclusions, and other operations. There were 46 deaths in these 229 operations. He comments on the fact that there were 58 operations with 7 deaths since 1900, a mortality of 12 per cent.

**Perforation in Intestinal Tuberculosis.**—By this term we mean perforation into the peritoneal cavity, not into another segment of gut and not into a mass of adhesions. It is supposed to be a rare accident. Cruice estimates that it occurs in from 1 to 5 per cent. of phthisis cases ("Am. Jour. Med. Sciences," Nov., 1911). It may occur in either the primary or secondary tuberculosis of the gut. It is very rare in ileocecal tuberculosis, commoner in tuberculosis of the small bowel. It causes sudden and severe pain, shock, and usually nausea and vomiting. The abdomen becomes rigid. Distention soon occurs. Death may be due to shock or peritonitis. Now and then a perforation causes no characteristic symptoms at all (Cruice, Ibid.).

**Tuberculosis of the Mesenteric Glands in Children.**—This condition is very common. Corner ("Lancet," Feb. 17, 1912) makes a statement exactly in accord with my own experience when he says that "tuberculous mesenteric glands will be found in practically every child patient submitted to an abdominal operation." They are often recovered from unrecognized. They may produce the well-known symptoms of *tabes mesenterica*, viz., greatly enlarged abdomen, constipation or constipation alternating with diarrhea, loss of flesh, anorexia, anemia, and perhaps a palpable mass. There are abdominal pains about the umbilicus, coming on after taking food and particularly common at night.

In some cases vomiting occurs and pain is in the right iliac region and the case is mistaken for appendicitis. In such a case the pulse and temperature are normal or only slightly elevated and there is no leukocytosis. In mesenteric tuberculosis the appendix is apt to be dilated, constricted, or kinked. Mesenteric tuberculosis may undergo cure, the glands becoming fibrosed and calcified. It may lead to tuberculosis of the intestine, tuberculous peritonitis, or tuberculosis in some distant part.

I agree with Corner that the abdomen should be opened, the appendix removed, and the patient placed a long time on full antituberculous treatment.

**Tumors of the Intestine.**—**Innocent Tumors.**—Adenoma, lipoma, fibroma, myoma, fibromyoma, papilloma, angioma, and chylangioma may occur. Such tumors are rare. If attached by a pedicle or stem the growth is called a polypus. Adenoma is the commonest tumor. It may or may not be a polyp. It arises from an intestinal gland. There may be one, several, or many. Adenomata may produce no symptoms, may cause hemorrhage, may produce blocking, or may lead to intussusception.

**Malignant Tumors.**—*Sarcoma* is very rare, but does sometimes arise, particularly in young persons, and it enlarges very rapidly. It is most prone to attack the large intestine. Jopson and White[1] reported 1 case and collected 22 others. The mesenteric glands frequently enlarge. *Cancer* is not uncom-

[1] "Am. Jour. Med. Sciences," Dec., 1901.

mon, attacking especially the middle aged. According to Rolleston, the average age in duodenal cancer is fifty-two years; in jejunal and iliac cancer, forty-seven; in cancer of the cecum, nearly forty-eight, and in cancer of the rest of the large intestine, about forty-nine years. It is most common in the neighborhood of the ileocecal valve and in the sigmoid flexure. Ewald collected 1148 cases of cancer of the intestine. In 64 cases the cecum was involved; in 24 cases the ileum was involved. There is pain at the seat of growth. After a time constipation is noted, or constipation alternating with diarrhea. Finally, intestinal obstruction occurs. In some cases the symptoms appear suddenly, acute obstruction taking place or intussusception occurring. It is usually possible to palpate the tumor, which is hard and immovable. The patient wastes rapidly and is apt occasionally to pass blood at stool. The growth does not enlarge very rapidly and glands are not involved early. In some cases the supraclavicular glands enlarge. In more than one-half of the cases which die of intestinal cancer there is no lymphatic infection.[1]

**Treatment.**—Early in the case exploratory laparotomy should be performed, followed, if possible, by excision with end-to-end or side-to-side approximation. This is done for either cancer or sarcoma. It may be possible to remove enlarged glands. In cancer of the cecum extirpate the cecum and implant the end of the ileum into the side of the colon (Wm. J. Mayo). If excision is impossible, the growth should be side-tracked by performing lateral anastomosis. In advanced cancer of the large bowel, if resection is impossible, make an artificial anus above the tumor. (See Cancer of Rectum, page 1187.)

**Appendicitis.**—The vermiform appendix is found in man and anthropomorphous apes. Chimpanzees in captivity are apt to die of appendicitis. Some mammals "closely allied to the anthropomorphous apes possess very large ceca; and in some of these the terminal segment of the cecum resembles the vermiform appendix in that it possesses a very large proportion of the peculiar kind of tissue known as adenoid or lymphatic" ("Evolution and Disease," by Sir J. Bland-Sutton). The appendix is usually regarded as a vestigial structure. Prof. Berry, Dr. Arthur Keith, and some others regard the appendix as a specialized region of the cecum. The fact that the appendix of a newborn child is as long absolutely as the appendix of a full-grown man is regarded by Bland-Sutton as proof "that the part was of great importance to the ancestors of the human species" (Ibid.). Appendicitis, which is an inflammation of the vermiform appendix of the cecum, is almost invariably the primary lesion of all of those various conditions known as typhlitis, perityphlitis, paratyphlitis, etc.—terms which seldom imply pathological entities, and are in most instances well relegated to obscurity. I say in most instances, not in all, because I believe there is such a condition as primary inflammation of the cecum, although it is extremely rare. This rare condition may cause perforation, perityphlitic abscess, or peritonitis when the appendix is sound. It is not to be distinguished clinically from appendicitis (McWilliams, in "Annals of Surgery," June, 1907). Involvement of the cecum as a result of appendicitis is common. Such a condition should be expected because the mucous membrane is continuous. It was recognized by some observers many years ago that such a disease as inflammation of the appendix existed, but the majority of the profession did not grasp the fact. In 1759 Mestevier, of France, reported a case of perforation of the appendix by a pin.[2] In 1812 a perforated appendix was shown to the Medico-Chirurgical Society of London,[3] and in 1835 Southam reported an appendiceal abscess (Manley). In 1848 Hancock reported an appendiceal abscess ("Lancet," 1848, p. 380). It is interesting to

[1] Wm. J. Mayo, "Jour. Am. Med. Assoc.," Oct. 19, 1901.
[2] "Jour. Méd. Chir. and Pharm.," vol. x, 1759.
[3] "Med. and Chir. Trans.," London, 1812, vol. iii.

note that this was a case of appendicitis in pregnancy. Ten days after a premature delivery an abscess was opened. About two weeks after operation two fecal concretions came out of the wound. In 1827 Dr. L. Mélier described appendicitis, and named among its symptoms fixed pain in the right iliac fossa and colic. This brilliant and original young Frenchman was years ahead of his contemporaries. He reported cases of undoubted appendicitis verified by autopsy, described gangrene, perforation, associated peritonitis, and appendiceal concretions. His original article, Manley[1] tells us, is in the "Journal of Medicine, Surgery, and Pharmacy" for 1827, second series, 110. Howard Kelly quotes it from "Jour. gén. de méd.," 1827, vol. c, p. 317. Mélier said: "If it were possible to establish with certainty the diagnosis of this affection, we could see the possibility of curing the patient by operation. We shall perhaps some day arrive at this result."[2] In spite of Mélier's writings, the profession adhered for half a century to the view of Dupuytren, put forth in 1833, that abscesses in the iliac region take origin from the cecum and not from the appendix. Dr. Reginald Fitz, of Boston, in 1886 persuaded the world that the appendix is the real seat of most inflammations in the right iliac fossa, and introduced the term "appendicitis" ("Am. Jour. Med. Sciences," 1886, vol. xcii). This structure is particularly liable to infection because of the large amount of lymphoid tissue in its make-up, because it is in a dependent position, is always full of bacteria, has a poor blood-supply, and is readily blocked by kinking or by swelling of its mucous membrane. Further, as a vestigial structure, it has a low resisting power. A functionless part, like a loafer in a city, is a dangerous element. Each is a menace. The loafer is apt to become a criminal; the appendix is apt to inflame and kill. The appendix is a long and narrow diverticulum (musculomembranous in structure), which comes from the posterior and internal part of the cecum, and which probably has no physiological function. The structure of the appendix is similar to the structure of the colon, except that the muscular structure is ill developed and trivial in amount. Lockwood[3] points out that there is an extensive lymph system in the appendix, and that the submucous and subperitoneal tissues communicate by numerous gaps in the muscles. This structure has a poor blood-supply, and in consequence gangrene occurs from rather trivial causes. It is supplied by a branch from the superior mesenteric artery. In women there is sometimes an additional supply by a vessel running in the appendiculo-ovarian ligament. The nerves are derived from the superior mesenteric plexus. The appendix averages about $4\frac{1}{2}$ inches in length, but varies in size between the limits of $\frac{1}{3}$ inch and a little over 9 inches. In 641 autopsies the longest appendix was $9\frac{1}{3}$ inches and the shortest was $\frac{1}{3}$ inch (Monks and Blake). Its diameter is, as a rule, about equal to that of a No. 9 English bougie; its canal is narrow and is partly closed by the valve of Gerlach (Talamon). The appendix enters the cecum at its posterior internal part, which is usually the seat of the most intense pain in inflammation, and corresponds to a point on the surface 2 inches from the anterior superior spine of the ilium, on a line drawn from the umbilicus to the iliac spine, which is known as *McBurney's point.* The free part of the appendix in one-third of all persons is in relation with the posterior surface of the cecum; in almost one-third of all persons it is fixed in the iliac fossa, so that if perforation occurs the contents will be voided into the retroperitoneal tissue (iliac abscess). In some cases it is external to the cecum; in some it passes downward, and in some inward. It is important to remember that the appendix may be met with in the most unexpected situations. When the ascending colon is displaced, the diverticulum may be upon the left side. It is not

[1] "Med. Record," July 19, 1902.
[2] See R. J. Lee Morrill's article in the "Amer. Med. Surg. Bull.," Dec. 19, 1896.
[3] "Brit. Med. Jour.," Jan. 27, 1900.

unusual to find its tip in the middle line, up toward or adherent to the gall-bladder, or in the pelvis. In about two-thirds of all cases the appendix is completely covered by peritoneum; in one-third of all cases it is in contact, in some part of its length, with cellular tissue (Talamon). Byron Robinson has called attention to the fact that the appendix in men is frequently in contact with the psoas muscle, and may be bruised by this muscle. In 10,000 autopsies the appendix is said to have been absent five times. In most cases in which surgeons have been unable to find the appendix it was not absent, but was covered by peritoneum. Occasionally the appendix is found in a hernial sac.

**Etiology and Pathology.**—Appendicitis is very rare in infants. I operated unsuccessfully on a male two years of age for gangrenous appendicitis. Savage operated unsuccessfully on a baby sixty-one days old, and Weiss operated unsuccessfully on a child twenty months old.[1] J. P. Crozer Griffith[2] has collected 15 cases in children under two years of age. One of these patients was three months old. Nine of the 15 were operated upon, with 7 recoveries. In 4 of the cases the appendix was in the scrotum. In 2 cases a diagnosis of intussusception was made. In children nine or ten years old the disease is by no means infrequent (see page 1013). Appendicitis is common at any period beyond childhood, being more frequent in young and middle-aged people than in the aged. It is about four times as common in males as in females. It is more common in summer than in other seasons, and in warm countries than in cold or temperate climes. Appendicitis is a bacterial disease. It is produced occasionally by pus cocci, but most commonly by the action of the Bacterium coli commune of Escherich. The colon bacilli, which normally inhabit the appendix, are harmless when the appendix is healthy, but become active for harm when the diverticulum is bruised, obstructed, irritated by the presence of uric acid, congested because of chilling of the cutaneous surface of the body, or distended by the ingress of colonic fluid (C. Van Zwulenburg, in "Annals of Surgery," March, 1905). It seems probable that flatulent distention of the colon may be responsible for forcing fecal matter in quantity into the appendix and may lead to plugging of the opening (Rubin, in "Jour. Am. Med. Assoc.," vol. xliii, No. 18). When inflammation occurs, swelling of the mucous membrane may occlude the opening into the colon. If this occurs, the lumen of the appendix is dilated, filled up, and becomes distended by a thick mucopurulent fluid. Ulcers sometimes form which may only involve the mucous membrane, may pass deeply into the coats, or may even perforate. Dieulafoy[3] maintains forcefully that appendicitis is due *always* to the conversion of the appendix into a *closed* cavity, but cases are met with which disprove this assertion. Various conditions may bring about this transformation. Partial obstruction may be caused by calculi, which are composed of stercoral material and hordes of bacteria mixed with salts of lime and magnesia. These calculi are not formed in the colon, but are formed in the appendix. The theory that concretions form in the colon and are forced into the appendix by peristalsis has been very largely abandoned. Dieulafoy speaks of the condition as *appendicular lithiasis*, and says it has a tendency to run in family lines, and has a kinship to gout and rheumatism. Obstruction may be caused by local infection of a catarrhal area, by the formation of a fibrous stricture, or by several causes acting in unison. The presence of a concretion is always dangerous. It is frequently associated with ulceration, either as cause or effect. It is a mass of virulent bacteria. It may lead to perforation or gangrene. Talamon taught that the appendix resents the presence of the concretion, reflex contraction of the muscular coat taking place, which is accompanied by violent pain (*appendicular colic*). The muscular structure is so rudimentary that it does not seem probable

[1] Manley, in "Med. Record," July 9, 1902. [2] "University of Penna. Med. Bull.," Oct., 1902.
[3] "Progrès médicale," No. 11, 1896.

that attempts at contraction, even should they arise, would produce violent pain and distant symptoms. Pozzi[1] believes that appendicular colic may be caused by torsion or bending of the appendix or malposition of the diverticulum, and holds that pain may arise when there is no lesion in the appendix and no inflammation of the peritoneum or pericecal structures. What is called appendicular colic is really inflammation of the appendix without involvement of the peritoneum. The term "appendicular colic" has led to much injudicious conservatism, and, as Lockwood shows, if an appendix is removed from an individual who suffers from attacks of appendicular colic, it will usually be found that the diverticulum is inflamed or the lumen contains a concretion. Foreign bodies, such as pins, fish-bones, nails, buttons, date-stones, cherry-stones, and grape-seeds, may enter the appendix, but they do so far less often than is generally supposed, most alleged grape-seeds from the appendix being fecal concretions. Fitz found concretions in 15 cases out of 300. Ranvier collected the records of 459 postmortems, and found reported 179 fecal concretions and 16 foreign bodies. In Burgess's 500 cases fecal concretions were found in 21 per cent. ("Brit. Med. Jour.," Feb. 24, 1912). Appendicitis due to a foreign body, such as a grape-seed or a pin, is known as *foreign-body appendicitis;* appendicitis in which a concretion is the assumed cause is known as *stercoral.* A foreign body may produce instant perforation. If impaction of a foreign body or concretion occurs, the orifice of the appendix is closed, the circulation is soon cut off, the secretions are retained, the coats become congested, the diverticulum enlarges enormously, microbes multiply with great rapidity, and the wall of the congested appendix inflames and may become gangrenous or ulcerated, and is finally perforated. Interference with the blood-supply of the appendix will predispose to appendicitis. This may be brought about by twists, bruises, adhesions, concretions, pressure, or bands; and the psoas muscle may play a part in the production of these conditions. In women appendicitis is occasionally secondary to tubo-ovarian disease. Appendicitis is rarer in women than in men, probably because in many females the appendix has a better blood-supply than in males, the additional supply coming through the folds of the appendiculo-ovarian ligament. In women disease of the uterus or adnexa frequently precedes or actually causes appendicitis. Catarrhal conditions of the intestine, habitual constipation, and indigestion with flatulence predispose to appendicitis. In fact, in a great many cases there has been a more or less prolonged history of diarrhea or constipation and flatulent indigestion before the development of acute appendicitis. An acute attack of appendicitis may arise after the eating of a large and indigestible meal, especially if such a meal was taken late at night. Bolting the food and eating large meals at irregular hours predispose to an attack. It seems probable that catarrhal appendicitis may result from extension of catarrh of the colon, and possible that, in rare cases, appendicitis may arise from external traumatism (*traumatic appendicitis*). In most cases, however, in which appendicitis seems to be produced by a blow, the injury at most simply "awakened a sleeping dog" and stirred into acute inflammation an appendix already diseased. It is well to be skeptical as to external force causing appendicitis. Sprengel ("Deut. med. Woch.," Dec. 14, 1911) says there is no case in literature, in which abdominal trauma is alleged as the cause, confirmed by scientific evidence. If before perforation the appendix adheres to the cellular tissue behind the cecum, cellulitis or abscess without peritonitis may result. When appendicitis goes on to perforation, there is always some peritonitis; but if the steps to perforation are gradual, and if the causative organism is the colon bacillus, the peritonitis may be local. Sometimes, by formation of adhesions, a barrier is made between the appendix and the peritoneal cavity before perforation occurs. When perforation takes place sud-

[1] "Progrès médicale," No. 19, 1896.

denly peritonitis is inevitable. When the causative organism is the streptococcus, general peritonitis is very apt to arise. Peritonitis may arise without perforation by contiguity of structure or by migration of bacteria through the congested walls of an obstructed appendix. In some cases perforation takes place into the peritoneal cavity, but pus is circumscribed by matting together of the intestines with plastic exudate. The appendix may become gangrenous very rapidly or after some time. A case of appendicitis in which gangrene and perforation come on very quickly is spoken of as *fulminating appendicitis*. In some cases, if the perforation is very small and the appendix is swathed in lymph, or if perforation does not occur, the inflammation may subside. Perforation rarely occurs from liquid pressure or from the pressure of a concretion; it is generally due to ulceration produced by the action of micro-organisms. Appendicitis which subsides may at any time recur, and the life of such a patient is under constant menace. An enormous number of people have had appendicitis. Toft recorded 500 autopsies, and in 36 per cent. of them there were positive signs of past attacks. The disease is occasionally unsuspected during life. These facts prove that the disease may subside without the aid of surgery.

**Forms of Appendicitis.**—In what is known as *appendicular colic* the appendix is temporarily obstructed because of transitory inflammatory swelling of the mucous membrane of the outlet, and the stercoral contents are retained in the diverticulum. The peritoneal covering is not involved in the inflammation. This condition is called by Fergusson *constipation of the appendix*. If not relieved, it will eventuate in appendicitis with involvement of the peritoneal coat. It is an unfortunate term, sometimes used as an excuse for avoiding operation. In such cases a concretion is frequently or usually present.

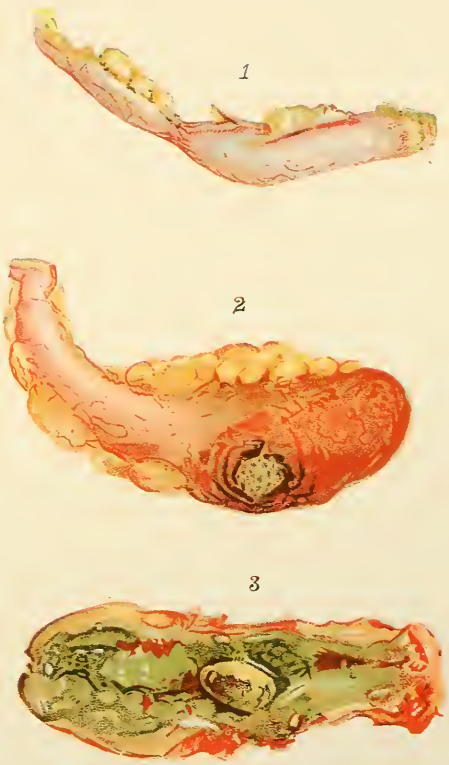
*Simple parietal* or *catarrhal appendicitis* does not remain limited to the mucous membrane; hence the term *catarrhal* is not strictly correct. The vessels of the appendix are distended with blood, the lumen at the intestinal end becomes partially or completely obstructed, the epithelium desquamates from numerous glands, the mucosa ulcerates, and the lumen of the appendix becomes filled with a mixture of mucus, bacteria, and portions of organic matter. Bacteria enter the lymph-spaces of the wall of the appendix, and pass rapidly from the submucous to the subperitoneal tissues. In from twelve to thirty-six hours after the mucous coat begins to inflame the peritoneal coat will probably be involved. The inflammation may undergo resolution and the patient get well, or a wait for cure may result disastrously. The appendix may thicken, ulceration may take place, and peritonitis may arise. Suppuration or gangrene may occur, perforation may take place, or pyemia, with abscess of the liver, may arise. The acute condition may pass into chronic appendicitis or ulcerations of the mucosa may remain; the mucous crypts may be filled with bacteria; a concretion may exist; cicatricial contractions may occur. In any of these conditions the patient is in danger of a fresh attack at any time. In a catarrhal inflammation secondary to catarrh of the colon the case may be chronic from the beginning. If the lumen of the appendix is gradually and completely obliterated the condition is denominated *obliterative appendicitis* (Senn). This progressive obliteration may result from repeated attacks of inflammation or may simply be a degenerative change. *Recurrent appendicitis*, it was once said, may be due to inordinate size of the mouth of the appendix, making of this diverticulum a drag-net for foreign bodies; but we now know that it is more probably due to smallness of the opening, so that it quickly closes from slight swelling and converts the appendix into a closed vase filled with septic material. *Suppurative appendicitis* is due to purulent infiltration of the walls. Pus in the lumen is not purulent appendicitis. Pus may form about the appendix, a condition known as *appendiceal* or *appendicular abscess*. *Gangrenous appendicitis* is a moist or septic

gangrene, due to interference with the circulation and to tissue destruction by the action of micro-organisms. Perforation occurs and multiple perforations are common. The entire appendix may slough off. Interference with circulation may be caused by an obstruction, by a bend or twist or bruise of the appendix, or by the action of virulent organisms on an appendix whose tissue resistance is lowered by injury or disease. In gangrenous cases the vessels of the meso-appendix are usually obstructed by thrombi of the veins or arteries. In rare instances appendicitis is due to tuberculous ulceration, in other cases to typhoid ulceration, and genuine appendicitis may arise during typhoid fever.

Fowler suggests the following classification of cases of appendicitis: Endo-appendicitis; parietal appendicitis; peri-appendicitis; para-appendicitis.

As a matter of fact, appendicitis is always one disease which varies in intensity and complication. It is useless to divide it into a great number of symptomatic groups.

**Symptoms and Signs.**—In what is known as *appendicular colic* the patient suffers from disorder of digestion and occasionally has a brief attack of abdominal pain associated with trivial and temporary tenderness in the right iliac fossa. The colicky pain is about the umbilicus and right iliac fossa; there is often nausea and usually constipation. This condition, if not soon relieved, is followed by the evidences of peritoneal inflammation. The symptoms of genuine acute appendicitis are as follows: In some cases the disease seems to begin suddenly, but in most of the cases there are noted for a few hours or even for a day or two distinct *premonitory symptoms*, among which are constipation or diarrhea, flatulence, nausea, anorexia, dyspepsia, coated tongue, weakness, general gastro-intestinal uneasiness, colicky pain about the umbilicus, and, perhaps soon, tenderness, a sense of weight, soreness, or uneasiness in the right iliac fossa. The acute symptoms suddenly appear after the premonitory symptoms have lasted a variable time, and the acute symptoms very frequently appear in the early hours of the morning. The first definite symptom is severe colicky pain. The tongue is coated and usually dry. Great thirst is often complained of. The face is expressive of pain or, later, in a severe case, becomes Hippocratic. The posture assumed for greater ease is one of recumbency, with the right thigh and knee or both thighs and knees partly flexed. Respirations in acute appendicitis are shallow and thoracic. The development of acute pain is usually the most prominent symptom. The pain is at first colicky and located about the umbilicus or through the abdomen in general, this distant, primary, or generalized pain, according to Treves, corresponding to the distribution of the superior mesenteric plexus. Mr. Burgess ("Brit. Med. Jour.," Feb. 24, 1912) states the present view when he says that the primary pain is referred to "the peripheral distribution of the spinal nerves arising from those segments of the spinal cord with which the appendix and small intestine are connected through their sympathetic nerve supply—eighth to eleventh dorsal." This primary pain may subside if the appendix succeeds in emptying its contents into the colon, but it may also subside if the appendix becomes gangrenous or ruptures (Murphy). Usually in from twelve to thirty-six hours the pain becomes localized in the right iliac fossa, and associated with tenderness and hyperesthesia of the skin—in other words, true inflammatory pain develops. It is due to peritoneal inflammation. "Thus, the closer the situation of the appendix to the parietal peritoneum, the earlier will the latter be irritated and the sooner will the pain be localized" (Burgess, Ibid.). The usual location of the pain in the right iliac fossa depends on the fact that the appendix is usually placed in that region. Occasionally, when the appendix crosses the belly, the pain is located on the left side, and occasionally, for like reasons, in the gall-bladder region, the right loin, or the pelvis. "If the appendix lies among coils of small intestine or in the pelvis there




Various forms of appendicitis (from drawings by Dr. M. H. Richardson): 1. Obstruction from stenosis of appendix. 2. Dilatation of distal end of appendix; perforation by a fecal concretion 3. Gangrene of nearly the whole of the appendix; fecal concretion in lumen.

may at no time be local pain, the initial umbilical pain becoming steadily generalized" (Burgess, "Brit. Med. Jour.," Feb. 24, 1912). If the pain of appendicitis is violent the patient presents some evidences of shock. Nausea is the rule in appendicitis; vomiting usually occurs early—about three or four hours after the beginning of pain. In children vomiting is often early, violent, and persistent, but in adults, after the early hours of the attack, vomiting occurs, as a rule, occasionally or not at all, although nausea is complained of. Early vomiting is a reflex symptom due to distention of the appendix (Murphy). If vomiting persists, it points to peritonitis, to pus formation, or to intestinal obstruction unless it results from the administration of morphin. There is usually constipation in acute appendicitis, although diarrhea occasionally occurs. In appendicitis there is always some elevation of temperature, although it may be very slight and of brief duration. The fever is not ushered in by a chill, but the temperature mounts in the course of a few hours to 102° or 103° F. or even higher. The fever does not begin until several hours or a number of hours after the onset of pain. In a very mild case the temperature remains elevated for a day or two and then falls to normal. In severe cases it is apt to remain elevated for a longer period, but it is always to be borne in mind that in very grave appendicitis the surgeon may find very little elevation of temperature, no elevation, or actually a subnormal temperature. In gangrenous cases, and in cases in which a large perforation suddenly forms, and when general peritonitis develops, there is usually, for a time at least, a subnormal temperature. A *sudden* drop of temperature indicates, as a rule, a calamity, particularly gangrene of the mucosa of the appendix, which prevents absorption (Murphy), or perforation of the appendix. Leukocytosis is usually present (see Diagnosis). The pulse in appendicitis is in most cases rapid. A very rapid pulse (well over 100) is significant usually of a severe case, and the auguries are especially ominous if the pulse is rapid but the temperature is normal or subnormal. Occasionally, however, a slow pulse exists, even in the worst cases.

Examination of the abdomen may discover, early in the case, general abdominal rigidity; but usually in the course of twenty-four hours or more the general rigidity passes away, the abdomen distends more or less, and rigidity of the lower half of the right rectus muscle becomes evident and persists. If general peritonitis begins early, general abdominal rigidity does not abate or pass away. If general peritonitis begins later, general abdominal rigidity, which was present at first but which passed away, returns. Rigidity may not exist in the very beginning of appendicitis, in a case in which the appendix is retrocecal or pelvic, in some abscess cases, or in a case with relaxed belly walls.

A symptom almost invariably present in appendicitis is tenderness. In some cases the tenderness is diffuse; in most it is localized, or at least most acute, in the right iliac fossa. The point where tenderness is usually most acute is a spot about 2 inches internal to the anterior superior spine of the ilium, on a line drawn from that bony point to the umbilicus (*omphalospinous line*). This is known as *McBurney's point*, and overlies the usual point of origin of the appendix. In some cases, however, the greatest point of tenderness is nearer the gall-bladder; in others, in the loin; in others, toward the umbilicus, in the midline, or on the opposite side; in others, in the rectum. The seat of greatest tenderness depends on the situation of the appendix, and it is usually at McBurney's point, because this usually overlies the origin of the appendix. The lesson is that in appendicitis there is a point of tenderness or of greatest tenderness in a region which the appendix could occupy. If tenderness exists on the right side and then develops in the left side, severe spreading peritonitis usually exists (W. Meyer). When the appendix becomes gangrenous, local tenderness may for a time disappear, because the peritoneum of the involved region has become anesthetic; later, however, it returns, spreads,

and may become general. In view of the fact that tenderness in the right iliac fossa is often demonstrable in tubal and ovarian disease, the sign in males "is of greater significance than in females" (A. H. Tubby, on "Appendicitis," in "Medical Monograph Series"). Pressure upon the left side will, in some cases, cause pain in the right iliac region. When rigidity abates or disappears the case may go on to cure, but sometimes a mass becomes evident in the right iliac fossa. The mass, of variable shape, is at first hard, and if of any considerable size, is dull on percussion. In some cases when no mass is palpable through the abdominal wall, rectal examination detects one. This mass may be agglutinated bowel and omentum or a collection of coagulated inflammatory exudate. It may gradually disappear or an abscess may form. The evidences of general peritonitis are: great distention because of intestinal paresis, general abdominal tenderness, rectal tenderness, very rapid pulse, hiccup, persistent vomiting which may become regurgitation, and, as Meyer points out, percussion dulness over the right iliac region or entire lower abdomen.

In some cases the symptoms, at first trivial, become grave. In some all the symptoms are violent from the beginning, the attack tends to linger, and is followed by persistent soreness of the appendix and harassing digestive disturbances. Any case of appendicitis may become all of a sudden desperately grave because of perforation or gangrene, and in any case general peritonitis may develop. After sudden perforation or rapid gangrene the temperature falls, hiccup begins, abdominal distention, pain, and tenderness become marked and general, and the pulse becomes very rapid. In some cases these grave symptoms are present almost from the start (fulminating cases). A sudden perforation produces collapse and, if reaction takes place, general peritonitis arises. Peritonitis, be it remembered, may arise without either perforation or gangrene. If pus forms, it may be unlimited by adhesions. In such cases there is the rapid onset of fatal peritonitis and septicemia. Pus may be limited by adhesions and be practically extraperitoneal. In such a case a lump is felt in the right iliac region, but dusky discoloration and edema of skin very seldom exist. The surgeon does not wait for fluctuation before he makes a diagnosis. In an abscess case there are usually irregular fever and sweating, but rigors do not occur. Hawkins says we should always suspect pus if the symptoms continue after the sixth day, and particularly when the symptoms abate and suddenly increase between the seventh and tenth days. A limited collection of pus may be liberated into the peritoneal cavity by rupture of the abscess wall. Such a rupture may be caused by pressure or muscular effort; rupture is followed at once by shock and later by diffused peritonitis. An abscess may rupture externally or into the vagina, intestinal tract, or bladder. It is desirable, if possible, to locate the situation of the appendix, and this is usually determined by locating the seat of swelling and of greatest tenderness. The surgeon should not lose sight of the fact that the appendix may be found in the most unexpected situations. In every case a rectal or vaginal examination should be made in order to detect swelling and tenderness, and thus determine if the inflammation took origin in or has come to involve the pelvic region. Pain at the end of micturition points to involvement of the vesical peritoneum.[1] In cases in which there is no *localized* swelling and no local tenderness—for instance, in gangrenous or perforative appendicitis with general peritonitis—"diagnostic localization" is impossible (Van Hook).

**Terminations and Prognosis.**—Acute appendicitis may terminate in death, in complete recovery, or in a condition of lowered vitality during the existence of which acute attacks are almost certain to occur. Sometimes after and sometimes without an antecedent acute attack the patient develops

[1] Van Hook, in "Jour. Am. Med. Assoc.," Feb. 20, 1897.

persistent soreness and tenderness in the right iliac region. Between the attacks of recurrent appendicitis there may be soreness, tenderness, and gastro-intestinal disturbance, or there may be no evident trouble whatever; yet, even in the latter case, there may be an ulcer or ulcers of the mucous lining. If a patient has once had appendicitis he will always be liable to suffer from another attack if the appendix has not been removed. The liability becomes almost a certainty if the intestinal end of the appendix is narrowed or if the lumen is obstructed at any point, if a concretion exists, or if there is an area of ulceration or of desquamating epithelium. After an attack the appendix may remain enlarged and tender; exercise or indiscretion in diet may cause it to become tender, or the patient may have occasional attacks of colicky pain. If any of the above conditions exist, another attack may be confidently anticipated if operation is not performed. In such cases the appendix can usually be palpated. The method of palpation proposed by Robert T. Morris[1] is very useful. It is applied as follows:

The surgeon stands to the right of the patient and uses three fingers of the right hand to feel with and three fingers of the left hand to press with. Morris insists that no muscular effort should be used by the hand which feels. The feeling fingers are pressed by the other fingers beneath the margin of the right rectus muscle on a level with the umbilicus, and are drawn toward the patient's right side, and the colon will be felt to roll under the fingers. The process is repeated several times until the end of the cecum is reached. The appendix is sought for by rolling the cecum from side to side with the finger-tips, and working toward the proximal end of the appendix.

Adhesions may form as a result of appendicitis, general peritonitis may arise, the appendix may slough or become perforated, or abscess may ensue upon local peritonitis. Lymphangitis of the appendix may accompany, and septic lymphangitis or phlebitis and secondary hepatic and lymphatic infections may follow, appendicitis. They are thought to be most common after mild attacks of appendicitis. The secondary *lymphatic* and *hepatic infections* are of the greatest importance. There may be abscess of the liver, subphrenic abscess, or retroperitoneal lymphangitis.

A subphrenic abscess may result from infection carried from the appendix by the lymphatics, from pus ascending along the posterior cellular spaces, or by direct invasion from the peritoneal cavity (John C. Munro, in "Annals of Surgery," Nov., 1905); such an abscess is usually on the right side, but may be upon the left.

Lymphangitis is the rule in appendicitis, and when we open the abdomen there is usually evidence of it in the lymph-glands of the mesentery, and in children particularly these glands are apt to be enlarged. One lymph path from the appendix is through the ileocecal glands, another is posterior to the cecum and retroperitoneal, and the latter reaches the liver and diaphragm (Munro). In lymphatic infection an abscess may form anywhere in the course of the lymphatics. Abscess of the liver usually results from portal invasion, but may result from lymphatic infection.

Among other possible consequences of appendicitis may be mentioned pyemia, empyema, inflammation of the parotid gland, and thrombosis of the right iliac vein. A positive prognosis of any case of appendicitis is an absolute impossibility. The future of every case is clouded with uncertainty, and the most that can be attained in the field of prediction is a scientific guess of more or less probability. All surgeons have seen apparently hopeless cases recover, and have observed cases with the most trivial symptoms grow progressively worse or suddenly develop a fatal complication. Further, after one attack other attacks are very apt to arise. The medical man who

[1] "Medical Record," Sept. 17, 1898.

estimates that 80 or 90 per cent. of cases get well without operation has probably dealt with many catarrhal cases, and he certainly is optimistic as to freedom from future attacks, because, as stated before, recovery from an attack does not of necessity mean freedom from the disease. In appendicitis there may be delusive evidences of improvement; for instance, the abatement of pain and the lessening of fever, being regarded by the patient himself as indubitable signs of improvement, may, in reality, be indicative of gangrene. In spite of the previously mentioned difficulties and obscurities we can in the majority of cases decide with a reasonable probability of accuracy whether or not the patient is becoming worse. In a delusive improvement some signs and symptoms improve, but all do not; and in endeavoring to form a prognosis, *all* the signs and symptoms must be noted and weighed: pain, tenderness, rigidity, distention, nausea and vomiting, delirium, intestinal obstruction, shock, the temperature, the rapidity of the pulse, the blood examination, etc. If *all* these elements, not only some of them, point to improvement, we may be reasonably confident that improvement is really taking place. If only some of them point to improvement we will in many cases be altogether uncertain as to the significance of the change.

The **diagnosis** is not invariably so easy, as many light-hearted operators seem to believe. It is frequently far from easy and is sometimes altogether impossible without exploratory operation. Sonnenburg maintains that we can diagnosticate the pathological condition of the inflamed appendix. Personally, I am unable to do this with any certainty, although I always try, and am often right and just as often wrong.

In attempting to make a diagnosis, besides the ordinary examination of the abdomen a rectal or vaginal examination should be made, associated in many cases with bimanual palpation. If an appendix is enlarged and an individual has a thin abdomen which is not rigid, it may be possible to palpate the appendix. Sometimes it can be felt after the administration of ether when it could not be detected before. In an acute case forcible or prolonged palpation is always unjustifiable, as it may force an ulcer to perforate, or may rupture an abscess, and the information gained is not of sufficient importance to justify the risk. In a chronic case information of great value may be obtained and there is no real risk in the maneuver. I am persuaded John B. Murphy is correct in attaching the greatest possible importance to the order in which symptoms appear in acute appendicitis. Pain *precedes* nausea and vomiting, elevated temperature, and abdominal tenderness. If fever precedes pain the condition is not appendicitis. If vomiting precedes pain the condition is probably not appendicitis.

The disease may be confused with a number of different conditions. It sometimes is confused with typhoid fever; in fact, early typhoid fever associated with marked abdominal pain gives a picture very similar to that furnished by appendicitis.

In typhoid fever the temperature is usually distinctly higher than that commonly encountered in appendicitis. Maurice H. Richardson[1] tells us that in every case in which typhoid is suspected, operation is not justifiable on the hypothesis of existing appendicitis, unless there are local pain and localized tenderness in the appendix region, associated with definite muscular resistance or distinct rigidity; and that operation should be postponed in a case in which the constitutional signs are severe and the local signs are difficult to detect; but when there are pain, tenderness, and rigidity with or without distention, operation must be performed, even when one recognizes the possibility of the existence of typhoid fever. Richardson lays down the following rule: Soft abdomen plus high temperature suggests ty-

[1] "Boston Med. and Surg. Jour.," Jan. 9, 1902.

phoid, even if there are pain and tenderness. In appendicitis there is usually leukocytosis; in typhoid, leukocytosis is absent, except when perforation is imminent or has occurred, or when some other complication exists. I have seen the operation performed twice for supposed appendicitis when the condition in each case was found to be early typhoid fever.

Acute intestinal obstruction is sometimes confused with acute appendicitis, and the mistake is particularly likely to occur if the obstruction is due to intussusception. In acute obstruction, as in appendicitis, the pain is first appreciated about the umbilicus; but in acute obstruction it remains in that region, does not pass to and localize itself in the right iliac fossa, and is not associated with tenderness of the right iliac fossa. In obstruction the vomiting is persistent; in appendicitis, except in the beginning, it is usually trivial and often absent, although in children it may be violent and persistent. In acute obstruction shock is much more pronounced than in appendicitis, and early and great distention of the abdomen is noted. The temperature in obstruction is seldom elevated and is usually subnormal; while in appendicitis, at least in the majority of cases, the temperature is distinctly elevated. Further, in acute intestinal obstruction the constipation is absolute, not even gas passing. In children, intussusception is capable of particularly confusing the diagnosis, because, after the first day, it is by no means unusual to have distinct fever in this condition, and occasionally a tumor-like mass is found in the right iliac fossa; but in intussusception the tumor does not remain fixed, but alters its position; it is movable; and the patient usually suffers from tenesmus and the passage of bloody mucus. One should bear in mind that in acute appendicitis associated with septic peritonitis acute obstruction may exist; and that the diagnosis of obstruction may be made without recognizing the appendicitis.

In those rare cases of typhlitis occasionally encountered the symptoms are much milder than in appendicitis: the temperature is not much elevated, the pulse-rate is only slightly accelerated, the leukocytosis is not marked, there is seldom rigidity, there may be tenderness, but is seldom pain. Pain when present is colicky rather than continuous. There may be a doughy mass or a mass feeling "like an air-cushion" in the right iliac fossa (Raymond Russ, in "Surg., Gynec., and Obstet.," Oct., 1912). Chronic typhlitis causes muscular atony and intestinal stasis (Russ, Ibid.).

Lesions of the kidney are sometimes mistaken for appendicitis, but in renal colic the pain runs into the groin and testicle of that side, and occasionally passes down the front of the thigh or into the rectum; and if any tenderness exists, it is found in the loin or in the groin, rather than in the right iliac fossa. Besides, there are other symptoms of kidney trouble. The urine may contain blood or pus, and there may be a history of difficult or of frequent urination, though one should bear in mind that in appendicitis with inflammation of the vesical peritoneum there may also be a record of urinary difficulties. An *x*-ray picture may exhibit a calculus in the ureter or kidney, and a movable kidney is distinctly palpable. In ordinary renal colic there is vomiting in the beginning, just as in the beginning of appendicitis. In movable kidney and renal colic the vomiting is often more violent and prolonged than is common in appendicitis. Movable kidney and appendicitis may exist coincidentally. Very confusing cases are those in which *hematuria accompanies appendicitis*. I have seen it twice and in neither case was there any apparent connection between the appendix and the kidney, ureter, or bladder. The hematuria must have been due to acute nephritis which is known to occur in some cases of appendicitis, the nephritis resulting from the toxins of a bacterial disease. This form of nephritis Dieulafoy calls "nephrite toxique appendiculaire." As pointed out by M. G. Seelig ("Annals of Sur-

gery," Sept., 1908), hematuria may also be due to direct involvement of the kidney, ureter, or bladder.

Gall-bladder difficulties, too, may be confounded with appendicitis. I have operated upon 2 cases of cholecystitis under the supposition that they were cases of appendicitis; and upon several cases of appendicitis in the belief that the condition in each case was cholecystitis. In an inflammation of the gall-bladder, with a distended gall-bladder hanging low down, and with muscular rigidity, the distinction between appendicitis and cholecystitis is always difficult and sometimes impossible. So it is when the cecum has not descended and the appendix is in the gall-bladder region. So it is when the tip of the appendix is adherent to the gall-bladder. In ordinary gall-stone colic the condition is generally sudden in onset; it is characterized by pain in the epigastric region, passing toward the shoulder-blade and the shoulder, the pain being most acute and becoming more or less localized in the region of the gall-bladder; and there is always tenderness over the gall-bladder region. In gall-bladder colic the vomiting is usually violent and often almost continuous.

The perforation of a gastric or of a duodenal ulcer may be diagnosticated as appendicitis. In perforation of a gastric ulcer there is usually a history of previous difficulty with the stomach, though this is not always the case. The onset of acute perforation is sudden, with greater shock than is characteristic of the onset of appendicitis. The pain is violent, the rigidity intense, and the pain, rigidity, and tenderness are in the epigastric region.

Among other conditions that may be confused with appendicitis may be mentioned malignant disease of the cecum, tuberculosis of the cecum, acute tuberculous peritonitis, twisting of the pedicle of an ovarian tumor, tubal disease, extra-uterine pregnancy, membranous colitis, perinephric abscess, tuberculous abscess of the loin or of the groin, and abscess from hip-joint disease.

Pneumonia of the right base and pleurisy may cause abdominal pain and be mistaken for appendicitis. The pain may be due to inflammation of the diaphragmatic pleura or may be reflected along the lower six intercostal nerves which supply the lower part of the pleura and the abdominal wall. Irritation of the eleventh thoracic nerve causes pain in the iliac region. There may even be superficial tenderness in the abdomen, but deep pressure is well tolerated (Donald W. Hood, "Brit. Med. Jour.," Dec. 30, 1905). There may be abdominal rigidity. The abdominal pain seldom persists more than a few hours. It is intensified by deep respiration and is accompanied by high fever. As Hood says, whenever a patient suffers from vomiting, abdominal pain, and high fever, examine the chest. Sir Thomas Oliver has described what he calls "the abdominal type of pneumonia." It is characterized by the sudden onset of severe abdominal pain. Vomiting often occurs. There is then a chill, usually a rise of temperature, and in some cases collapse. The pain is accompanied by tenderness, and both these phenomena may be in the right iliac region. Early there are no physical signs of pneumonia. In a few hours the collapse passes away, the abdominal pain and tenderness subside, the temperature rises, and signs of pneumonia become evident. In young children pneumonia is particularly apt to cause abdominal pain and rigidity. Beyond a doubt, more than one abdomen has been opened for supposed appendicitis when the real condition was pneumonia.

In reaching a diagnosis in doubtful cases of appendicitis I believe that the blood-count is often of service. It is, of course, not to be maintained that the diagnosis of appendicitis may be made by counting the blood; but the blood-count may furnish evidence that, when added to the other signs and symptoms, may be of great importance. In nearly every case of acute appendicitis the hemoglobin is diminished by at least 30 per cent. In a catarrhal appendicitis or in an interstitial appendicitis the leukocytosis is trivial; but

in cases of abscess or of gangrene of the appendix the leukocytes, as a rule, rise from 15,000 to 20,000. It is to be remembered, however, that when the patient is profoundly septic the systemic condition is so depressed that leukocytosis is impossible; hence leukocytosis may be absent in trivial catarrhal cases or in grave cases with overwhelming general sepsis. This latter condition, however, is extremely rare. The blood-count will not help one in making the differentiation between appendicitis and an inflammatory disorder of the pelvis or abdomen, but will aid one in making a diagnosis from typhoid fever, intra-abdominal or pelvic neuralgia, and movable kidney (See J. C. DaCosta, Jr., study of 118 cases, "Am. Jour. Med. Sciences," Nov., 1901.)

**Appendiceal Dyspepsia.**—Indigestion may, for a longer or shorter time, precede an attack of acute appendicitis. A like condition may follow an attack.

In chronic recurrent appendicitis dyspepsia may so dominate the clinical picture as to lead the physician to regard the case as one of gastric disease. Such a patient has prolonged attacks of epigastric pain. There may or may not be tenderness in the appendix region. The condition may be due to hypersecretion of the gastric juice resulting reflexly from appendix inflammation. Fenwick regards chronic hypersecretion as a direct cause of this form of dyspepsia. The epigastric pain may be due to pyloric spasm or gastritis (Eusterman, "Jour. Missouri State Med. Assoc.," May, 1913). At times, however, during some of the attacks appendiceal tenderness is demonstrable and perhaps there is pain in the right iliac fossa. The epigastric or abdominal uneasiness may be constant. Food may immediately cause pain. Nausea and flatulence are common. "As a rule there is not the regularity of onset of pain after food—the periodicity of attack—which characterizes gastric ulcer" (Eusterman, Ibid.). Food does not relieve pain except in the less common cases in which there is hyperacidity.

**Appendicitis in Children.**—The disease is much more common than was once thought (see page 1003). Russel S. Fowler ("Am. Jour. Diseases of Children," August, 1912) collected 183 cases occurring in children under twelve years of age and brought to the German Hospital of Brooklyn. During the same period (1900–1912) the total number of cases was 1115, the proportion of children being 16.41 per cent. The youngest patient in this series was two years and nine months old. There is usually a history of antecedent attacks of gastro-intestinal disorder. The onset is apt to be sudden, but may be insidious, the symptoms as a general thing are violent, and the progress of the disease is rapid. Vomiting is usually more violent and prolonged than in adults. There is a great likelihood of pus formation, and general peritonitis is more common than in adults. Marked leukocytosis usually exists. Occasionally in young children pneumonia begins with so much pain and rigidity in the lower abdomen that the signs seem to point to appendicitis, and an attack of appendicitis may begin coincidently with or soon after a pulmonary inflammation. I have seen 4 cases in children in which pneumonia was ushered in by abdominal pain and rigidity. The surgeon should be awake as to the possibility of typhoid fever, indigestion, fecal impaction, intussusception, and tuberculous peritonitis. In children the appendix occupies a lower position than in adults, the point of abdominal tenderness is usually lower than in adults, the inflammation usually reaches the right side of the pelvis, a painful point can generally be discovered by a finger in the rectum, hence a digital rectal examination must always be made. This usual involvement of the pelvis is responsible for the frequent and painful micturition which is very common (Karewski). Sometimes when the bladder symptoms are very prominent they dominate the clinical picture and the bladder is thought to be the real seat of disease. An attack of peritonitis in a child is more apt to result in general peritonitis than is the same disease in an adult (Selter). I agree with Springer ("Prag. med. Woch.," 1909,

xxxiv, Nos. 7 and 8) that operation in children should invariably be prompt and that purgatives should not be given. In Fowler's series of cases ("Am. Jour. Dis. of Children," Aug., 1912) the operative mortality was 3 per cent.

**Appendicitis in Pregnant Women.**—Appendicitis is a very dangerous, but, fortunately, a very rare complication of pregnancy. In 731 women operated on for appendicitis in the Mount Sinai Hospital, of New York, from 1898 to 1907, only 7 were pregnant (Cooke, in "New York Med. Jour.," May 1, 1909). Lobenstine states that in 30,000 cases under the care of the New York Lying-in Hospital there were but 5 cases of acute appendicitis (Cooke, Ibid.). Most of the patients who develop appendicitis during pregnancy have had previous attacks.

The condition may arise at any stage of pregnancy. It is usually violent, rapid in progress, and accompanied by vomiting. Early in pregnancy the pain and tenderness are significant and are located regionally, as when pregnancy is absent. Cooke points out that later in pregnancy the pains may be so spasmodic as to cause them to be attributed to beginning labor, and they are often located in the region of the liver or even on the left side of the belly (Ibid.). Two hundred and fifty cases have been reported and over 100 have been operated on (Renvall).

Fig. 614.—Tuberculous appendix with perforation and abscess.

Appendicitis in the pregnant is far more dangerous than in the non-pregnant. In about 40 per cent. of cases abortion occurs, and usually the child dies from infection. In some cases of successful operation pregnancy continues to term. The diagnosis is often very difficult because of the enlarged uterus.

**Appendicitis Following Childbirth.**—Hilton collected reports of 29 cases and added 1 of his own ("Surg., Gynec., and Obstet.," Oct., 1907). Hilton demonstrates that childbirth and the puerperium may be causal of appendicitis. The signs and symptoms are apt to be masked or are thought to be due to the puerperal state. The prognosis is grave. In cases developing within ten days of labor 45.5 per cent. died (Hilton, Ibid.).

**Tuberculous Appendicitis** (Fig. 614).—Acute symptoms may develop resembling acute appendicitis. There is usually a history pointing to intestinal stenosis, the stenosis existing at the ileocecal valve.[1] There is always great thickening, and an abscess of large size is apt to form. The cecum usually, but not always, is involved in the tuberculous process. Chronic cases, with palpable enlargement, are sometimes mistaken for cancer of the cecum.

**Malignant Disease of the Appendix** (Fig. 615).—This is a very rare condition (less than $\frac{1}{2}$ of 1 per cent. of appendices removed for supposed inflammation). It is impossible of recognition clinically, but is sometimes discovered postmortem or during operation for supposed acute or chronic appendicitis or pelvic disease. Inflammation does not cause the malignant disease, but the malignant

[1] Andrews, "Annals of Surgery," Dec., 1901.

disease is apt to block the appendix and so cause inflammation. The condition may be carcinoma, sarcoma, or endothelioma, and usually there are distinct inflammatory changes. It is more common in women than in men. Rolleston and Jones collected 42 cases. McWilliams reported 3 cases and collected 45 not in Rolleston's table. This makes 90 reported cases ("Am. Jour. Med. Sciences," June, 1908). Since writing his article McWilliams has found 15 more cases reported. No case is counted in which the colon is diseased. The combined statistics show the average age of the patients to be only twenty-nine years. Two patients were only eight. In most cases the appendix alone is diseased; in some the colon or glands of the mesentery are involved. In three-fourths of the cases the growth is distal to the middle of the appendix. Glands are involved late. Out of 90 cases in McWilliams's table only 8 had enlarged glands, and in 4 of these it was proved that the glands were not cancerous. In about 5 per cent. of cases concretions were found. The chance for permanent cure after removal of an appendix the seat of malignant disease is very good if the disease is limited to the appendix, and is particularly good if the growth is spheroidal-celled carcinoma (Rolleston and Jones). Metastasis is rarely noted. The growth is seldom larger than an almond.

**Treatment.**—If the diagnosis were always certain from the beginning, and if the case were seen at the very start by a surgeon, immediate operation in every case would be eminently proper. If this plan could be followed, the mortality from appendicitis would be extremely small. At this early stage the peritoneum is free from infection, and the appendix can be rapidly and easily removed without risk of infecting the peritoneum. Whenever I see a case early, that is, during the first thirty-six hours of the attack, I practically always advise operation. Unfortunately, this plan cannot be habitually followed. As a rule, when the physician first sees the case the appendicular peritoneum is inflamed, and the surgeon usually sees the case at even a later period than the physician. At this time the barriers of leukocytes are being heaped up to limit the spread of infection, and delicate encompassing adhesions are usually being formed. Even in these later cases I often, in fact, usually, advise operation. Operation at this stage may be imperatively necessary because of the rapid spread and dangerous nature of the process; but when operation is not done, in some cases at least, a temporary limitation will be secured and the case will go on to an interval. Operation in the acute period is always dangerous; operation in an interval is safe. In some instances, when the case is not seen early, it is wiser to avoid operating at the time, and it is proper to wait for an interval. The period in which the surgeon usually sees the case for the first time was said by Maurice Richardson to be "too late for an early operation and too early for a late operation." Those

Fig. 615.—Carcinoma of bowel and belly wall after appendix operation. Appendix removed by English Army Surgeon in Burmah. Portion of bowel removed at Heidelberg.

who say "operate as soon as the diagnosis is made," operate, as a rule, in this dangerous period, and in this period I do not believe that every case should be promptly cut. Many cases, it is true, must be operated on as soon as seen, irrespective of the duration of the disease. We must operate promptly if the pulse is small, tense, and well above 100; if there is persistent vomiting; if there is delirium; if intestinal obstruction exists; if a chill has occurred; if the pain and rigidity are very marked; if a mass can be felt in the right iliac fossa or by rectal examination; if there is marked abdominal distention; if there are evidences of pus formation; if the patient is growing worse; if there is or has been shock; or if the pain suddenly passes away without the use of opiates.

In an ordinary mild case, not seen early, in which none of the above-named conditions or symptoms exist, it is best to defer operation. Those who advocate operating upon every case consider such delay reprehensible and dangerous, point out that even in apparently mild cases gangrene or perforation may quickly occur, and cite striking cases to emphasize their belief. There is much force in this view, and it must not be hastily rejected. The choice, however, is not between a dangerous delay and a safe operation, but rather between a dangerous delay and a dangerous operation. It is a question of two dangers, and each side chooses the danger which seems to it the least. Richardson's elaborate study of 750 cases, showing a mortality of 18 per cent. in operations for acute appendicitis, determined us in the practice of the more conservative plan.

In an ordinary mild case of appendicitis in which operation is refused, it is a common custom to purge by means of Epsom or Rochelle salt. This practice was begun because of the belief that inflammation of the appendix is associated with fecal impaction in the head of the colon. This belief has been exploded, but the treatment is still used by some who regard it as beneficial. If the condition of the stomach prevents the administration of salines, high enemata are often given. My own belief is that if operation is refused, or if the surgeon determines to wait for an interval, he should not give a purgative, but follow the plan of treatment suggested by Ochsner to control peristalsis and favor limitation of infection. The patient is kept perfectly quiet, is placed in the Fowler position, no cathartics are given, no food or drink is administered by the mouth, and thirst is allayed by enemata of salt solution. Nutritive enemata may be given. It is also my custom to place a hot-water bag instead of an ice-bag over the appendix region.

To permit peristalsis favors diffusion of the infection; to prevent peristalsis is to favor the formation of encompassing and defensive adhesions. A purgative is very dangerous. It may cause rupture of the appendix. By causing peristalsis it diffuses the infection.

Many surgeons use the ice-bag, but I do not believe in it in these cases. We have already shown (see page 97) that cold as a remedy for inflammation is useful only in the brief stage of hyperemia, and when a surgeon sees a case of appendicitis there is certainly more or less stasis. Cold adds to stasis and does harm, and I am persuaded that the routine use of the ice-bag is responsible for some cases of gangrene. Again, cold actually antagonizes the migration of leukocytes and the formation of adhesions. For a number of years I have believed and taught that the ice-bag weakened resistance in appendicitis. These views seem to find confirmation in the article of Dr. A. M. Fauntleroy, U. S. N. ("The Ice-bag and Appendicitis," "Med. Record," August 3, 1912). The study of a number of cases led him to the conclusion that the ice-bag is often responsible for "a noticeable lack of effort on the part of Nature to wall off from the rest of the abdominal cavity the appendix." Further: "It was also noted in the ice-bag cases that there was a surprisingly low white count when one took into consideration the condition." These reports are most significant.

They are in strict accord with my own results. It is my belief that the use of the ice-bag antagonizes the limitation of the infection and favors dissemination of toxins and bacteria. I believe that its employment is a grave mistake.

Heat is a remedy which favors limitation of the process. It relieves stasis, draws leukocytes to the part and stimulates their activity, favors the formation of an encompassing barrier of phagocytic cells, and aids the cellular proliferation which leads to the formation of adhesions. Hence I prefer the hot-water bag.

The ice-bag, when applied before the diagnosis has been made, that is, in the earliest hours of the attack, when it might be thought to be most serviceable, allays pain and lessens rigidity in some cases almost like a full dose of opium, and hence masks the symptoms as does that drug.

Opium should never be given until the diagnosis is made. In the first place, it is not needed, for if the pain is so violent as absolutely to demand opium, operation should be performed. In the second place, opium masks the symptoms, makes the patient feel comfortable, and gives the physician an unfortunate and ill-founded sense of security. The pain about the umbilicus, if severe, can be distinctly and safely relieved by the administration of 30 min. of spirits of choroform every half-hour until three doses have been taken. Opium should not be given if the surgeon, having decided not to operate at once, is awaiting an interval, because it may prevent or delay the recognition of some disastrous change. If a patient refuses operation, it can be given.

When we are inclined to wait for an interval, the case should be seen again within six hours. We are accustomed to follow McBurney's rule, which is as follows: If on seeing the patient again, six hours after the first visit, the patient is worse, operate at once. If he is no worse there is no pressing danger.

If in twelve hours after the beginning of the attack the symptoms are not intensified, they will soon begin to abate; if the symptoms have become worse during this time, operate. If in twenty-four hours after the beginning of the attack the severity of the symptoms lessens, it is usually possible to wait for an interval; but if during the second twenty-four hours the abatement in the severity of symptoms has not gone on and there is doubt as to the condition, operate at once.[1] When the attack has subsided, and about three weeks or more have passed, the appendix can be removed with remarkable safety. After a patient has had two or more attacks of appendicitis all surgeons agree that the appendix should be removed.

If pus is present some surgeons delay operation in the hope that firm adhesions will form around the pus, and that the necessary operation will simply be the opening of an abscess. I do not believe it is safe to delay operation in a pus case. The pus may become limited, but it may instead pass up toward the liver or down into the pelvis. Delay is fraught with peril.

If only one attack has occurred, there may never be another, and the question arises, Should the appendix be removed after one attack? We do not know that a man has really recovered after purely medical treatment. Many cases reported as cured by medical means have subsequently required operation. As Lockwood[2] puts it, "To say that a man with appendicitis has been cured by medical means is in many cases equivalent to saying that a man with a stone in his bladder has recovered from calculus after the cure of a cystitis by rest in bed."

Even after a first attack, if the appendix remains tender or becomes tender after exercise, or if attacks of colicky pain occur, operate.

In some cases a single attack of appendicitis is followed by persistent dyspepsia and ill health, and in such cases operation should be performed. In the majority of cases, even after one well-marked attack, operation is

[1] For McBurney's views, see "New York Polyclinic," Jan. 15, 1897.
[2] "Brit. Med. Jour.," Jan. 27, 1900.

necessary. It is always necessary after two attacks. (See Operation for Appendicitis.)

Appendicitis cases which are far advanced in general peritonitis when seen by the surgeon some operators decline to touch. If we make a custom of operating on such cases we will lose very many, but will save some few, and these few would have died if we had not operated. To operate spoils statistics, but occasionally saves lives. The operation should consist of a simple incision to relieve tension and afford exit to infected fluids—rapid removal of the appendix if it is easily accessible, otherwise leaving it alone—and drainage of the pelvis. After such an operation the patient is placed in Fowler's position and a continuous stream of salt solution at low pressure is caused to trickle into the rectum. (See Murphy's Treatment for Peritonitis, page 1024.)

Appendicitis in a child is treated exactly as in an adult. Appendicitis in the pregnant woman is treated as in the non-pregnant. Early operation is particularly indicated, and it is not proper to induce premature labor in a patient far advanced in pregnancy unless there is general peritonitis. Then it is proper to empty the uterus—primarily, to obtain drainage and to give the patient a chance for life, and secondarily, to obtain a living child.

When operating upon a woman for appendicitis, bear in mind that ovarian, tubal, or uterine disease may have preceded, actually caused, or resulted from the appendicitis; examine the adnexa and remove them if necessary.

An operation for tuberculous appendicitis is rather apt to be followed by a fecal fistula. An ordinary laparotomy is sometimes followed by cure, but the rule of an operator should be, when possible, to remove the appendix and resect the diseased bowel. Andrews[1] mentions as expedients suited to special cases of tuberculous disease: total exclusion; partial exclusion; lateral anastomosis, and the formation of an artificial anus.

**Intestinal Diverticula.**—Congenital diverticula sometimes exist in the duodenum. Pressure diverticula may arise in the small intestine; they are, as a rule, small and multiple. The descending colon and the pelvic colon are the most common seats of diverticula. They occur at any portion of the circumference of the tube, are usually multiple, and vary much in size. Occasionally a diverticulum becomes enormous. A diverticulum contains fecal matter and, perhaps, fecal concretions. When free from acute inflammation and when unobstructed a diverticulum may cause no symptoms whatever. A diverticulum may inflame, suppurate, cause pericolonic suppuration, perforate, or become the focus of a great fibrous area (fibromatosis), which is usually mistaken for cancer.

**Acute diverticulitis** occurs in "adults, mostly in males in midlife and given to obesity" (Joseph Ransohoff, in "Annals of Surgery," August, 1913). There may be catarrhal inflammation. An abscess may form about the diverticulum. Perforation may occur, followed by abscess or general peritonitis. As most diverticula are in the lower colon the symptoms are usually left sided and strongly resemble appendicitis. Suppuration about the sigmoid does not of necessity arise from acute diverticulitis. Acquired diverticula do not exist in children, and yet Ransohoff (Ibid.) reported cases of acute perforating sigmoiditis in children.

**Meckel's Diverticulum.**—(See page 988.)

**Treatment of Acute Diverticulitis and of Perforating Sigmoiditis.**—As for appendicitis.

**Fibromatosis of the Colon.**—This condition produces symptoms strongly resembling those caused by carcinoma, and many cases have been operated upon under this conviction, the truth having been discovered by microscopical

[1] "Annals of Surgery," Dec., 1901.

examination of the specimen. It is an inflammatory condition most often met with in the pelvic colon. A large, hard mass forms and constriction occurs. The mucous membrane is thrown into deep folds and ulceration occurs in the hollows.

The fibromatosis is a reaction to infection. The infecting agent gains entrance through a desquamating area, a wound of the mucous membrane, an ulcer, or an inflamed diverticulum. Even when cancer exists there may be extensive fibromatosis about it, the area of real malignancy being much less than the induration suggests.

Fibromatosis may arise at any age, but is most common during and beyond middle age. There is continuous abdominal uneasiness now and then rising to pain. The general health deteriorates. The victim suffers from habitual constipation, but occasional attacks of diarrhea occur. Blood and mucus are at times found in the stools. If the constricting mass is within reach of the finger, it will be found that the induration is beneath thick and soft mucous membrane. If it can be seen by the sigmoidoscope, the folds of mucous membrane will be obvious. Abdominal palpation often detects the mass, which is sausage shaped. (See Miles, in "A System of Surgery," edited by C. C. Choyce.)

**Treatment.**—Resection and anastomosis if possible. If real obstruction exists, or if the mass seems irremovable, do colostomy. After colostomy the mass usually shrinks greatly and may actually disappear.

**Congenital Idiopathic Dilatation of the Colon (Hirschsprung's Disease; True Megacolon).**—This condition is of prenatal origin. The large intestine is chiefly involved. The rectum and small bowel seldom suffer, "and in more than one-third of all the cases the sigmoid flexure is alone involved" (Finney, in "Surg., Gynec., and Obstet.," June, 1908). There is no definite mechanical obstruction demonstrable at autopsy or operation. It is in this that Hirschsprung's disease (true megacolon) differs from pseudomegacolon (Finney, Ibid.). The diameter may reach 6 or 8 inches, and the colon may seem elongated and be in loops. Dilatation and hypertrophy produce marked changes in the wall of the gut. The condition may be obvious in early life or it may not become so until adult years, being aggravated and developed, but not caused by habitual atony of the bowel. The supposed cause is an anatomical anomaly (perhaps elongation) leading to looping of the colon, a muscular aplasia leading to dilatation and valve formation. Various causes have been suggested.

The victim of this condition is obstinately constipated and has a distended abdomen, usually from early infancy, although, as previously stated, the condition may not manifest itself until childhood, youth, or even adult life. It is most difficult to get the bowels to move at all. Gay reported a case in which there was no bowel movement for three months. Periods of several weeks without a movement are by no means uncommon. Now and then an attack of diarrhea may cause the emptying out of great quantities of feces. The abdomen is enormously distended and the patient is emaciated.

The abdominal veins are distended and the rectus muscles may be separated. In Finney's cases (as in some other reported cases) the cords of distended gut could be seen or felt to be more prominent on one side than on the other. There is no abdominal tenderness and pain is absent unless there is diarrhea. Borborygmus is often very loud. Vomiting is rare. The urine shows a marked increase of indican.

The disease does not directly cause death, but the ill-nourished condition lessens the chance for recovery from any attack of illness.

**Treatment.**—Medical treatment consists of the ordinary plans for combating constipation. Some surgeons have removed almost the entire large intestine; others have performed entero-anastomosis; others, colopexy; others

have established a permanent artificial anus; others have made an artificial anus preliminary to entero-anastomosis. Finney believes that the operation of choice is resection of the affected gut followed by entero-anastomosis.

**Splanchnoptosis (Visceroptosis).**—Coffey ("Surg., Gynec., and Obstet.," Oct., 1912) points out that in man special provisions are made to keep the viscera from riding down because of the upright posture. He names four forms of support: (1) Peritoneal fusions before birth to the parietal peritoneum; (2) a shelf above each psoas muscle; (3) tone of the abdominal wall; (4) packing of subperitoneal fat which regulates intra-abdominal pressure. Visceroptosis is generally said to be due to relaxation of the abdominal walls and decrease of intra-abdominal tension, which leads to gradual stretching of suspensory ligaments and finally to movement of the viscera downward. The prolapse may involve all the abdominal viscera, one viscus, or several viscera. According to Coffey (Ibid.), when the intestine descends kinks occur at the junctions of fixed and movable parts, and general ptosis is an attempt on the part of Nature to prevent intestinal kinks. Prolapse of the stomach is known as gastroptosis (see page 976); prolapse of the liver, as hepatoptosis (see page 1039); prolapse of the spleen, as splenoptosis (see page 1065); prolapse of the kidney, as nephroptosis (see page 1275); and prolapse of the intestines, as enteroptosis or *Glenard's disease* (see below).

The causative relaxation of the abdominal walls is most common in women, but is by no means confined to that sex. It may be produced by ascites, pregnancy, muscular effort, febrile maladies, or wasting diseases. In some cases no cause can be assigned. Such a relaxed abdomen may or may not be thin. The fascial strands and muscular fibers are stretched, and usually attenuated and separated, the belly bulges downward and forward, and a viscus or the viscera follow because of lack of support.

**Enteroptosis, or Glénard's Disease.**—This disease is a prolapse of the intestine. It may be but a part of ptosis or prolapse of all the abdominal viscera; it may exist alone; it may be associated with movable kidney, prolapse of the stomach, of the liver, or of the spleen.

In Glénard's disease the intestines occupy the lower portion of the abdomen, and the belly below the costal margins is flat, is dull on percussion, and the pulsations of the aorta are very evident. The right portion of the transverse colon begins to descend first, and other portions of the intestine follow. The splenic and hepatic flexures are elongated, and sometimes there is venous engorgement of dependent parts of the mesentery (Lambotte, in "Presse Med. Belge," Nov. 24, 1901). The victims of this disease are dyspeptic, anemic, and neurasthenic. Normally the tenth rib is firmly attached by fibrous tissue to the ninth costal cartilage. In enteroptosis the tip of the tenth rib is freely movable and obviously separated from the ninth costal cartilage (*Stiller's sign*). The *x*-rays used after a bismuth meal are of the greatest help in diagnosis. The ptosis may arise without apparent cause, but may follow the wearing of ill-fitting corsets, falls, blows, lifting heavy weights, or prolonged vomiting. The dyspepsia is due to dragging on the duodenum, the tube becoming flattened out (A. K. Stone). The flattening of the duodenum may be followed by kinking of the pylorus, and in such a case the stomach dilates, otherwise it does not dilate. Where a movable portion of the gut has a junction with a fixed portion a kink forms and intestinal stasis ensues.

**Treatment of Visceroptosis.**—In many cases medical treatment is of benefit. The following is the usual plan: Employ lavage, abdominal massage, and electricity; order a proper abdominal corset; insist on regular exercise, and treat the anemia and dyspepsia. Surgery is resorted to if intestinal stasis exists and cannot be relieved by medical and dietary treatment. The surgical methods

applicable to special organs are discussed under those headings. When the intestines are ptosed and there is stasis there are two commonly employed surgical plans, and each has its advocates: (1) The suture of the prolapsed intestine to some adjacent structure or the shortening by sutures of its supporting ligaments. (2) Plastic operation to lessen the area of the abdominal wall and thus increase intra-abdominal tension. Depage makes the abdominal wall less in both directions. Webster, in cases with separation of the recti, resects and sutures the fascia of the muscles. I believe that Depage's method of shortening the diameters of the abdominal wall cannot permanently succeed, because the viscera, hanging to relaxed ligaments, will eventually stretch the wall; it will be stretched easily because of damage done to its nerve-supply by operation, and hernia will be apt to occur, and if it should, the patient will be worse off than before operation. Of course, whatever operation is done, diastased rectus muscles should be approximated and sutured. For ptosis of the small intestine the mesentery may be shortened, as suggested and performed by Davis, of Omaha, in 1897.

In prolapse of the transverse colon good results are said to have been obtained by attaching the splenic and hepatic flexures to the abdominal wall (*Lambotte's operation*). The surgical treatment of ptosis of the stomach is considered on page 977; of the liver, on page 1040; of the spleen, on page 1065; of the kidney, on page 1278.

## The Peritoneum

**Acute Peritonitis.**—Peritonitis, or inflammation of the peritoneum, is a common and usually a very dangerous disease.

Aseptic irritation by a traumatism or a chemical irritant produces *aseptic peritonitis*, a condition which is strictly limited; which may produce local pain and tenderness; which may cause aseptic fever from the absorption of fibrin-ferment and the products of tissue change; which leads to the formation of temporary or permanent adhesions, and which is, in reality, a process of repair.

"Peritonitis," as the term is used by the surgeon, is always due to bacteria. Bacteria may reach the peritoneal cavity by means of an abdominal wound or the entrance of foreign bodies; by extravasations from the stomach, bowel, vermiform appendix, gall-bladder, urinary bladder, kidney, Fallopian tube or uterus, or by the passage of micro-organisms through the damaged walls of any of these viscera or structures; by way of an open Fallopian tube; from the breaking of an abscess into the peritoneal cavity; from areas of necrosis due to volvulus, strangulation, or intussusception of the intestine; twisting of the pedicle of an ovarian tumor, a floating kidney, or a floating spleen; blocking of a mesenteric vessel by a thrombus or an embolism; gangrene of the pancreas or spleen, and fat-necrosis.[1] In some cases the peritoneum may contain a point of least resistance, and bacteria contained in the blood reach this point and produce infection. It was once taught that cold could produce peritonitis, but it seems probable that it can only act by producing an area of least resistance. The capacity of the rheumatic poison to produce peritonitis is doubtful.

The peritoneum, as Byron Robinson pointed out and Fowler confirmed, is, in reality, a great lymph-sac, and peritonitis is lymphangitis. "When the peritoneum is infected the lymphatics furnish an exudate which clots in the lymph-channels, blocks them, and limits or prevents absorption. This blocking of the lymph-channels serves to preserve the life of the subject, on the one hand, while a failure in this respect, either because of the enormous and overwhelmingly rapid increase of septic material and the large

[1] See Park's "Surgery by American Authors."

size and number of channels necessary to destroy and obstruct, on the other hand, permits the destruction of the organism."[1] Absorption takes place most actively from the region of the diaphragm, hence peritonitis in this region is peculiarly fatal. Absorption takes place very rapidly from the intestinal region, although not quite so quickly as from the diaphragmatic area. Absorption takes place slowly from the pelvic region, hence peritonitis of this region is much less dangerous than is the disease in the intestinal region, and vastly less dangerous than is the disease in the diaphragmatic region (Fowler).

When severe bacterial infection of the peritoneum occurs, exudation of blood-liquor takes place, luekocytes migrate from the blood-vessels beneath the endothelial layer, particularly into the peritoneal cavity, and the causative bacteria rapidly spread about the cavity. The fibrinous exudate, in many infections, coagulates in masses on the free surface of the peritoneum, and thus serves a useful purpose by blocking the lymph-channels and hindering the absorption of toxins and bacteria. The fibrinous exudate may break down in a widespread suppuration or may be organized into an adhesion. In very virulent streptococcic infections a patient may die and there may be scarcely any coagulated exudation or may be none at all. Exudation and migration take place also in the subserous tissues and into the muscular coat of the bowel, and the segment of bowel which is attacked becomes paralyzed and distended with gas, the gas within causes it to rise up, and, as peristalsis is absent, obstruction occurs (James P. Warbasse, in "Am. Jour. Med. Sciences," July, 1905). Absorption of poison in peritonitis takes place in part from the peritoneal cavity and in part from the subserous tissues. Warbasse believes that the inflamed peritoneum is scarcely an absorbing surface, but in cases in which coagulated exudate has not formed or has been destroyed, it seems probable that it is an active absorbing surface, and absorption may occur from some regions, but not from others.

Various bacteria may be responsible for peritonitis, especially staphylococci, streptococci, pneumococci, and colon bacilli. The infections which spread most rapidly and widely are due to streptococci. In streptococcus infection the protective exudate does not coagulate, barriers of leukocytes are not heaped up, encompassing adhesions do not form, there is rapid absorption of toxins, and overwhelming systemic poisoning. Colon bacilli cause a very grave form of peritonitis, but less rapid and diffuse than that caused by streptococci—in fact, the process is often encompassed for a time by coagulated lymph, leukocytes, and adhesions. The omentum particularly is thickened, and is apt to apply itself about the area of infection. Staphylococci and pneumococci produce peritonitis which is more apt to be limited than that produced by colon bacilli. In most cases of peritonitis a mixed infection exists; for instance, colon bacilli and staphylococci or colon bacilli and streptococci. In some apparently severe cases of acute peritonitis cultures have remained sterile.

**Forms of Peritonitis.**—An accurate bacteriological classification is not as yet possible.

Peritonitis can be named, according to regions, *pelvic*, *subdiaphragmatic*, etc.; it can be divided pathologically into *diffuse septic*, *putrid*, *hemorrhagic*, *suppurative*, *serous*, and *fibrinoplastic* (Senn); it can be classified, etiologically, into *traumatic*, *puerperal*, *perforative*, *metastatic*, *scarlatinal*, etc.; and it can be divided, clinically, into *circumscribed suppurative*, *diffuse suppurative*, and *diffuse septic*.

**Circumscribed Suppurative Peritonitis.**—In this condition, which is frequently met with in appendicitis, the area of infection is circumscribed

[1] George R. Fowler, "Diffuse Septic Peritonitis," in "Medical Record," April, 14, 1900.

by coagulated exudate, leukocytes, and adhesions, and an abscess forms. After a time distinct localization becomes evident.

The *symptoms* of circumscribed peritonitis are pain, at first general and then local, tenderness in a particular region, muscular rigidity, distention, vomiting, rapid and often wiry pulse, constipation, fever, great weakness, and dorsal decubitus with the thighs flexed. After a time a distinct mass can usually be detected by palpation, and there may be dulness on percussion, local rigidity, irregular temperature, sweats, and possibly edema of the belly wall. An abscess, though limited for a time, is always liable to break through its walls and produce general peritonitis. Such an accident may be produced by muscular effort on the part of the patient or by injudicious palpation on the part of the surgeon; its occurrence is announced by shock, and the symptoms of general peritonitis quickly arise.

**Diffuse** or **general septic peritonitis** is apt to destroy life even before the peritoneum presents any marked change. Death ensues from the absorption of toxic alkaloids. Septic peritonitis may arise during puerperality, through lymphatic infection; it may be due to infection from without by an operation or an accident; to perforation of an ulcer; to gangrene of a portion of the intestine; to rupture of an abscess into the peritoneal cavity; or to migration of micro-organisms through a damaged wall of the bowel. Peritonitis due to perforation is called *perforative peritonitis*. Perforation is made manifest by pain, a chill, shock, or perhaps collapse. Gas may pass into the peritoneal cavity, and if it does so, the area of liver dulness may be lessened or abolished. Symptoms and signs of hemorrhage may arise. Diffuse septic peritonitis is announced by a very rapid pulse, which is at first wiry and later gaseous; a temperature which may at times be febrile, but which is apt to be subnormal or which soon becomes so; general abdominal pain and tenderness, dry tongue, delirium, persistent vomiting, constipation, and collapse. Rigidity exists, and also intestinal obstruction due to paralysis of the gut. Usually, but not invariably, there is distention. In puerperal peritonitis or septic peritonitis from operation there is often no severe pain. In perforative peritonitis there is acute pain. Victims of general septic peritonitis if unoperated upon usually die within five or six days.

**Diffuse** or **general suppurative peritonitis** differs clinically from diffuse septic peritonitis in the fact that it is less apt to be fatal and widespread. In fact, adhesions may form about an area representing a considerable portion of the peritoneal cavity. The causes of both are identical. In septic peritonitis death occurs from absorption of toxins before obvious pathological changes occur in the peritoneum; in suppurative peritonitis the microbes are fewer, are less virulent, or vital resistance is more decided, and suppuration follows marked changes in the peritoneum. In suppurative peritonitis the pyogenic bacteria are always present, and there exists in the peritoneum a wound or damaged area to constitute a point of least resistance.

*Symptoms.*—Chilliness or a rigor is common, followed by fever, the temperature rising to 102° or 104° F.; pain is intense, and is accentuated by motion and pressure; the attitude of the patient is assumed to relieve pain (he lies upon his back, with the shoulders raised and the thighs drawn up); there are vomiting, obstinate constipation, and rigidity of the abdominal walls, followed by distention when the intestine becomes paretic from septic poisoning. The pulse is rapid; is at first wiry, but may become gaseous. The constipation may be due either to tympanitic distention or to the shock and toxemia inhibiting intestinal peristalsis. Obstruction arises. Vomiting is frequent. In perforation gas often passes into the peritoneal cavity, and it may obscure the liver dulness; in tympanites without perforation the liver is apt to be pushed up and its dulness remains, but on a higher level. Pus unconfined by adhesions will

gravitate to the most dependent part of the peritoneal cavity. In some cases of suppurative peritonitis there is no tympanitic distention or rigidity; in some cases there is no elevation of temperature, in fact, the temperature may be actually subnormal.

**Treatment of Peritonitis.**—After an abdominal operation the patient may have pain, slight rigidity, constipation, nausea, flatulence, etc., and the surgeon is in doubt if peritonitis is beginning or about to begin. Our custom is in such cases to give a saline cathartic, which will empty the peritoneal cavity of fluid, will favor the elimination of microbes, and will combat inflammation. The old-time remedy was opium, but Tait denounced it as inefficient, and showed that it masked the symptoms and often created a false sense of security in the very midst of imminent dangers. The usual method of administering salines is to give 1 dram of Rochelle salt and 1 dram of Epsom salt every hour until a free movement occurs. Administer an enema of turpentine at the time the first dose of the saline is given. Atropin, eserin, and pituitrin may be useful (see page 992). This treatment will often abolish pain and distention and will perhaps prevent peritonitis after an abdominal operation. If, however, genuine peritonitis is known to actually exist no purgative should be

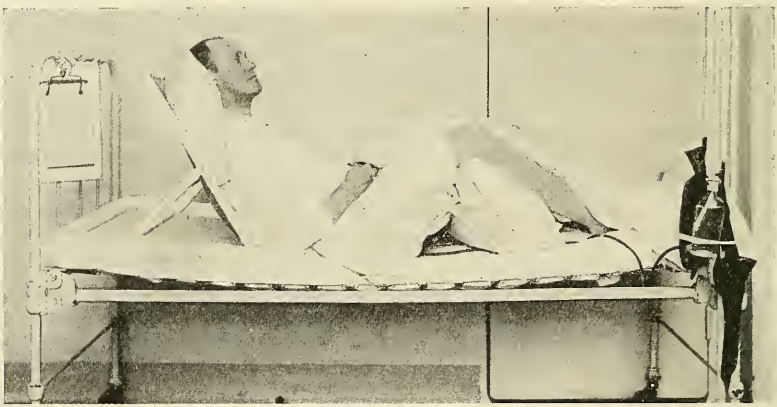


Fig. 616.—Murphy treatment for suppurative peritonitis.

given. It is a deadly dangerous thing to give one, as it diffuses infection. Operation is required. Prompt operation is the only hope in genuine postoperative peritonitis and any delay means certain death. When diffuse septic or suppurative peritonitis exists from any cause and the surgeon sees the case early (within thirty-six hours) the abdomen should be opened. In a perforative case operate even if the case is first seen later than thirty-six hours. If a nonperforative case is seen later it may be wise to use Ochsner's treatment and wait for localization (Stanton, in "New York Med. Jour.," Aug. 27, 1910). If a perforation exists, it should be closed. A perforated or inflamed appendix should be removed. Until recently it was surgical custom to break up adhesions, eviscerate, wash the belly with gallons of very warm salt solution, wipe out the space between the liver and diaphragm, wipe out the pelvis, wipe off the intestines, and remove masses of adherent coagulated exudate. We thus produced dreadful shock, tried to cleanse the peritoneal cavity when it is impossible thoroughly to cleanse it, carefully removed the exudate which was doing good by plugging the lymph-spaces, and yet we did not reach the infection inside of the lymphatics, which is, after all, the greatest source of danger. Then we drained through two or more incisions and put the patient recumbent in bed, and thus permitted infected material to flow up to the diaphragm, where

it is quickly absorbed. The mortality from this procedure was dreadful. John B. Murphy has taught us wisdom and has combined some of the conservative views of Ochsner with the use of the semi-erect position of Fowler, and with the continuous rectal irrigations that several advocated. Murphy's plan is founded upon the following principles:

First, that the initial lesion of the peritonitis should be got rid of as quickly as possible and with the slightest possible amount of handling. For instance, we should remove a gangrenous appendix; we should close a perforation in the bowel, etc. Flushing of the peritoneal cavity with gallons of salt solution is inadvisable. It cannot thoroughly cleanse the peritoneum; it may diffuse the infection to regions that it had not previously reached, and it may tear up adhesions. Inflammatory exudate should not be removed from the intraperitoneal structures. It is Nature's method of sealing the lymph-spaces, and if we remove it we open thousands of channels, previously sealed, for the dissemination of the infection. A drainage-tube should be introduced through the operation wound, and suprapubic incision should also be made and a drainage-tube be carried through this into the pelvis. When the operation is completed the patient should be placed in the semi-erect position, which is commonly called *Fowler's position.* This is done in order that the intraperitoneal fluids may gravitate away from the diaphragm, where absorption is extremely rapid, and into the pelvis, where absorption is much slower.

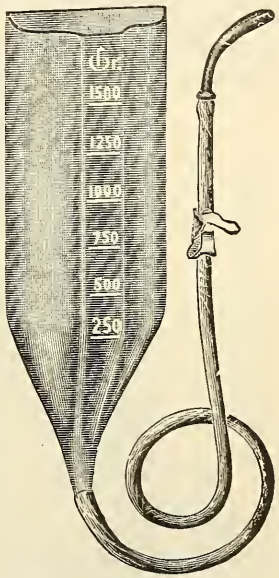



Fig. 617.—Proctoclysis apparatus consisting of fountain syringe, large rubber tube, and vaginal hard-rubber or glass tip (Murphy).

When the patient is placed in the bed quantities of warm salt solution are passed slowly into the rectum. The mucous membrane of the large intestine absorbs fluid with great rapidity when that portion of the gut is in its normal condition of moderate distention. Overdistention leads to spasm, which expels the fluid. Hence the fluid must be given at low pressure and administration should be continuous. The simplest sort of apparatus is shown in Fig. 617. It consists of a fountain syringe, a large rubber tube, and a rectal tip of hard rubber. The nozzle that is used is angled, has one opening on the end and several on the side, and this nozzle is passed so that the angle fits to the sphincter (see Fig. 619). The tube is strapped to the thighs by adhesive plaster. The hose that comes from the nozzle is attached to a reservoir, the base of which is hung from 4 to 6 inches above the level of the patient's buttocks; and the fluid, therefore, enters the rectum only about as fast as the rectum will absorb it. The reservoir is kept warm by bags of hot water hung about it. The fluid is allowed to enter continuously, unless it should run out from the side of the tube; if this happens, the flow may be cut off for a short time and then allowed to begin again. Gas from the bowel passes into the openings of the tube, and every now and then bubbles up through the reservoir. By this continuous, low-pressure instillation (*proctoclysis*) an enormous quantity of fluid is absorbed by the rectum. In some cases a number of quarts are taken up in twenty-four hours. The absorption of this fluid greatly increases the amount of urine eliminated, removes toxins, and stimulates the heart. The reservoir must not be high. Increase of pressure will cause expulsion of fluid and defeat the possibility of continuous administration. The plan so often followed of keeping the reservoir

high and limiting the flow by a clip on the tube is a mistake. Murphy says: "It should never have a headway of more than 15 inches hydrostatic pressure, and it gives the best and most uniform results at 4 to 7 inches" ("Jour. Am. Med. Assoc.," April 17, 1909). A straight tube is sometimes responsible for expulsion of the fluid, because it touches the posterior rectal wall of a patient in Fowler's position. Fig. 618 shows a more elaborate apparatus than that just described.

After the water has been entering the rectum for some time a profuse discharge of sour-smelling material comes from the drainage-tube. This discharge may be profuse for one day, two days, or longer, when its sour smell disappears and it greatly lessens in quantity. The outflow of this fluid from the wound means that saline fluid from the rectum has entered the lymph-spaces and flowed into the peritoneal cavity. Murphy thinks the lymph-current has been reversed. Whether this is true or not the peritoneum certainly seems to become a secreting instead of an absorbing surface, and the lymphatics are washed out. During the time that this treatment is pursued the patient

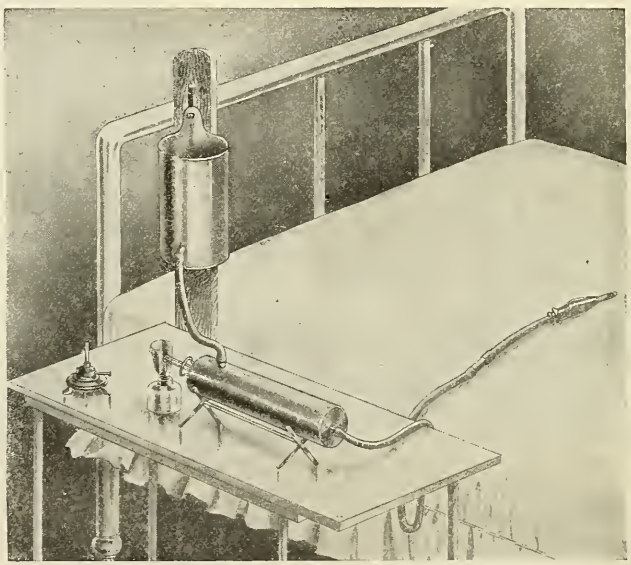


Fig. 618.—Alcohol or gas heater in operation, showing it properly connected. A short glass tube connects catheter to rubber tubing (Murphy).

has no food or water given him by the mouth. Stomach feeding is rigidly forbidden in order to prevent peristaltic movements. Small amounts of opium may be given to prevent peristalsis. If the patient is in a weak condition, stimulants or food can be given by the rectum, the solution in the reservoir being allowed to reach a low level, and then the material that it is desired to give being poured into the receptacle. Besides the above method of treatment antistreptococcic serum is usually given.

I am convinced that this method of treatment is of the greatest value, and that the principles upon which it rests are entirely sound, and I have had a number of striking successes from its employment.

If a case of diffuse peritonitis has lasted for three or four days and any sign points to localization, do not operate at once. Wash out the stomach, place the patient in Fowler's position, give salt solution by the rectum, and withhold purgatives and food. The inflammation may subside or may localize into

a circumscribed suppuration. When in a patient with peritonitis the skin is blue, cold, and moist, the pulse very rapid and weak, the abdomen immensely distended, and the temperature subnormal death is almost certain, and the only chance is the conservative plan set forth above. We wait in hope that the infection may localize in an abscess. (See Buchanan, in "Med. Record," Jan. 28, 1911, and Stanton, in "New York Med. Jour.," Aug. 27, 1910.)

A *circumscribed suppuration* is treated as follows: Open the abscess. It will be possible, if the abscess is adherent to the abdominal wall, to open the abscess directly without opening the peritoneal cavity. If this is not possible, after opening the abdominal cavity pack gauze pads in such a manner about the abscess as to prevent the diffusion of pus when the abscess is evacuated. After opening the abscess the primary lesion is sought for and, if possible, removed. The surgeon should not, in most cases, tear away the abscess walls in an attempt to find the primary lesion, but should rather let it go undiscovered. Pack iodoform gauze against the intestines to reinforce the barrier of lymph and insert a tube. It is frequently advisable to leave the wound wide open and drain by means of gauze.

Every patient with peritonitis requires stimulants.

**Tuberculous Peritonitis.**—Tuberculosis of the peritoneum is not very common. In 1170 autopsies in the Boston City Hospital tubercle existed in some region in 197, and in 14 of these the peritoneum was involved.[1] Primary local peritoneal tuberculosis is occasionally, though rarely, seen by the surgeon. In a great majority of cases of peritoneal tuberculosis other distant structures are involved. In about half of the cases the lungs are involved. In 28 cases reported by Bottomly[2] not one was primary. In every one of these cases the diagnosis was confirmed by the microscope, by the tuberculin test, or by autopsy. In most supposed cases of primary peritoneal tuberculosis another focus of disease exists, but is not demonstrable by clinical methods or has been overlooked. The disease sometimes exists as a part of general tuberculosis. Tuberculous peritonitis may be only a part of acute miliary tuberculosis. Bacteria may be swallowed with tuberculous food or a tuberculous patient may swallow tuberculous sputum and intestinal tuberculosis may result, the peritoneum being involved later. Peritoneal infection may follow a tuberculous lesion of the intestine, the bacteria may enter by way of the Fallopian tube, the initial lesion may be tuberculous appendicitis or tuberculosis of the mesenteric glands. The germ may lodge from the blood or lymph. The lymphatic form most commonly attacks the cecum. Tuberculous peritonitis is four times as common among women as among men, and most frequently attacks those between twenty and forty years of age, but I have seen it in a child of five and in a colored man of sixty. There are two groups of cases—the common chronic form and the rarer acute condition. The acute form begins suddenly, and such cases, as pointed out by Lejars, resemble acute appendicitis. In either the acute or chronic condition it is frequently

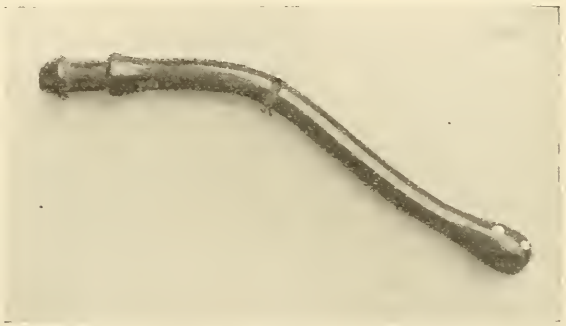


Fig. 619.—Tube with orifices filled with chalk in order to show openings better.

[1] Bottomly, in "Amer. Med.," Feb. 15, 1902. [2] Ibid.

the case that pulmonary phthisis exists. Cirrhosis of the liver is sometimes found with tuberculous peritonitis. There are three forms of chronic tuberculous peritonitis: the *ascitic*, the *fibrinoplastic*, and the *caseous*,[1] although, as a matter of fact, these so-called forms are only stages of the same disease. Tuberculous infection may exist for some time without causing symptoms, acute symptoms may suddenly arise, or intestinal obstruction may take place. Symptoms sometimes develop quickly after pregnancy. In other cases the symptoms appear gradually and progressively grow more positive.

*Symptoms of the Chronic Form.*—Usually the disease begins insidiously. The digestion is found to be disturbed, there is nausea, the bowels are out of order, the abdomen is distended and tender, there is occasional colicky pain, and the patient is weak, loses flesh rapidly, and becomes very anemic. Frequently pain is the symptom which leads the patient to seek advice. The pain may be present from the very beginning, it may arise after malaise and gastro-intestinal disorder have existed for some time, but sooner or later it will develop.

In many cases there is ascites, but the amount of fluid is rarely very great. In some cases the fluid is serous, in some seropurulent, in some purulent, and in some bloody. Chylous fluid occasionally exists because of fatty degeneration of tuberculous masses. Ascites may be either unconfined or sacculated by adhesions. In some cases, and especially in early youth, there is little or no ascites, and the condition is characterized by the production of a quantity of adhesions which bind coils of intestine to each other, to the omentum, to the stomach, liver, and other viscera. In this condition, which develops very slowly, small cavities are formed between adhesions and the spaces contain fluid and bacteria. This is the most chronic form of the disease. In any case of tuberculous peritonitis the mesenteric glands may enlarge. There is usually moderate fever, but there may be episodes of high fever and protracted periods of subnormal temperature, or the temperature may be slightly elevated in the evening and subnormal in the morning. When the temperature becomes markedly elevated, pain, tenderness, and distention notably increase. In some cases there is a continued fever resembling typhoid. Tumor-like formations may be detected. These formations may consist of indurated omentum, encysted exudate, or enlarged mesenteric glands. If diarrhea exists for a long period there is probably tuberculous ulceration of the gut.

In every suspected case a bimanual examination should be made under ether, in order to discover if there are any matted masses of intestine (Thomson).

In many cases a careful examination will detect tuberculous disease of other regions of the body, particularly of the lungs. If tuberculous disease of the lungs or pleura is detected, if tuberculous glands exist or have been present, if a nodule not due to gonorrheal inflammation is palpable in an epididymis, or if there are indurations in the prostate, the probability of the presence of tuberculous peritonitis is much enhanced. In many cases there is dilatation of the superficial abdominal veins. In some cases tuberculous peritonitis undergoes spontaneous cure. In the majority of instances death ensues from the tuberculous peritonitis directly or from associated or secondary disease in other organs.

If an intraperitoneal tuberculous area caseates, a large cold abscess may form, and such an abscess may break into the intestine or may be opened externally, and may be responsible for the formation of a fecal fistula.

In a case of tuberculous peritonitis intestinal obstruction may occur, the gut getting caught by bands or adhesions, or becoming a rigid tube because of the formation of tubercles.

[1] Parker Syms, in "Medical Record," April 2, 1898.

*Symptoms of the Acute Form.*—This is sometimes mistaken for appendicitis. It comes on rather suddenly, but a carefully elicited history will usually show the previous existence of malaise, gastro-intestinal disturbance, loss of flesh, and anemia. The symptoms are not so strictly localized to the right iliac fossa as in appendicitis. There are abdominal distention, a certain amount of rigidity, nausea and vomiting, colicky pain which may be very severe, general abdominal tenderness, fever, and exhaustion. It may be possible to palpate masses like tumors, or to feel nodules in the prostate or epididymis, or to detect tuberculosis in some other part.

*Treatment.*—In some cases there is a tendency to spontaneous cure, and in them medical treatment is of great service. The patient should be placed under antituberculous conditions, nutritious food and tonics should be administered (see page 230), the abdomen should be counterirritated and massaged, and purgatives should be given frequently. Guaiacol applied daily to the abdomen is thought by some to be of service, but I doubt it. A mixture is made of 1 part of guaiacol and 5 parts of olive oil; 1 dram of this mixture is rubbed into the abdomen, and the part is covered with a piece of flannel held in place by means of a binder. If medical treatment is not soon productive of benefit, the advisability of operating must be considered. It is a curious fact, but one confirmed by ample evidence, that after simple abdominal section, without the introduction of germicides and without drainage, at least 30 per cent. of the cases recover from the disease in from six months to one year. Some surgeons doubt the curative effect of operation. For instance, the late Professor Fenger was strongly of the opinion that many patients recover after operation, but not as a result of operation. In his opinion they recover because they are strong, free from fever, and well nourished, and because the disease tends to spontaneous cure. He further believed that some die from operation because the traumatism lessens the already lowered tissue resistance. The majority of surgeons, however, believe that operation in many cases tends to cure. Ochsner, in a paper before the American Surgical Association in 1902, apparently proved that simple incision and evacuation of fluid tends to cure. It is uncertain how an operation tends to cure. It has been thought that the ascitic fluid is a culture-medium for bacilli, and when it is withdrawn the bacilli die, but opposed to this view is the fact that aspiration is rarely curative. It has been suggested that the operation brings numerous phagocytes to the peritoneum; that it stimulates vital resistance; that it leads to the exudation of antitoxic serum. The entrance of air seems to play a definite and important part in effecting a cure.

The ascitic cases are most frequently benefited by operation. In encysted fluid operation often cures.

In cases in which there are numerous adhesions operation is not so likely to produce a cure. Great care should be exercised in separating adhesions, because the bowel is apt to be torn and a fecal fistula may result. It may be necessary to separate adhesions or short-circuit a portion of gut to relieve obstruction. Drainage should not be used unless a cold abscess exists. Not only is drainage of no service, but it is dangerous; death is more apt to ensue in a drained case and a fecal fistula will arise in nearly one-fourth of the drained cases. If operation is performed for cold abscess, tube-drainage must be used for some days. In a woman with tuberculous peritonitis the abdomen should be opened in the midline, and if the Fallopian tubes are tuberculous they should be removed. In a man the incision should be made over the appendix, and if this is tuberculous it should be removed. In either sex it may be necessary to resect tuberculous intestine or perform anatomosis because of stricture. (In confirmation of these views see W. J. Mayo, in "Jour. Am. Med. Assoc.," April 15, 1905.) The Mayos have performed 26 radical tubal operations

on cases of tuberculous peritonitis and 25 recovered. Of these, 7 had previously been operated on from one to four times by simple laparotomy ("Jour. Am. Med. Assoc.," April 15, 1905). In a very advanced case, in a case with notably high temperature, or in a case with marked and advancing tuberculosis in another region, an operation should not be performed except to relieve obstruction or drain an abscess. If a patient does not die within a few months after an operation, he will probably recover, and in most cases operation secures at least temporary improvement (Bottomly, "Amer. Med.," Feb. 15, 1902). The mortality from operation is 1 or 2 per cent. (Fenger).

**Pneumococcus Peritonitis.**—This condition is an unusual one. It is most apt to arise during the progress or after the termination of pneumonia or some other pneumococcic lesion, but is sometimes primary—is far commoner in females than in males and in children than in adults. Out of 74 reported cases, 57 were children under five years of age (Dr. Max von Brunn, in "Beiträge zur klinischen Chirurgie," Bd. xxxix, Heft i). In primary cases the bacteria may enter by way of the blood-stream or, perhaps, in some cases through the bowel wall or Fallopian tube. In secondary cases infection may arise from an adjacent pneumococcic area (pleura) or be carried by the blood from a distant point (*pneumococcic septicemia*). The condition may appear in a sufferer from otitis media. The symptoms in children are sudden in onset. The first symptoms are general abdominal pain, usually a continuous pain with colicky exacerbations, tenderness, rigidity, vomiting, elevated temperature, distention, and diarrhea. In a few days the symptoms abate and some of them disappear, although pain, tenderness, and rigidity are apt to localize at some point, particularly about the umbilicus, and may remain for a number of weeks. In such a chronic case physical signs of a fluid collection are usually demonstrable. In the chronic stage, as Brunn points out, there is seldom severe tenderness and there may be no fever at all, and a septic temperature is very rarely observed. Pus may form, and if it does, it contains pneumococci. Adhesions practically always form. These adhesions glue the intestines together and often encompass pus. Rapid emaciation and progressive weakness are always noted. In adults the symptoms are irregular and less characteristic than in children (Brunn). The prognosis is excellent.

*Treatment.*—Incision and drainage.

A **subphrenic abscess** is a collection of pus beneath the diaphragm. It is a rare condition. The pus may occupy a part of the lesser peritoneal cavity; it may be extraperitoneal (when it is of **renal origin**); in some cases it is contained in the area between the diaphragm, cardiac end of the stomach, and liver or spleen. It is impossible to classify accurately these abscesses by anatomical position. George A. Ross ("Jour. Am. Med. Assoc.," August 12, 1911) classifies them as right, left, anterior, and posterior. Most are on the right side, but even an abscess due to appendicitis may be on the left side. It is an unusual thing for such an abscess to break into the general cavity of the peritoneum, but it may break into the pleural sac (Maydl).

**Causes.**—Perforation of a gastric ulcer, perforation of the gall-bladder or gall-ducts, ulceration of the duodenum, disease of the liver, spleen, pancreas, intestine, appendix, or kidney, hydatid disease, internal injury, metastasis, external injury, caries of rib, disease of the pleura, general peritonitis, or portal infection may be responsible for a subphrenic abscess. We have abandoned the notion that the infecting source must be in the upper abdomen. Appendicitis is the most common cause. Charles A. Elsberg[1] has collected 73 cases of subphrenic abscess after appendicitis. He points out that the condition may arise from direct extension or by way of the lymph-channels, and may be either intraperitoneal or extraperitoneal, although in the majority of cases it is

[1] "Annals of Surgery," Dec., 1901.

intraperitoneal. In rare cases extension is by way of the portal vein. Ross claims that in most cases due to appendicitis the infection extends by cellular tissue directly upward from the lower peritoneal fossæ. In some cases infection ascends between the colon and the parietal peritoneum (the *parietocolic sinus* of our French colleagues). In all but 7 of Elsberg's cases there was suppuration about the appendix. The pus was thick and foul in all the cases. In 15 per cent. of them gas was also present, and in 25 per cent. of these cases the diaphragm was perforated. In 3391 consecutive cases of appendicitis operated on in the German Hospital of Philadelphia there were 30 cases of subphrenic abscess (Ross, "Jour. Am. Med. Assoc.," Aug. 12, 1911). Subphrenic abscess may develop soon after an appendicitis, it may develop extraordinarily late. Ashhurst's case arose four years after appendicitis ("Trans. Phila. Acad. of Surg.," 1910), 1 of Ross's cases, one year after (Loc. cit.). If ascending retroperitoneal infection exists during appendicitis, removal of the appendix does not arrest it (Lance, in "Gaz. d. Hôp.," 1909, lxxxvii). In 2 cases on which I operated the abscess developed after cholecystitis; in 2 others, after suppurative appendicitis.

The **symptoms** usually come on suddenly, but may do so gradually. There may or may not be abdominal symptoms. A patient with subphrenic abscess usually complains of pain in the lower part of the chest on the right side. Usually there is high temperature and often delirium, but, as Jopson ("Annals of Surgery," July, 1910) says, the temperature may be only moderately elevated and the pulse may be nearly normal. The area of liver dulness is, in many cases, distinctly enlarged, and there is tenderness in the lower part of the right chest when pressure is made through one or through several intercostal spaces. Frequently friction sounds may be heard about the region of the dome of the liver. Breath sounds and vocal fremitus are lessened. The signs are usually best heard posterior, but may be lateral or anterior. There is cough and bulging of the chest wall. Jaundice is absent in uncomplicated cases. Sometimes the symptoms are obscure or indefinite, and not accompanied by particular pain. If the abscess happens to contain not only fluid but also a considerable amount of gas—and about one-half of such abscesses do contain gas—not only will there be no increase in the area of liver dulness, but the normal area of dulness may be diminished or obliterated. The presence of gas may be due to some connection with an organ which contains gas or to gas-forming bacteria. It is very common for a pleural effusion to be associated with a subphrenic abscess. A pleural effusion will be preceded by or accompanied by symptoms pointing to the lung or pleura; and it is to be remembered that the area of percussion-dulness found in the pleural effusion shifts its position whenever the position of the patient is changed, which is not true of the area of dulness found in subphrenic abscess. When the abscess breaks through the diaphragm the patient collapses, cough and other thoracic symptoms develop; and if the abscess breaks into a bronchus the patient will expectorate pus. In subphrenic abscess the diaphragm of the diseased side is paralyzed—a condition rarely met with in liver abscess. There are general symptoms of suppuration and a swelling in the subdiaphragmatic region following some recognized causative condition. The history of chills with recurrent fever and sweats is rather indicative of abscess of the liver; but in abscess of the liver there is usually pain in the shoulder-blade of the right side, and this is rarely encountered in subphrenic abscess. The *x*-rays show that the diaphragm is elevated on the side of the lesion. The proof of the diagnosis is not, however, obtained until an exploratory incision has been made and the purulent matter has been found. Empyema and subphrenic abscess resemble each other. In empyema the upper limit of the fluid is concave; in subphrenic abscess it is convex. In empyema the flow of pus through an aspirating-needle will be most marked

during expiration; in abscess, during inspiration. The same is true of the rush of gas. In empyema the needle does not oscillate; in abscess it does.[1] If an abscess contains gas, percussion elicits a tympanitic note over a part of the cavity, and there is an alteration in the area of tympany with an alteration in the position of the patient. An abscess of the liver almost never contains gas and decidedly changes the outlines of the organ.[2] Empyema may follow subphrenic abscess.

**Treatment.**—Incision and drainage. The incision is made in the lumbar region if the abscess points there. In some cases it is made through the abdominal wall (epigastric region, iliac region, hypochondrium). In other cases the chest wall is incised, the ninth or tenth rib is resected, and the abscess is opened below the pleura or the pleura is opened, the parietal and diaphragmatic layers are sutured together, if possible, and the diaphragm is incised. If appendicitis was the cause, be sure the appendicitis is well; and if it was not, open the appendix region and drain freely (Elsberg). If it is necessary to open the pleural sac, first try to stitch the parietal to the diaphragmatic layer of the pleura, or, if this is impossible, protect the cavity with iodoform gauze to prevent infection.

## The Liver, Gall-bladder, and Bile-ducts

**Rupture and Wounds of the Liver.**—Rupture of the liver is due to very great force, and is usually accompanied by injury of other viscera. It may be produced by a blow, by a fall, or by the end of a broken rib. The superior surface or margin most often suffers. It is a very fatal accident. Out of 543 reported cases over one-half died of hemorrhage within twenty-four hours of the accident.[3] At least 80 per cent. will die if not operated upon. Wilms[4] collected 19 cases, and only 3 recovered after operation. Eisendrath[5] has collected 37 cases of suture of the liver for rupture and 22 of them recovered (59.5 per cent.). The first operation was performed by Willette in 1888. Out of Kraussine's 13 stab-wounds of the liver 7 died (Murphy, in "Practical Medicine Series," 1910, vol. ii). An attempt should certainly be made to save the patient by opening the abdomen and arresting hemorrhage, and in a suspected case an exploratory operation should be performed. A wound of the liver causes violent hemorrhage, which is usually rapidly fatal. Such a wound is apt to divide bile-ducts and allow bile to escape into the peritoneal cavity, and perhaps externally. Bile, if sterile, will do little harm to the peritoneum, but if it contains bacteria it will produce diffuse peritonitis. Even sterile bile is corrosive and may cause fibroplastic peritonitis. The symptoms of a rupture or wound of the liver are those of severe intra-abdominal hemorrhage, with collapse, accompanied by hepatic tenderness and respiratory embarrassment. Soon after the injury the abdomen is soft and flat, but it quickly becomes rigid and ultimately distended. The diagnosis becomes more probable when it is known that violence was applied to the hepatic region. Usually there is abdominal pain and often pain in the back. Sugar may appear in the urine. In a few cases after several days jaundice and skin itching have been noted. The area of liver dulness is usually increased. Patients do not always die from a serious traumatism of the liver. Some recover because operation has saved them. Some few recover without operation. This last fact is proved by reports of autopsies in which scars were found in the liver parenchyma (Nussbaum). The fatality which usually ensues on a liver injury may be due to hemorrhage or peritonitis. If a surgeon is called

[1] Wharton and Curtis, "Practice of Surgery."

[2] In a case of abscess of the liver secondary to appendicitis operated upon in the Jefferson Hospital the abscess did contain gas produced by gas-forming bacteria.

[3] Mercade, in "Rev. de Chir.," Jan. 10, 1902.

[4] "Deut. med. Woch.," Nos. 34 and 35, 1901. [5] "Jour. Am. Med. Assoc.," Nov. 1, 1902.

to a patient suffering from wound of the liver, he must open the abdomen to arrest hemorrhage. If a penetrating wound is suspected, it may be desirable to enlarge the wound in the abdominal wall layer by layer, in order to determine that the liver is wounded. If the left lobe of the liver is wounded, or if it is uncertain which lobe is wounded, the incision should be median. If the right lobe is wounded, a curved incision is made along the line of the costal cartilages. In some cases these two incisions are joined.[1] The convex surface of the liver can be reached by Lannelongue's plan. Lannelongue resects the eighth, ninth, tenth, and eleventh costal cartilages and draws the ends of the ribs well out. It can also be reached by Langenbuch's plan, that is, by cutting the coronary ligament and the right lateral ligament. This allows the liver to be pulled well up into the wound in the belly wall. The site of the wound can be discovered if the hepatic vessels are grasped between the thumb and a finger (the finger in the foramen of Winslow and the thumb in front on the gastrohepatic omentum). This completely arrests hemorrhage, and the blood that has gathered may be sponged out and the wound sought for in a clear field. (See Pringle, of Glascow, in "Annals of Surgery," Oct., 1908.) When the wound in the liver is discovered and well exposed, deep sutures of catgut should be inserted in the liver and the capsule should be stitched with fine silk (Schlatter). If sutures fail to arrest hemorrhage, the organ should be sutured to the belly wall and the wound in the liver packed with iodoform gauze. It is useless to try packing without first attaching the liver to the abdominal wall, because pressure will simply push the liver away and will not arrest the bleeding. The cautery is a very useful means of arresting bleeding. It should be avoided if possible in a large wound, because, even if it arrests primary hemorrhage, secondary hemorrhage may occur. After arresting hemorrhage, wash out the abdomen with hot saline fluid, insert drainage, and close the abdominal wound. In a case of the author's in the Philadelphia Hospital the liver was wounded by the sharp ends of fractured ribs. The abdomen was opened, a wound was found, and bleeding was arrested by suturing the liver to the belly wall and packing the wound. The patient died, and necropsy showed another wound on the posterior portion of the organ. The possibility of such an occurrence should not be lost sight of.

**Tumors and Cysts of the Liver.**—The liver may be the seat of primary carcinoma, sarcoma, endothelioma, angioma, lymphangioma, adenoma, fibroma, myxoma, or lipoma. Many tumors called adenomata are really adenocarcinomata. Secondary malignant growths are far more common than primary neoplasms—in fact, 96 per cent. of liver tumors are secondary. Primary cancer of the liver is found once in every 2000 autopsies (Eggel). The commonest variety is nodular, but the diffuse form, known as *cancerous cirrhosis*, may occur. The nodular form is most often encountered in the right lobe, and it has been found in persons under the age of twenty. Metastases occur early. "There is always more or less coexisting cirrhosis of the liver" (Leonard Freeman, in "Trans. Am. Surg. Assoc.," 1904). It takes origin from the hepatic cells. The frequency of cancer of the liver secondary to cancer of the stomach has already been alluded to. The commonest primary tumor of the liver is cavernous hemangioma. It is especially apt to take origin in the atrophying liver of an elderly individual. Primary sarcoma may arise at any age and may even be congenital. The growth is rapid and emaciation is soon noted. The liver enlarges, often greatly. Jaundice and ascites are rather rare. The patient soon becomes very weak. There is always pain. As Knott ("Surg., Gynec., and Obstet.," Sept., 1908) points out, the condition may simulate abscess, and if it arises in a middle-aged or elderly person can scarcely be differentiated from carcinoma.

[1] See Schlatter, "Beiträge zur klinischen Chirurgie," Bd. xv, Heft ii, 1896.

Knott ("Surg., Gynec., and Obstet.," Sept., 1908) has collected 59 cases of primary sarcoma from literature, and adds 14 reported by personal communications and 1 of his own, 74 in all.

He shows that 28 cases have been operated upon. In 9 the operation was exploratory and no attempt was made to remove the growth. In 19 the growth was extirpated, with 10 recoveries and 9 deaths. One of these patients was well after nineteen months, 1 after two years, 1 after seven months. Operation is indicated for a circumscribed growth.

Among the cysts occurring in the liver are blood cysts, congenital cysts, bile cysts, and hydatid cysts. Terrier and Auvray in 1901 collected 52 operations for hepatic tumors.

Angiomata have been removed successfully by hepatectomy, a cautery-knife at a red heat being used to cut through the normal liver tissue around the base of the tumor, the large vessels being tied with catgut. Enucleation is not feasible because of excessive hemorrhage. If a tumor is pedunculated, the base may be encircled by an elastic ligature held in place by a steel needle, and five or six days later the tumor may be cut across by the cautery.[1] I assisted Prof. W. W. Keen in such an operation.

Carcinoma of the liver has been extirpated, but it is seldom that a growth is recognized early enough and is found to be sufficiently limited to justify such a procedure. Operation is proper only when there is a limited nodule of primary cancer. In 1901 Terrier and Auvray collected 9 operations for primary cancer. In most cases there has been rapid recurrence or secondary growth, but Schrader's case was well at the end of seven years and Leonard Freeman's at the end of sixteen months. (For operative methods, see Leonard Freeman, in "Trans. Am. Surg. Assoc.," 1904.) Hunbald has collected 96 cases of resection of the liver, with a mortality rate of 26 per cent. Probably the best method of arresting hemorrhage is the use of suture ligatures of doubled catgut passed by round, blunt needles, as advised by Mikulicz.

**Hydatid cysts of the liver** may be of small size and productive of no signs or symptoms; or may be of large size and productive of the signs of tumor. In the epigastrium the mass may be prominent and fluctuate. In cyst of the right lobe the dulness is found in the axillary line and the growth encroaches on the pleura. In a large cyst fluctuation and hydatid fremitus may exist. Hydatid fremitus is a vibration imparted to the palpating fingers of one hand when the fingers of the other hand knock upon the cyst. There may be no discomfort produced by even a large cyst, but, as a rule, the patient suffers from a dragging sensation in the epigastrium and pressure symptoms. Suppuration in the cyst produces the symptoms of abscess of the liver and septicemia. Rupture of the cyst produces shock and even death. Rupture may take place into the pleural sac, the lung, or the peritoneal cavity. If the shock is recovered from, inflammation arises, the area of which depends upon the structures damaged. The escape of even a small quantity of hydatid fluid into the peritoneal cavity produces urticaria (*hydatid toxemia*). Aspiration for diagnostic purposes is not advisable.

**Treatment.**—Exploratory incision may be necessary to confirm the diagnosis, and the operation is completed at this time. After exposing the cyst it is packed around with gauze and a trocar is introduced. If there is a considerable thickness of liver tissue over the cyst, incise the liver by the cautery knife. When the fluid is evacuated the sac is incised and is drawn partly through the wound in the abdominal wall, and is attached to the wound margins (*marsupialization*). The endocyst can then be removed by the hand or by irrigation. A large drainage-tube is introduced.

**Syphilis of the liver** is a very able actor and often impersonates with

[1] Russell S. Fowler, on "Tumors of the Liver," "Brooklyn Med. Jour.," Dec., 1900.

surprising accuracy various other diseases. It may be congenital or acquired. The congenital condition may cause cirrhosis, miliary gummata, spots of fibrosis, and occasionally large gummata. Sometimes the liver manifestations of congenital or hereditary syphilis may be postponed for years (ten, fifteen, or more) and then appear as ordinary tertiary lesions. Acquired syphilis may cause hepatic disease in the secondary and in the tertiary stage.

In secondary syphilis there may be temporary jaundice due, Rolleston thinks, to catarrh of the smaller bile-ducts within the liver. It is possible, according to Rolleston, for temporary pericellular cirrhosis to exist in the secondary stage.

The tertiary lesions are the most common and important. Among these lesions are gummata and scars. They are most common ten years or more after the primary sore. Tertiary syphilis may appear as irregular patches of inflammation in Glisson's capsule (a condition apt to eventuate in hepatic sclerosis)—as a large solitary gumma (only one-eighth of cases of gumma present a solitary lesion)—or as multiple and usually small gummata. Gummata are most usual upon the anterior surface of the right lobe. A gumma adjacent to the common duct or the hepatic duct causes jaundice. A large gumma is often mistaken for cancer of the liver, and it is a curious fact that in many cases of hepatic gummata we are unable to obtain any history of syphilis. Syphilis may be mistaken for ordinary cirrhosis, but in the latter disease the general nutrition is more impaired than in the former, and vomiting of blood, dilated cutaneous veins, ascites, and indigestion are far more apt to be present (Archibald MacLaren, in "Annals of Surgery," August, 1908). The victims of gumma are apt sooner or later to develop jaundice, colicky pain, moderate fever, and palpable enlargement of the liver. The spleen may enlarge. The fever may be continuous or may occur episodically. In some cases it continues for weeks. It may be intermittent, preceded by a chill and followed by a sweat. Such a fever may be due to Spirochæta cholangitis. It may be due to absorption of toxic material from a breaking-down gumma. The Wassermann reaction is a valuable aid to diagnosis. A gumma sometimes undergoes secondary infection and an abscess forms. A gumma may rupture into the pleural or peritoneal cavity or some viscus.

**Treatment.**—Mercury and iodid will cure most cases. If these drugs fail, it is proper to remove the tumor, if solitary, by resecting the involved area of the liver. MacLaren (Ibid.) collected 9 cases of resection for solitary gumma and added 1 of his own. There were 2 deaths in this series. MacLaren's 10 cases added to Keen's 12 ("Annals of Surgery," Sept., 1899) and Cumston's 15 (quoted by Rolleston in his work on "Diseases of the Liver") make 37 cases.

If an area is opened for exploration and a solitary gumma is discovered, the abdomen should be closed and specific treatment be tried before resorting to resection, that is, if specific treatment has not been tried before.

**Abscess of the Liver.**—An abscess of the liver may be produced by bacteria, especially staphylococci and streptococci. These organisms reach the liver by the general circulation or, what is more frequent, are taken up from the intestinal tract and reach the liver by the portal circulation, or pass to the liver by the lymphatics. Appendicitis with lymphatic infection may result in hepatic abscess. A subphrenic abscess may break into the liver and thus induce a liver abscess. Liver abscess may directly result from peritoneal infection. The fact that abscess of the liver is in hot countries frequently preceded by amebic dysentery led to the presumption that the Amœba coli produces the abscess, and in a large majority of cases of tropical abscess amebæ exist in the pus or at least on the abscess walls. Habitual intemperance and constant overeating predispose to abscess of the liver. The disease may follow traumatism, dysentery, diarrhea, cholangitis, suppuration of a hydatid cyst,

gall-stones, typhoid fever, appendicitis, and a chill to the surface of the body.[1] Abscess of the liver may be metastatic, and such abscesses are multiple. It may be caused by foreign bodies and parasites. A tropical abscess is an abscess of the liver in an inhabitant of a hot country.

There are three forms of abscess of the liver: traumatic, pyemic, and tropical.

**Traumatic abscess** may result from a wound of the liver or may follow a contusion without a break of the skin. In the latter case bacteria from the blood are arrested in the injured liver tissue. Such an abscess is usually solitary. Streptococci, staphylococci, or colon bacilli may be found. Traumatic abscesses are more common in children than in adults, are situated superficially, and the symptoms are usually acute. Recovery is usually rapid and permanent after incision unless the causal injury brings danger or fatality.

**Pyemic Abscess.**—Multiple abscesses exist, but they may fuse into one. It is frequently due to suppurative inflammation of radicles of the portal vein, infected emboli forming and reaching the liver; it may follow ulceration of the intestine, hemorrhoids, or appendicitis.

Occasionally abscess may arise from the extension of an infective process, such as pylephlebitis. It may arise from cholecystitis or cholelithiasis with obstruction. In these latter cases both the Bacillus typhosis and the pneumobacillus of Friedländer have been found as the direct bacterial agent. Colon bacilli are a common cause. Abscess of the liver following appendicitis may be due to portal infection (*portal pyemia*) or to lymphatic infection. It is usually multiple, but in a case of mine in the Jefferson Hospital it was solitary, several cavities having probably joined to form one. Echinococcus cyst of the liver may suppurate and form abscess. I operated unsuccessfully on 1 such case which was brought to me by Dr. Hultsizer. The round-worm, the liver fluke, and the Balantidium coli sometimes cause abscess, and, finally, it has been observed in measles, epidemic influenza, and perforating ulcer of the stomach.[2]

**Tropical abscess of the liver** is rare in temperate climates, but is extremely common in the tropics. Its usual antecedent in either climate is dysentery. The reason for the great frequency of the disease in tropical regions is that the chief causative agent, the Amœba coli, is found widely distributed in hot countries; and that passive congestion of the liver is a common condition among the white inhabitants of tropical regions. It has been pointed out that tropical abscess is particularly common among white persons who abuse alcohol, the condition of passive congestion of the liver making that organ a nutritious soil for a fruitful infection. Predisposing factors are protracted malaria and chilling of the surface of the body.

Major Charles F. Kieffer, U. S. A.,[3] in a lecture on tropical abscess of the liver, stated that in his own experience he found, in a series of 33 abscess cases in soldiers, that dysentery was present in every case; and that in a second series of 25 cases in natives and civilians he elicited a history of dysentery in 22 cases. Some observers—notably McLeod—state that dysentery is the antecedent factor in 97.5 per cent. of cases. Kieffer points out that in all the figures allowance must be made for a number of latent dysenteries, as well as for cases in which no effort was made to elicit a history of dysentery one or two years previously. It is also to be remembered that a case of amebic infection of the colon may have been so mild in the beginning as to have caused but a transient diarrhea, which the patient may have forgotten. Amebæ occasionally exist in the colon without producing any dysenteric evidences. From 20 to

[1] G. B. Johnston, "Annals of Surgery," October, 1897.
[2] Major Charles F. Kieffer, U. S. A., in "Phila. Med. Jour.," Feb. 21, 1903.
[3] Ibid.

25 per cent. of severe amebic dysenteries lead to the formation of abscess of the liver, and that at least 85 per cent. of all tropical abscesses are due to infection with the Amœba coli. Occasionally, an abscess begins very soon after the dysentery; but, as a rule, it does not form for some time afterward —weeks, months, a year, or even two years.

When an abscess of this sort forms in the liver that organ becomes enlarged and congested, and an area or areas of necrosis exist in it. But one abscess may be present; there may be an abscess with satellite abscesses about it; several abscesses may coalesce, making a very large cavity; or genuine multiple abscesses may exist. In about 70 per cent. of cases, however, the tropical abscess is solitary.

The right lobe of the liver is the region most frequently involved. The abscess is found in the right lobe in at least 90 per cent. of cases, and it is more often toward the convexity of the liver than toward the base.

An abscess of the liver contains characteristic and peculiar material; it is different from the pus found in other abscesses, and, in fact, is not pus, but is necrotic liver substance. Liver abscesses due to pyogenic organisms contain true pus; a tropical abscess, free from pyogenic infection, does not. Ordinary pus contains hordes of leukocytes, but the fluid of a tropical abscess contains very few. Riesman is of the opinion that the reason there are so few leukocytes is that the abscess contains a substance that, by chemotaxis, repels leukocytes. The matter is of a reddish-brown color, is thick, and frequently contains some blood. Occasionally it is offensive in odor. Microscopic examination shows it to contain portions of necrotic liver tissue, some liver-cells that are not destroyed, elastic tissue, blood, pus-cells, and amebæ. On bacterial examination it may be found that the fluid is infected, containing staphylococci, streptococci, or pyogenic bacteria. In about 20 per cent. of the cases the matter contains neither bacteria nor the Amœba coli. In over 60 per cent. of the cases the matter of a recently opened abscess is free from bacteria. In cases in which the fluid is sterile it is possible that bacteria were originally present, but have died. The reason for the death of micro-organisms in this matter is in great doubt, because, as Riesman points out, bile cannot kill them and organisms may be grown in the fluid. In the large majority of cases amebæ are readily demonstrable in the matter; but that in some few cases it is necessary to rub a piece of gauze on an abscess wall in order to obtain amebæ, and that in others they can be demonstrated only after the abscess has been discharging for some days. The causative rôle of the amebæ has been doubted by some observers, but most surgeons who have had experience in the tropics believe it to be a fact.

The *symptoms* may be very definite and positive; they are frequently misleading and obscure; and in some cases nothing whatever directs the surgeon's attention to the liver until the patient passes a huge quantity of puriform fluid at stool or coughs up an enormous amount of the characteristic material. If rupture takes place, death usually ensues. As a rule, the symptoms of a tropical abscess are positive and marked.

Kieffer sums up the chief symptoms under four heads: *fever, sepsis, enlargement of the liver*, and *pain*. In about three-fourths of the patients fever and sweats are definitely present; in about one-fourth they are absent or are very trivial. The type of fever met with is what has been previously spoken of as hectic. Usually there is an evening rise, preceded by a chilly sensation or by a chill; and as the temperature begins to fall, toward morning, there is a profuse sweat. It is seldom that there is any violent chill, though there are frequently slight ones. The sweats are extremely exhausting. They may occur either during the night or in the daytime, according to the time in which the patient sleeps. Kieffer says that they should not be called

night-sweats, but rather *sleeping sweats*. In very chronic cases there may be no pyrexia. As a rule, the temperature resembles that of malaria, but it is not controlled by quinin and the blood is free from malarial parasites. Sometimes the temperature suggests typhoid, with the exception that from time to time there are episodes of subnormal temperature. The patient loses flesh and strength, the appetite fails completely, and the skin becomes pasty or dirty yellow.

The entire liver is usually enlarged, and the enlargement may be detected by percussion, and in some cases a hard, smooth area can be palpated. Sometimes the liver reaches as high as the third rib anteriorly, or to the spine of the scapula behind, and it may extend downward to the anterior superior spine of the ilium. It is rarely, however, that the enlargement takes place in a downward direction; it is usually upward. In many cases the right side of the chest appears to be rather full, and sometimes there is actual obliteration of several intercostal spaces. If an abscess becomes adherent to the surface, there may be skin edema and dusky discoloration. In rare instances, if a very large abscess comes near the surface, fluctuation may be obtained. By auscultation it is frequently possible to obtain friction sounds in the region of the diaphragm and the superior surface of the liver.

The liver becomes tender. This tenderness may be developed particularly by pressure upon the lower edge of the organ, and sometimes by pressure through the intercostal spaces. There is not always pain, but, as a rule, there is. The pain may be dull and heavy, but as the abscess nears the surface of the organ the pain becomes sharp and lancinating. The pain is persistent and is not strictly localized, but radiates to the back, the right shoulder-blade, and the point of the shoulder. Pain is increased by pressure, coughing, sudden or violent movement, and is sometimes felt in the esophagus when food is swallowed. When the upper surface of the liver is involved the patient breathes as if he had pleurisy; and pleurisy frequently does develop, with marked effusion.

Paralysis of the diaphragm rarely occurs in abscess of the liver, and the respiration is not much affected unless the diaphragm of that side and the pleura become involved, though the patient frequently has a dry cough. A severe cough suggests that the abscess is on the convex surface of the organ. Such a cough is aggravated by recumbency. Kieffer points out that the patient lies on his right side, and almost on the right front aspect, the shoulder being drawn down and the right knee drawn up to relieve the tension of the abdominal muscles. In about one-fourth of the cases of tropical abscess of the liver jaundice occurs. It is most apt to occur when the abscess is on the inferior surface. Jaundice does not occur unless the common or hepatic ducts are compressed or cholangitis exists. The leukocyte count is of no particular help in the diagnosis, as there may or may not be leukocytosis. The urine is usually scanty. Diarrhea is a common accompaniment, but constipation may exist, and nausea and vomiting are by no means unusual.

*Diagnosis.*—With an antecedent history of dysentery the diagnosis is easy. Without such a history, it is always difficult and may be impossible. In the tropics exploratory aspiration is freely used, but exploratory incision, if necessary, with subsequent exploratory aspiration of the liver after the organ is exposed, would seem to be safer and more certain.

*Symptoms of Traumatic Abscess.*—Similar to those of tropical abscess.

*Symptoms of Pyemic Abscess.*—The liver is enlarged and tender, there is slight jaundice, and the general symptoms of pyemia are present.

*Treatment of Tropical Abscess.*—If in doubt as to the diagnosis, make an exploratory incision, exposing enough liver surface to permit of exploration by finger and needle. If pus is not found, pack the wound with gauze to keep it

open, and when adhesions form explore again. The operation for abscess is incision and drainage. The *abdominal route* is used when the liver bulges front or when it extends well below the costal margin (McGill, in "Surg., Gynec., and Obstet., Nov., 1911). If the abscess is adherent to the parietal peritoneum and is not covered by liver substance open it at once. If it is not adherent, or is covered by a considerable layer of liver substance, make a ring of gauze about the periphery of the abscess cavity. The abscess may be opened at once within the ring, the gauze being a coffer-dam to protect the peritoneal cavity. It is safer to catch the gauze to the parietal peritoneum with two or three fine catgut sutures and wait for forty-eight hours before opening. This is an easier plan and just as safe as attempting to stitch the liver to the visceral peritoneum or to the parietal peritoneum. The operation consists in evacuating the pus by a trocar and cannula, incising the abscess, stitching its edges to the edges of the abdominal wound, irrigating, and inserting a drainage-tube. If the abscess is covered by a layer of liver tissue, after locating it by an aspirating cannula open into it by a cautery knife and arrest hemorrhage by packing. When the parietal and visceral layers of peritoneum are adherent, packing will arrest bleeding; if they are not adherent, packing will only push away the movable liver (John O'Connor). The *transpleural route* gives the best access to the right lobe, which is far and away the commonest region for liver abscess. The operation devised by McGill (Ibid.) may be at first exploratory; it avoids pneumothorax and empyema. It is performed as follows:

The ninth and tenth ribs are exposed by a curvilinear incision; the flap (which does not include the fascia of the muscles) is raised; 4 inches of the tenth rib are resected; the gutter left by the removal of the bone is closed by catgut suture. This gutter can be pushed against the diaphragm by two fingers of an assistant, and while the pressure is being made an incision is carried along near to the upper border of the eleventh rib. The incision goes directly into the peritoneal cavity through the layers of chest wall and through the diaphragm, but, as the parietal pleura is being pressed directly against the diaphragmatic pleura, pneumothorax does not occur. The edges of the chest wall and diaphragm are clamped together and sutured, the pleural cavity being thus closed. The liver is exposed and may be needled for exploration, or an abscess can be drained at once or after causing adhesions by gauze, as previously described.

Rogers and Wilson ("Brit. Med. Jour.," June 16, 1906) advocate aspiration and examination of the pus. If amebæ only are present, they inject a solution of quinin, a material quickly fatal to amebæ. The dose is 30 gr. of bihydrochlorate of quinin in a sterile solution. If the abscess holds less than 10 oz. of matter the quinin is given in 2 oz. of fluid; if it holds more, in 4 oz. of fluid. The authors report 2 cases cured by this method.

*Treatment of Traumatic Abscess.*—Same as for tropical abscess.

*Treatment of Pyemic Abscess.*—Surgery is usually futile, because multiple abscesses exist, but an operation should be performed in the hope that it may do good. In a case in the Jefferson Hospital in which abscess of the liver followed appendicitis the patient recovered after operation.

**Hepatoptosis (Floating or Movable Liver).**—Hepatoptosis may be congenital, but is usually acquired. In a congenital case certain ligamentous supports of the liver are absent. In the following discussion the acquired form is the variety referred to. This condition is rare. Ninety-eight cases have been reported.[1] It is a form of splanchnoptosis and is due to relaxation of the abdominal wall and stretching of the supports of the liver. It may occur alone, but it is more often a part of a general abdominal relaxation or of Glénard's disease, and often a kidney is movable, or uterine displacement or hernia may exist. The liver may descend into the lower abdomen, may

[1] J. H. Carstens, "Jour. Am. Med. Assoc.," May 17, 1902.

be upside down (Demarquay), may rotate on its transverse axis (Griffiths), the anterior surface may become posterior, or the organ may lie with the superior surface in the right flank and the inferior surface looking to the left,[1] may be movable, or may be anchored by adhesions. It is most common in women. The liver is supported by ligaments and also by the inferior vena cava (which vessel is firmly adherent to the central tendon of the diaphragm—Faure), by the abdominal wall, and by the intestines (Glénard). The cause of the condition is in dispute. It can result from relaxation of the belly wall, relaxation of the ligaments, enteroptosis, great enlargement of the gall-bladder, increase in weight of the liver, atrophy of the connective tissue between the liver and diaphragm, pregnancy, the growth of a liver tumor, and tight lacing. Either a strain, cough, or the dragging of an adherent tumor may be the exciting cause.

**Signs and Symptoms.**—An abdominal mass may appear suddenly after a blow or a strain, and if it does appear suddenly there is always pain in the hepatic region, nausea, and weakness. When the condition comes on gradually there may be no symptoms for a long time, but, as a rule, there is some pain in the loin which becomes worse after exercise or effort. In rare cases jaundice appears, and occasionally there is ascites. The abdominal walls are relaxed and the signs of splanchnoptosis are manifest. When the patient stands, a transverse furrow of skin covers the lower part of the umbilicus (*Glénard's sign*). In most cases the shape, the movability, and the absence of the liver from its proper position are diagnostic. Even when the organ is dislocated and attached in its new situation, it is missed from its proper abode, and palpation outlines the characteristic shape. When the patient lies down the liver usually returns to place, and in most cases it can be restored by manipulation. In some cases, however, it will not return to place and cannot be restored by manipulation. A floating liver causes a recognizable enlargement in the right loin, and the mass usually moves on respiration.

**Treatment.**—In many cases the patient can be kept comfortable by wearing an abdominal support, and can be distinctly improved by the use of massage and electricity to the abdominal wall, the administration of tonics, and a course of forced feeding. If these means fail and the patient suffers, an operation should be performed. The operation of *hepatopexy* was devised by Marchant. He opens the abdomen and tries to restore the liver to its proper position. This can usually be accomplished. In some cases it can be done after adhesions have been separated. In other cases it can be only partially accomplished. After the liver has been restored, he sutures it by means of catgut or silk to the abdominal wall or costal cartilages, the stitches passing through the hepatic parenchyma and being carried through the liver by means of a round and blunt needle. The sutures attaching the liver to the belly wall are tied beneath the skin. Marchant scarified the dome of the liver in order to favor adhesions. Ramsay rubs the upper surface of the liver with gauze to promote adhesion and transfixes the round ligament with a suture which is carried around the cartilage of the seventh rib. In a severe case Depage advises us to associate hepatopexy with an excision of a portion of the abdominal wall to amend relaxation (*laparectomy*). If, in operating on a floating liver, it is found impossible to get the liver back into its normal position, fix it with sutures as near its proper abode as is possible. Terrier and Auvray report 11 cases of hepatopexy. One case died and 8 completely recovered.

**Floating Hepatic Lobe (Partial Hepatoptosis).**—This condition is not uncommon in cases of chronic disease of the gall-bladder and is most

[1] Terrier and Auvray, "Rev. de Chir.," Aug. and Sept., 1897.

often met in cholelithiasis. It is believed that it can be caused by tight lacing. A tongue-like projection forms upon the right lobe of the liver (*linguiform lobe, lacing lobe*). It can be palpated below the costal margin and the dulness of the mass on percussion is continuous with liver dulness. A linguiform lobe can usually be moved laterally and forward and backward; it is always tender and is sometimes the seat of pain.

**Treatment.**—When this condition is associated with gall-bladder trouble, it may disappear, or at least cease to cause pain, when the gall-bladder is drained by cholecystostomy. Langenbuch has successfully removed a linguiform lobe.

**Cholecystitis (Inflammation of the Gall-bladder).**—Inflammation of the gall-bladder is produced by infection. Healthy bile is sterile; and when bacteria are found in the bile, the condition is one of disease. Micro-organisms may find entrance into the gall-bladder by way of the blood, the bile becoming infected secondarily to the infection of the gall-bladder; or they may enter by way of the ducts, from the intestine. The conditions that follow infection depend upon the characteristic tendency and the virulence of the infecting germs. A trivial infection produces mucous catarrh; a more active infection causes suppuration, and possibly ulceration; a very violent infection leads to gangrene.

In most cases of cholecystitis an inflammatory swelling blocks the cystic duct, and obstructs it so that the bile stagnates in the gall-bladder. In many cases this condition lasts but a short time; and when the obstruction is relieved, bile flows down the duct. Occasionally, as a secondary consequence, cholangitis, or infection of the hepatic duct, follows.[1] Occasionally, also, the obstruction of the duct is not relieved, and a quantity of clear, thin mucus gathers in the gall-bladder and overdistends it—the condition known as *hydrops*. The gall-bladder may likewise become distended with pus, constituting an *empyema* of the gall-bladder; and any overdistended gall-bladder may rupture. A gall-bladder may distend to a most enormous size. Terrier reported a case of distended gall-bladder in which the viscus contained 42 pints of fluid. F. W. Collinson ("Brit. Med. Jour.," May 29, 1909) reports the case of a woman thirty-one years of age who was tapped twice before operation and at each tapping 25 pints of fluid were withdrawn. At the operation 22 pints were obtained. Collinson's case arose from blocking of the common duct as a result of traumatism, followed by kinking of the cystic duct and subequent opening of the common duct. In cases of very chronic inflammation of the gall-bladder this structure becomes fibrous and contracts, until it may become no larger than the thumb, in which condition it may contain a very small amount of thickened bile. In some inflammatory conditions due to infection the bile mixes with thickened mucus, and micro-organisms form the nucleus upon which bile salts are deposited to form gall-stones. As the same author points out, cholelithiasis may result from cholecystitis, and may cause chronic cholecystitis, because the stones existing in a gall-bladder are sources of irritation.

**Bacteriology of Cholecystitis.**—It has been proved by abundant observation that the fact that bile contains micro-organisms is no evidence that the gall-bladder is inflamed; but that when the gall-bladder is inflamed micro-organisms are demonstrable in the bile. We know that the bile is infected during the course of typhoid fever, and that it is frequently so in pneumonia. The colon bacillus is not unusually demonstrable in cholecystitis; and pus cocci, either in pure culture or mixed with other germs, constitute the most common cause of the inflammation. It is probable that bacteria entering the gall-bladder and not being particularly virulent produce

[1] Joseph McFarland, "Proceedings of the Phila. County Med. Soc.," Sept., 1902.

no immediate harm when the flow of bile is unobstructed, though even then they may become the nuclei of gall-stones; but if the bacteria are very virulent they may actually lead to obstruction. Stagnation of the bile favors infection, and infection may be the cause of stagnation. Each influence reacts upon the other and aggravates the other, and it seems more than possible that infection of the gall-bladder is to be regarded as serious only when there is obstruction to the outflow of bile. The same variety of germ may, under some circumstances, cause catarrhal, and under others suppurative, inflammation; that is, when bacteria are virulent and tissue resistance is slight, suppurative cholecystitis results; but when the bacteria are not virulent and the tissue resistance is powerful, the gall-bladder is not infected at all, or only catarrhal inflammation is produced. I operated upon a case of acute suppurative inflammation of the gall-bladder three weeks after the termination of an attack of typhoid fever. The culture taken from the gall-bladder showed an unidentified bacillus, which was not the colon bacillus or the paracolon bacillus, and which was not identical with the typhoid bacillus or the paratyphoid bacillus. It strongly resembled the typhoid bacillus, but possessed no agglutinative power (the author, in "New York Med. Jour.," April 8, 1905).

A patient in the medical ward of the Jefferson Hospital was supposed to be developing a typhoid relapse, but no fresh spots appeared, and there were pain, tenderness, and rigidity in the region of the gall-bladder. I operated and found the gall-bladder full, dark colored, and surrounded by numerous recent adhesions. It could be emptied slowly by pressure. There was no pus. It was drained and the symptoms promptly passed away and the man recovered. The culture was reported sterile. I cannot understand this finding, as inflammation undoubtedly existed. It may have been peritonitis rather than cholecystitis, but from what cause is unknown. No culture was taken from the peritoneal cavity. The finding of sterile bile at the end of an attack of undoubted typhoid is of interest.

**Catarrhal Inflammation of the Gall-bladder and Bile-ducts.**—This condition is known as catarrhal jaundice, acute or chronic, and is usually treated by the physician; but, as A. W. Mayo Robson points out, chronic catarrhal jaundice sometimes resembles the jaundice of organic disease, and is occasionally associated with gall-stones, malignant disease, or hydatid cyst. The same authority asserts his belief that chronic catarrhal jaundice usually results from interstitial pancreatitis and duct obstruction. This condition usually comes on without pain. If there is pain it means some complication. Robson ("Surg., Gynec., and Obstet.," Jan., 1908) names among such complications catarrhal cholecystitis, cholangitis, gall-stone, and duodenal ulcer. The jaundice is striking. There is loss of flesh and anemia, and the liver is enlarged and smooth. Robson tells us that if the gall-bladder is not shrunken from stone, and if there is great duct obstruction, the bladder will be distended. Chills and fever mean infective cholangitis. In a case of chronic catarrhal jaundice in which medical treatment fails, surgical treatment must be considered (cholecystostomy or cholecystenterostomy).

**Catarrhal Cholecystitis.**—This is a catarrhal inflammation of the gall-bladder usually without jaundice. The gall-bladder becomes thick and its mucous membrane is frequently plicated. Very thick mucus is secreted, which gathers in masses, and the descent of these plugs causes pain that is sometimes indistinguishable from that produced by the passage of a gall-stone. Such a plug may temporarily block the cystic duct. In catarrhal cholecystitis the gall-bladder is frequently distended, but rarely admits of palpation; and there are no adhesions to surrounding structures, unless gall-stones have been present (Robson). Catarrhal cholecystitis may lead to the

formation of gall-stones; may result from the presence of gall-stones; or may be found in cases in which gall-stones have been present, but have passed. In 1 case upon which I operated the gall-bladder was enlarged, thick, and without adhesions; the mucous membrane was convoluted; and the viscus was filled with thick, tenacious mucus, and the mucous membrane of the gall-bladder contained many minute concretions. In this case stone formation was probably beginning to follow upon catarrhal cholecystitis. In another case a woman had presented violent symptoms of gall-stone colic, and stones had been recovered from the feces; but on opening the gall-bladder no stones were found—only a condition of catarrhal cholecystitis. Jaundice is rare in catarrhal cholecystitis unless gall-stones are present; it is, however, occasionally noted. Even if jaundice does occur, it is slight and lasts but a short time. The painful attacks that occur during catarrhal cholecystitis are similar to gall-stone attacks; but the pain is less violent and of briefer duration, and jaundice is not apt to follow the passage of a plug of mucus and is apt to follow the passage of a gall-stone. Further, as Robson has shown, in catarrhal cholecystitis with gall-stones there may be tenderness, but there is rarely tenderness in uncomplicated catarrhal cholecystitis.

**Treatment.**—The majority of the cases recover under medical treatment. If a case fails to recover under medical treatment, one cannot be sure whether there are gall-stones or not; but an operation is indicated in either case. Cholecystostomy should be performed and the gall-bladder should be drained for a week or two. This treatment will almost always produce cure.

**Croupous Inflammation of the Gall-bladder and the Bile-ducts.**—This is an extremely rare condition, due to the formation of a thick membrane in the bile-passages, which causes obstruction to the flow of bile and spasmodic contraction of the gall-bladder. The symptoms are identical with those of gall-stones. Robson points out that a study of the evacuations may discover membranous intestinal casts; and that, as membranous enteritis is usually associated with croupous inflammation of the gall-bladder and bile-ducts, a diagnosis may thus be reached. The same author says that one may, in some cases, even find a cast of the gall-bladder in the evacuations.

**Treatment.**—If medical treatment fails, cholecystostomy should be performed and drainage should be employed for a considerable time.

**Suppurative Inflammation of the Gall-bladder and Bile-ducts.**—Adopting the classification of Mr. Robson, we divide these suppurative inflammations into simple suppurative cholecystitis, suppurative and infective cholangitis, phlegmonous cholecystitis and gangrene of the gall-bladder, ulceration of the gall-bladder and bile-ducts, pericystic abscess with adhesions, and certain consequences of these conditions, such as stricture of the gall-bladder and bile-ducts, perforation of the gall-bladder and bile-ducts, and fistula of the gall-bladder and bile-ducts. Suppurative inflammations of the gall-bladder and the bile-passages are due to infection by virulent organisms or to infection when the tissue resistance is at a low ebb.

One fact must strike the physician in regard to these cases; that is, that there is a strong similarity between the possible changes of acute cholecystitis and the possible changes of acute appendicitis. In the gall-bladder, as in the appendix, there may be a catarrhal inflammation, which may not advance beyond this stage, or which may advance into a more dangerous form; in each structure blocking and stagnation favor infection and aggravate existing infection; in each there may be suppuration, ulceration, gangrene, and perforation; in each there may be grave complications and disastrous and fatal consequences; and in each prompt surgical operation is usually life-saving.[1]

[1] The author, "Proceedings of Phila. County Med. Soc.," Sept., 1902.

**Simple Suppurative Cholecystitis.**—This condition is also spoken of as *suppurative catarrh* of the gall-bladder or *simple empyema* of the gall-bladder. It is a rare condition unless gall-stones exist or unless some infectious disease—especially typhoid fever—has antedated the condition. I operated for this condition upon a boy eleven years of age three weeks after the termination of an attack of typhoid fever. It is not only typhoid fever that may be causative, but also other continued fevers. No matter, however, what organism is primarily responsible—be it colon bacillus, typhoid bacillus, or what not—a mixed infection with pyogenic cocci usually takes place. Pyogenic cocci may alone be causative. In simple suppurative catarrh of the gall-bladder, when the duct becomes blocked, the condition known as simple empyema exists; and when hydrops of the gall-bladder undergoes suppuration simple empyema is produced.

In an ordinary case of suppurative catarrh following gall-stones one usually obtains the history of a number of attacks of biliary colic, the pain finally having become persistent instead of intermittent, and a definite swelling being palpable in the gall-bladder region. This swelling is tender on pressure. There are usually constitutional symptoms, sometimes trivial,

Fig. 620.—Gall-bladder filled with calculi. Removed by cholecystectomy.

often severe. The trivial symptoms are a somewhat rapid pulse, sweating at night, and some elevation of temperature. The more severe symptoms are chills, a remittent fever, and profuse sweats. The development of severe symptoms indicates that a dangerous change is taking place—usually ulceration of the gall-bladder, occasionally phlegmonous cholecystitis. Distinct jaundice is rare in simple empyema, though the patient usually shows loss of flesh, has a very poor appetite, and suffers considerably from thirst.

To distinguish an enlarged gall-bladder from any other intra-abdominal mass is sometimes difficult. Very large gall-bladders, such as have been placed on record by Collinson, Terrier, Lawson Tait, Gersuny, and others, may be mistaken for ovarian cysts. Alban Doran discusses such cases in the "Brit. Med. Jour.," June 17, 1905. An enlarged gall-bladder moves on respiration unless the mass becomes adherent to the abdominal wall, when it will cease to do so. An enlarged gall-bladder is sometimes mistaken for a movable kidney, and the diagnosis between these conditions is discussed in the section on Movable Kidney (see page 1277).

**Treatment.**—The gall-bladder should be opened and drained by the operation of cholecystostomy. After it has been exposed, it is packed about

with gauze pads, a considerable portion of the contents is removed through an aspirator, the gall-bladder is opened and irrigated with salt solution, and a search is made for any cause of obstruction in the cystic duct. This cause should be removed, and any gall-stones that are present should, of course, be taken away. The walls of the gall-bladder will frequently be found diseased and softened, so that it is impossible to apply stitches. In some cases, if the gall-bladder is badly diseased, it should be removed, but in others incision with drainage is sufficient.

**Recurrent Simple Empyema of the Gall-bladder.**—In this condition a person develops, at intervals, pain, fever, tenderness, and enlargement of the gall-bladder. Then the symptoms clear up and he is well for a time, but they again become manifest; and at last they may become persistent or violent because of the development of some complication. In these cases it is impossible, after a number of attacks, to palpate any enlargement of the gall-bladder; and when an operation is performed the gall-bladder is found shrunken, thickened, and deeply placed, containing some purulent matter, and strongly fixed to the surrounding structures by adhesions.

**Treatment.**—Cholecystectomy is usually the proper operation.

**Acute Phlegmonous Cholecystitis.**—Some call this condition *acute empyema*. It is extremely dangerous, and is apt to cause gangrene of the gall-bladder. It is due to infection by extremely virulent organisms. It may, even without perforation, produce rapid peritonitis and death. As a rule, in advanced cases perforation takes place. It is generally associated with the presence of calculi, but sometimes none are found; and the condition sometimes develops during typhoid fever or septicemia.

This disease begins with sudden and violent pain in the gall-bladder region. This pain usually radiates toward the right shoulder-blade, and soon becomes general throughout the abdomen. There are tenderness in and great rigidity over the gall-bladder region, thoracic respiration, exhausting vomiting, septic fever, and in some cases jaundice. If an operation is not performed promptly general peritonitis quickly takes the patient's life. In one case upon which I operated there were intense jaundice, tenderness, violent pain, abdominal rigidity and distention, chills, and septic fever; and when the abdomen was opened it was found that a portion of the gall-bladder was gangrenous and that a calculus projected through the gangrenous opening.

It is this form of cholecystitis that is especially likely to be mistaken for appendicitis. In making a diagnosis the situation of the primary pain is of importance, and likewise the situation of the tenderness; but a displaced gall-bladder or an abnormally situated appendix may lead to error. Acute phlegmonous cholecystitis is usually accompanied by absolute constipation, and the sudden onset and the abdominal distention may lead to the disease being mistaken for intestinal obstruction. It may also be confused with perforating ulcer of the stomach or of the duodenum.

**Treatment.**—In any case of doubt an exploratory incision should be made. If phlegmonous cholecystitis is found to exist, the gall-bladder should, whenever possible, be extirpated; but if the desperate condition of the patient forbids this operation, the bladder should be incised, surrounded with iodoform gauze, and a drainage-tube should be carried well up toward the cystic duct.

**Pericystic abscess** may follow infection of the gall-bladder. It is especially common in the condition known as recurrent simple empyema. When a pericystic abscess exists there are great localized abdominal tenderness and rigidity and the temperature is usually indicative of suppuration. The causative micro-organisms may have passed through a diseased gall-bladder wall, rupture not existing; or the abscess may follow ulceration or perforation of the gall-bladder wall.

**Treatment.**—Operation should invariably be performed, though it is frequently difficult. After a pericystic abscess has been drained it will be found necessary in some cases to extirpate the gall-bladder, whereas in others incision of the gall-bladder and drainage will prove sufficient.

**Infective Cholangitis.**—Cholangitis is usually inaugurated by infected bile, but when it arises during a general infection the bacteria may be brought by the blood. It may arise in a case of hepatic cirrhosis. Naunyn ("Deutsche medizinische Wochenschrift," Berlin, Nov. 2, 1911) says that most germs can cause cholangitis; that mixed infection may occur, and that during an acute general infection cholangitis is seldom recognized. The usual cause of infective cholangitis is gall-stones lodged in the common duct. Some maintain that duct-stones are causal, particularly in those cases in which a gall-stone acts as a ball-valve. A. W. Mayo Robson, though he believes that infective cholangitis does occur when the gall-stones are freely movable in the common duct, sets it forth as his experience that it is much more common in such cases to find gall-stones impacted in the common duct.

In such cases the patient gives a history of attacks of gall-stone colic without jaundice for several years, and then of attacks followed by temporary jaundice (see page 1051). Finally comes an attack that is followed by a chill and fever; and jaundice, varying in intensity, ensues upon this, and now, though it may fade, it seldom completely disappears between the attacks of pain. Robson points out that the interval between the attacks may be short or long, and that the rigors may be repeated daily or at uncertain intervals; that the gall-bladder is usually, but not always, contracted; and that after the condition has persisted for some time the liver becomes distinctly enlarged. There are tenderness over the gall-bladder or in the epigastric region, loss of flesh, and persistent jaundice which may vary in hue.

Infective cholangitis, even after it has lasted for a considerable length of time, may be recovered from; but it may pass on into an acute condition in which poisoning takes place from the biliary elements, suppurative cholangitis may arise, an empyema of the gall-bladder may develop, and there may be an abscess of the liver or some other dangerous or fatal complication. The ague-like attacks of infective cholangitis have been called by Charcot *intermittent hepatic fever* (see page 1051).

**Treatment.**—After an incision has been made the common duct is opened, the cause removed, and the duct drained; but, as Mr. Robson points out, the complication should be anticipated. When one finds that carefully applied medical treatment has failed to free the patient from gall-stones, they should be removed surgically.

**Suppurative cholangitis** is usually a development of the ordinary infective cholangitis, which has just been discussed. Among the other causes that Robson sums up are acute infectious diseases, particularly typhoid fever and influenza, cancer of the bile-ducts, and hydatid disease.

In this condition the liver enlarges notably and becomes tender. In some cases there is an empyema of the gall-bladder, but this is rare; in fact, the gall-bladder is usually very much shrunken. When, in a chronic case, there are enlargement of the liver, blocking of the common duct, and enlargement of the gall-bladder the inference is in favor of cancerous obstruction of the common duct. If the obstruction is due to cancer there will usually be little pain; but when it is due to gall-stones there will be violent attacks of pain, accompanied by rigors and fever, with deepening of the jaundice. In this disease there is always jaundice, usually unfading; but in cases of ball-valve gall-stone in the duct it will be mitigated from time to time (see page 1051). The patient suffers form septic fever and there is very rapid loss of flesh.

The condition is generally fatal unless operation is performed early. There is a strong tendency for abscess of the liver to form, and in 1 case upon which I operated a subphrenic abscess had developed.

**Treatment.**—Cholecystostomy with free and prolonged drainage. If an abscess of the liver exists it should also be drained. If gall-stones are gathered in the common duct they should be removed.

**Typhoid Cholecystitis.**—Typhoid bacilli were first found in the bile by Futterer in 1888, and typhoid cholecystitis was first described by Giroche in 1890. As previously stated, typhoid bacilli are usually present in the bile during, and perhaps are present months or years after, an attack of typhoid fever. They are not always present, however, for in a case of cholecystitis following typhoid on which I operated an unidentified bacillus was found ("New York Med. Jour.," April 8, 1905); in a case on which I had made an artificial anus for typhoid perforation and subsequently performed intestinal resection I drained a greatly distended gall-bladder at the second operation and cultures of the bile remained sterile; and in a case of typhoid with distended and apparently inflamed gall-bladder on which I operated the bile was reported to be sterile. Because typhoid bacilli are usually present in the bile during typhoid does not mean that most cases of typhoid have cholecystitis; cholecystitis is not very common, and arises when bacilli are very numerous or very virulent, when vital resistance is lowered, when there is antecedent inflammation of the gall-bladder, when there are gall-stones, and particularly if there is a block of the duct causing stagnation of bile. Bacilli in bile may do no harm at all, but they may cause catarrh, purulent catarrh, suppuration of the gall-bladder walls, suppuration outside of the gall-bladder, or perforation. When bile or inflammatory exudate contains typhoid bacilli, agglutinins are present and may precipitate masses which become nuclei for gall-stones.

The usual period for cholecystitis to arise is during the third week of the fever, but it is not uncommonly met with during convalescence and is perhaps mistaken for a relapse.

The condition may arise months or a year after the attack of typhoid, and yet a pure culture of typhoid bacilli may be obtained from the gall-bladder. Strange to say, cases of cholecystitis have been operated on in persons giving no history of having had typhoid, and typhoid bacilli have been obtained from the gall-bladder. Such a person may have had a very mild attack of typhoid, or he may be immune to typhoid fever and yet the bacillus may be capable of causing inflammation. Many cases of typhoid cholecystitis are probably unrecognized because of the trivial symptoms, or because a high position of the liver renders the real seat of pain obscure, because the general symptoms are uncertain, because toxemia blurs perception of pain, or because the condition is confused with appendicitis. It is rare in children, more common in adults. Most infections result from the bacilli ascending the common duct, some are by way of the lymphatics (Charles H. Mayo), some by an adhesion of the gall-bladder to the bowel, some by way of the portal circulation and the bile-ducts. Mixed infection may occur, and a secondary staphylococcus infection may be followed by disappearance of the typhoid bacilli. The symptoms of typhoid cholecystitis are pain and tenderness in the gall-bladder region, rigidity of the upper half of the right rectus muscle, perhaps a palpable mass, an elevated and remittent temperature, sweats, perhaps jaundice, and sometimes leukocytosis. In some cases perforation occurs. Erdmann reported 1 case and collected from literature 34 cases of perforation ("Annals of Surgery," June, 1903).

**Treatment.**—In an ordinary case without perforation incise and drain the gall-bladder. If perforation exists, do cholecystectomy if possible; if not, drain. No attempt should be made to suture the perforation. If perforation

exists and operation is not done, death is practically certain. Of 27 cases not operated upon, all died; of 7 cases operated upon, 4 recovered (Erdmann).

**Gall-stones** are formed during life in the gall-bladder or bile-ducts by the agglutination of materials which have precipitated from bile. The nucleus of a gall-stone may be a mass of bacteria, a blood-clot, epithelium, crystals of cholesterin or carbonate of lime, or a cast of a small duct.[1] A condition of the body thought to lead to the formation of gall-stones is designated by the term *cholelithiasis* (Brockbank). But one stone may be present or great numbers may exist. Solitary stones may be nearly round or cylindrical. When several stones or many stones exist the mutual pressure often leads to the formation of facets (Naunyn). In color, calculi may be pale yellow, green, black, or brown. Some are heavier than bile and some are lighter. Brockbank gives the following varieties of gall-stones: pure cholesterin stones, stratified cholesterin stones, common or gall-bladder calculi, mixed bilirubin-calcium calculi, pure bilirubin-calcium calculi, and certain rare forms.[2] Gall-stones usually take origin in the gall-bladder, but may arise in the common duct, the cystic duct, the hepatic duct, or the smaller ducts of the liver. As a rule, however, calculi in the common or cystic duct were not formed there, but were transported from the gall-bladder or hepatic ducts.

**Causes.**—Gall-stones are very commonly found postmortem. In Germany it is estimated that they are found in 12 per cent. of all cases. In 1655 autopsies in the Johns Hopkins Hospital gall-stones were present in 6.94 per cent. of all cases.[3] The usual estimate is 5 per cent. of autopsies. The cause is a catarrhal condition of the bile-ducts, due particularly to the entrance of bacteria from the intestine (colon bacilli, typhoid bacilli, pus organisms, pneumococci). This catarrhal condition causes stagnation of bile. Healthy bile is sterile, but not germicidal, and bacteria will grow in it. Bacteria have been found in bile years after the termination of an attack of typhoid fever. Experimental infection of the gall-bladder producing mild cholecystitis is almost always followed by gall-stone formation.[4] Welch pointed out that recent gall-stones have bacteria in their center. Cushing tells us that 30 per cent. of gall-stone cases operated upon in the Johns Hopkins Hospital had previously suffered from typhoid fever, but the experience of the Mayos is not in accord with this opinion. In view of the fact that bile containing typhoid bacilli may contain agglutinins we can understand how masses could be precipitated to form nuclei—30 per cent. of Ochsner's cases had had appendicitis.

The chief predisposing causes are advancing years, insufficient exercise, the daily consumption of unnecessarily large quantities of food, gouty tendencies, and conditions which interfere with the emptying of the gall-bladder. Cardiac disease and cancer of the liver predispose. Gall-stones rarely form before the age of thirty-five. The youngest patient from whom I have removed stones was a girl of twenty. The disease is more common in the insane than in the mentally sound, in the white race than in the black, and in women than in men. In 25 per cent. of all females beyond sixty years of age gall-stones are present (Naunyn). The special liability of women may be brought about by tight lacing, pregnancy, inactivity, or movable right kidney. Stout and lazy women are particularly liable to gall-stone formation, and women who have borne children are far more liable than those who have not. Total abstainers seem to possess a greater predisposition than users of alcohol, probably because they are more apt to be large eaters (Herbert F. Waterhouse, in "Lancet," May 8, 1909). There

[1] Bevan, in "Chicago Med. Recorder," April, 1898.
[2] Brockbank's treatise on "Gall-stones."
[3] C. D. Mosher, in "Johns Hopkins Hosp. Bull.," Aug., 1901.
[4] Gilbert, in "Archives générales de méd.," Aug. and Sept., 1898.

are two forms of the condition to be considered: the acute type, due to efforts made by the gall-bladder or duct to expel the concretion; and the chronic condition, in which a calculus is lodged for a long time, or in which, as soon as one calculus is passed into the intestine, "another begins its journey" (Brockbank's treatise on "Gall-stones"). The fact that bacteria cause the condition must not lead us to infer that pus is of necessity formed. If the bacteria are present in small numbers, or if their virulence is greatly mitigated, they produce only catarrhal inflammation, the bile stagnates, and a stone forms. There may be one stone, two, several, or many stones. I have removed 200 from a patient. Multiple stones are usually facetted. Solitary stones are not facetted.

Many observers believe that inflammation of the mucous membrane causes the secretion of quantities of cholesterin, which material forms a large part of most gall-stones. Others maintain that cholesterin is a normal constituent of bile and is not obtained from the mucous membrane.

Bachmeister ("Münch. Med. Woch.," Feb. 18, 1908) demonstrates that if pure sterile bile is permitted to stand for a considerable time cholesterin will be precipitated, and that if epithelial cells are added to this the cholesterin is precipitated much more rapidly. The catarrhal inflammation furnishes quantities of epithelial cells and the cells precipitate cholesterin, and in this way inflammation causes gall-stones. It is probable that when gall-stones exist they are all due to a common cause and all began to form at the same time. It is not likely that one begins and then another, and so on. After a stone once begins it may progressively increase in size. In many cases the stone or stones never cause trouble. A gall-stone may begin to descend because of violent muscular exertion, external pressure, or at the onset of a fresh inflammation which leads to loosening of the stone. A very small stone usually passes freely. A larger stone in passing causes colic. A still larger stone remains in the gall-bladder, or becomes fixed in the cystic duct or in the common duct. In most cases gall-stones form in the gall-bladder. In some they form in the common duct if stones have previously existed in the gall-bladder. When the common duct retains a stone and is suffering from some degree of obstruction and from infection, stones not very unusually form in the hepatic ducts (Wm. J. and Chas. H. Mayo, in "Am. Jour. Med. Sciences," March, 1905). Stones are occasionally found at necropsy in the radicles of the hepatic duct.

**Symptoms.**—The formation of a stone requires several months, and during the antecedent period of gastro-intestinal catarrh, *the prodromal state* of Kraus, certain symptoms may exist, viz.: constipation, flatulence, loss of appetitie, migraine, uneasy sensations in the epigastrium or right hypochondrium, sallowness of the skin, slight yellowness of the conjunctivæ, scantiness of urine, which excretion is saturated with uric acid, and may after a time contain a little bile. If this condition is not arrested by treatment, it grows worse. The abdomen becomes decidedly distended; pressure over the stomach or liver may cause distinct uneasiness or even pain; acid indigestion is very troublesome; violent attacks of migraine occur; constipation becomes more decided, the feces become clay colored, gastralgia may occur, the skin is apt to be slightly jaundiced, itching is complained of, the patient is irritable and sleeps poorly. The liver is found to be enlarged and the urine contains distinct amounts of bile. When the patient reaches this stage, gall-stones have formed or are very liable to form. These symptoms may pass away even if a concretion forms. It is quite true that in some cases a stone exists for years without causing trouble. This is particularly true in elderly people. A stone seldom fails to cause symptoms, but often the symptoms are unrecognized. In many cases the symptoms which stones cause are thought to be due to disease of the stomach (indigestion, flatulence, pain after eating, pyloric spasm,

etc.). Most of the cases I have seen long thought they had stomach trouble, and the real condition was recognized only when there was a seizure of colic or an attack of inflammation.

As Waterhouse ("Lancet," May 8, 1909) says, the symptoms do not bear any relation to the size or the number of the stones. In fact, gall-stones give rise to active symptoms only when infection occurs or when the ducts become occluded and cease to drain, or when a stone starts to pass. If infection occurs, it may pass away spontaneously, but seldom does so. When a stone forms, pain is apt to become a marked feature of the case. John B. Murphy ("Med. News," Nov. 2, 1903) points out that in a person with stones in the gall-bladder there may be:

1. The pain of acute inflammation, the result of a severe infection. In this condition there are abdominal rigidity and contracted gall-bladder.

2. The pain of tension. In this there is not persistent abdominal rigidity, but pressure always causes sudden and transient tension of the belly muscles. Murphy's method of demonstrating tenderness of the gall-gladder is most valuable, and I always use it. It is as follows: Hook the fingers well up under the liver and tell the patient to take a deep inspiration. On inspiration pain becomes acute and respiration suddenly ceases.

3. Referred pain, which may exist with either of the above conditions. *Colic* is spasmodic pain, and means that a stone has left or is trying to leave the gall-bladder, and is in or is trying to enter a duct. Many persons with a stone or with stones in the gall-bladder never have colic. A sense of pressure or of soreness in the hepatic region, the result of cholecystitis, has added to it sudden and transient paroxysms of pain, due to the passage of thick bile from the gall-bladder and small ducts, or of gravel from the small ducts, urged on by bile pressure. When any stone but the very smallest begins to pass from the gall-bladder, violent colic is experienced. Such a colic usually comes on very suddenly, and often about three hours after a meal. It may, however, come on gradually, the patient complaining greatly of flatulence. In some cases it is so sudden and violent as to simulate perforation of the stomach or duodenum. The reason colic is particularly apt to come several hours after a heavy meal is that at that time bile is passing down into the intestine. A bladder containing calculi often tolerates the presence of the foreign bodies for an indefinite length of time, and then suddenly resents their presence and ejects them forcibly or tries to eject them. The pains are violent, spasmodic, and paroxysmal, and over the hepatic and epigastric regons, "radiating upward over the right half of the thorax" (Kraus), and passing particularly from the epigastrium to the right shoulder-blade. The patient is profoundly nauseated and usually vomits. In many cases the vomiting is violent. The abdomen is distended and a condition almost of collapse is soon reached. The temperature is usually normal or subnormal, but is occasionally somewhat elevated. The patient may shiver and sweating may follow, but rigors are rare. The respirations are shallow, the patient groans, cries out, flings himself about in the bed, and often, in seeking relief, assumes some strange or contorted position. He frequently holds one hand over the liver region. His expression is indicative of intense suffering and apprehension and sometimes of abject terror. The pain is one of the most awful a human being can feel, and women who have felt it assert that the pains of parturition are trivial in comparison. The attack lasts a variable time, and terminates when the stone passes into the intestine or drops back into the bladder. The usual duration of an attack is from four to twenty hours. I have seen attacks that lasted three days, four days, or even five days, almost without intermission. It terminates suddenly if the stone passes or falls back in the gall-bladder. It abates very gradually if the stone becomes wedged in a duct.

In many cases at the termination of the attack an enormous amount of clear pale urine is passed. During and for a time after the attack the gall-bladder may be very tender. After the cessation of colic, if the feces are examined carefully during several days, the stone may be discovered. The fact that no stone is discovered does not prove that one was not passed, because a cholesterin stone may be destroyed in the intestinal canal. If the stone is passed, jaundice almost invariably follows the colic in from twenty to thirty-six hours and lasts several days. The jaundice results from the stone being in or having passed through the common duct. If stones do not pass from the cystic duct so as to enter or protrude into the common duct, jaundice does not occur. In 80 per cent. of my cases of gall-stones (excluding common duct cases) there was no history of jaundice. Even when a stone is lodged in the common duct jaundice may be slight or absent. When jaundice arises after a colic, it comes on gradually, bile appears in the urine, and often, but not always, the stools become clay colored from absence of bile. Jaundice may be first noticeable in the urine or in the conjunctiva. The skin is apt to itch annoyingly, even atrociously. The patient is constipated and very thirsty. The liver is enlarged and tender and the spleen is enlarged. Some writers state that the pulse is slow in jaundice. My experience is in agreement with the much larger experience of Moynihan, who says: "I have not found any reduction in the pulse-rate in jaundice unless a degree of chronic pancreatitis is present" ("Gall-stones and Their Surgical Treatment"). If the stone becomes impacted, after a time the pains become gradually less violent, and may entirely cease. If it ceases and the stone does not move, pains do not recur; if the stone moves pains recur, and, usually, again and again the patient suffers from severe pain. An individual may get about when a stone is impacted, but again and again fierce attacks of colic occur, and if the stone is wedged immovably in the common duct, producing absolute obstruction, the patient becomes and remains deeply jaundiced. Continued deep jaundice is seldom seen when stones are lodged in the common duct, because they are not often absolutely fixed and hence rarely produce complete obstruction. Usually the stone moves from time to time or is at least lifted, so that bile gets by it at intervals. This condition constitutes the *ball-valve stone*, and in it jaundice, though present more or less, is at times much more intense than at other times. It is a jaundice in which the hue is yellow, not deep brown, and it is a jaundice that wanes and deepens. It deepens after each colic and later wanes, but seldom entirely disappears while the stone remains in the duct.

In persistent jaundice due to gall-stones the gall-bladder is seldom enlarged. Courvoisier showed that when persistent jaundice is associated with enlargement of the gall-bladder the cause is usually pressure on the duct from without (malignant disease of the pancreas).

Slight jaundice is not always easy of recognition. Recognition is particularly difficult in sallow individuals and by artificial light. Moynihan praises Hamel's test for slight jaundice. It is made by drawing a little blood from a puncture of the lobe of the ear into a capillary tube and permitting the tube to stand for a few hours. If any jaundice is present the serum, which collects in the upper part of the tube, will be yellow (Moynihan).

In certain cases when a stone is in the common duct an attack of colic is followed by or accompanied by a chill or chills, which may be very violent, moderate, or slight, and by a febrile seizure resembling malaria and called *hepatic fever* or *Charcot's fever*. The temperature rises rapidly, and in an hour becomes 104° F. or more, remains high for several hours, and then drops suddenly to normal. It may remain normal for a few hours, a day, two days, several days, or weeks. In this condition there are jaundice and tenderness of

the liver. Charcot's fever is brief in duration. It usually means stone in the common duct. If stones are in the bladder, we are more apt to get a persistent slightly elevated temperature. These intermissions distinguish Charcot's fever from the remittent fever of sepsis, and the absence of the plasmodium in the blood and the history of colic distinguish it from malaria. The fever is due to intoxication with toxins from infected bile retained in the ducts by obstruction. The condition is ominous because it is due to infection, and means inflammation of the large ducts (*cholangitis*).

The chart of Charcot's fever shows sudden elevations, precipitate descents, and complete intermissions. Moynihan calls it the "steeple chart" ("Gall-stones and Their Surgical Treatment"). When infection spreads widely in the smaller intrahepatic ducts the temperature is high and does not remit. Continuous fever of this type has usually been preceded by Charcot's fever.

If a stone lodges in the cystic duct, it does not cause jaundice unless an end of the stone projects into the common duct. It grows in size from incrustation, prevents the entrance of bile into the gall-bladder, and the bladder may shrivel and thicken or become distended and filled with mucus, the bile being absorbed (*hydrops* of the gall-bladder). If a bladder so blocked becomes infected, pus forms, and the condition known as *empyema* of the gall-bladder arises. An empyema of the gall-bladder may rupture into the bowel, the peritoneal cavity, or even through the skin.

The common duct is involved in 1 out of 5 or 6 cases of gall-stone disease.[1] Brewer points out that in 67 per cent. of common duct cases the stone is in the duodenal extremity, in 15 per cent. in the hepatic extremity, and in 18 per cent. in the middle. If a stone *blocks* the common duct, jaundice always exists and persists. Blocking may be complete and the stone may ulcerate into the bowel or the peritoneal cavity. Blocking may be incomplete, the stone acting as a ball-valve and producing *intermittent* colic and jaundice, which wanes and deepens (see page 1051). Fenger pointed out that if a stone remains fixed in the common duct the liver becomes tender and enlarged, but if a stone floats about in the common duct the gall-bladder undergoes atrophy. In complete obstruction the stools become clay colored and bilirubin is found in the urine. Fluctuating jaundice, with attacks of pain and fever, and a shrunken gall-bladder are strongly suggestive of a "ball-valve" stone in the common duct. Persistent deepening, painless jaundice, the color of the skin becoming brown or even of a mahogany hue, associated with a distended gall-bladder, is strongly suggestive of malignant disease compressing the common duct. The above statements constitute *Courvoisier's law*. It is found true in 90 per cent. of cases. We may add that a persistent jaundice of yellow hue, varying somewhat, and associated with pain or with actual colic, suggests blocking of the duct by an immovable stone.

Gall-stones may lead to suppurative inflammation of the gall-bladder or bile-passages, ulceration, occlusion of the neck of the gall-bladder, dilatation of the stomach from the formation of adhesions which kink the pylorus, pericystic abscess, peritonitis, empyema of the gall-bladder, and cancer of the gall-bladder. In cancer of the ducts gall-stones are seldom found, at least are seldom found in the ducts. Eddes collected 22 cases of cancer of the papilla. In 3 of these cases there were stones in the gall-bladder, in 1 there was a stone in the common duct ("Boston Med. and Surg. Jour.," March 7, 1901). If the patient develops distinct infection of the gall-bladder or bile-ducts, he will suffer from chills, fever, and sweats.

Gall-stones may lead to cirrhosis of the liver. A stone may ulcerate into the bowel and cause intestinal obstruction. It may be difficult to make a diagnosis between gall-stones with icterus and cirrhosis of the liver with

[1] Robson, in "Lancet," April 12, 1902.

icterus. In the former case the urine contains bilirubin and in the latter case urobilin.

**Treatment.**—In the prodromal stage and after recovery from an attack insist on the patient taking considerable outdoor exercise. Direct him to take a cold sponge-bath every morning, to move the bowels freely every day, and to employ a simple diet. He should avoid all highly seasoned foods, pastry, rich soups, fatty food, cheese, alcohol, and sweets. Alkalis internally are of value.

During colic give a purgative enema, apply hot turpentine stupes over the hepatic region, and administer hypodermatic injections of morphin and atropin. If vomiting does not occur, let the patient drink a large amount of warm water to favor it. After the attack administer a saline purgative.

When the attack has terminated, examine carefully for any evidence of inflammatory trouble in the hepatic region.

In certain cases operation becomes necessary. Mr. A. W. Mayo Robson[1] advises operation in the following cases: in frequently recurring biliary colic without jaundice, whether the gall-bladder is enlarged or not; in cases of enlargement of the gall-bladder without jaundice, even if there is no pain; in persistent jaundice which was ushered in by pain, painful seizures occurring, whether or not febrile attacks occur; in empyema of the gall-bladder; in peritonitis beginning in the gall-bladder region; in intrahepatic abscess and in abscess about the liver, gall-bladder, or bile-ducts; in some patients in whom the stones have been passed, but adhesions remain and produce pain; in fistula cases; in some cases of persistent jaundice due to obstruction of the common duct, although there may be a possibility of cancer existing; in phlegmonous cholecystitis and gangrene of the gall-bladder. Besides these conditions, which may be produced by gall-stones, Robson operates for wounds of the gall-bladder, infective and suppurative cholangitis, and for some conditions of chronic catarrh of the bile-ducts and gall-bladder.[2] The tendency to operate early for gall-stones is growing. It is true that stones *may* cause no trouble, but sooner or later they are apt to cause it, there is no tendency whatever to spontaneous cure, and medicine cannot dissolve them in the bladder. Early operations are easy and comparatively safe; late operations are difficult and dangerous, and by early operation dangerous complications (infection, adhesions, obstructive jaundice) are avoided. As Maurice H. Richardson[3] says: An early operation is less dangerous than the passage of a stone; complications are avoided or lessened; even if the diagnosis is wrong, the real condition may be found and removed. If obstructive jaundice exists operation is dangerous because of the possibility of fatal oozing of blood.

The common operation is *cholecystostomy*, which consists in opening the gall-bladder, removing the stones, and making a temporary fistula in the gall-bladder. The drainage cures the diseased mucous membrane. The fistula is permitted to heal after a time, hence many call the operation cholecystotomy rather than cholecystostomy. Operation should be done promptly and should not be delayed. Delay permits the gall-bladder to thicken and shrink, and allows the stone to enter the duct. After drainage gall-stones rarely re-form. Wm. J. Mayo collected 1000 operations done by six surgeons, and in not 1 case did stones re-form. Kocher has seen stones recur in 3 out of 31 cases of cholelithiasis after ideal cholecystotomy (suturing the gall-bladder after removing stones). The operation of incision, removal of the stone, and suture of the gall-bladder is known as *ideal cholecystotomy* or *cholecystendysis*. It is not a

[1] On the "Gall-bladder and Bile-ducts."

[2] Robson's treatise, from which the above is taken, is a valuable exposition of the surgery of the gall-bladder and bile-ducts.

[3] "Boston Med. and Surg. Jour.," Sept. 5, 1901.

proper procedure, as it does not cure the diseased mucous membrane and stones are apt to re-form. *Cysticotomy* is incision of the cystic duct. If calculi exist in the common duct, it may be possible, after celiotomy, to manipulate them back into the bladder and extract them from that viscus by a scoop, but this maneuver is impossible unless the cystic duct is dilated. In some cases the gall-bladder is incised, a fistula is made, and the duct and bladder are frequently irrigated. In other cases the stone may be crushed by the fingers manipulating the duct and the concretion within it (*choledocholithotrity*). Robson points out that crushing of the stone is apt to leave fragments which may cause trouble, and it should be done only when the stones are soft. It is wrong to endeavor to force a stone from the common duct into the duodenum. The attempt will fail, and in some cases the patient will be placed in a worse condition by the stone lodging in Vater's diverticulum.[1] The duct may be opened, and after the removal of the stone closed by sutures (*choledochotomy*) or drained for a time (*choledochostomy*), strands of gauze being carried down to the opening and in some cases a tube being carried up a dilated duct toward the liver. If the stone is impacted near the outlet of the duct, it may be necessary to incise the duodenum in order to remove the stone (*duodenocholedochotomy*). A dilated common bile-duct may be anastomosed to the bowel (*choledocho-enterostomy*) or to the surface (*choledochostomy*). The obstruction may be side-tracked by anastomosing the gall-bladder to the bowel (*cholecystenterostomy*) (see page 1127). Cholecystenterostomy affords drainage, but does not remove the cause of trouble, and infection is apt to be received from the bowel. In some rare cases of common duct obstruction, in which the gall-bladder is distended and the condition of the patient is desperate, anastomose the gall-bladder to the colon (Robson). In some cases of diseased gall-bladder the viscus is removed (*cholecystectomy*). Wm. J. Mayo and others have pointed out that a danger in operations on the common duct is a sudden fall in blood-pressure when the duct is being manipulated. All operators have observed it. Ransohoff maintains that it arises only when the portal vein is compressed.

**Carcinoma of the Gall-bladder.**—In 405 operations on the gall-bladder and biliary passages the Mayo brothers found malignant disease 20 times (5 per cent. of cases). (See Wm. J. Mayo, in "Med. News," Dec. 13, 1902.) Malignant disease may be primary or secondary. In primary carcinoma calculi are always present, and are apparently the cause of cancer by maintaining chronic irritation. Stones are seldom present in secondary malignant disease.

Carcinoma of the gall-bladder can usually be palpated. It is hard and nodular, and seldom accompanied by much abdominal rigidity. There will be a long history of attacks of biliary colic and of recent or comparatively recent grave loss of flesh. Sooner or later jaundice arises, deepens, and persists.

Cholecystectomy has been employed for this condition, but offers but little hope. In 2 cases in which I opened the abdomen without suspecting malignant disease of the gall-bladder the liver was hopelessly involved. In 1 case in which I operated for a supposed impacted stone in the common duct an inoperable cancer of the common duct was found.

## Injuries and Diseases of the Pancreas

**Injuries of the Pancreas.**—The pancreas is very rarely ruptured alone, although this sometimes occurs as the result of blows or crushes. In the majority of cases in which the pancreas is damaged other organs are involved; for instance, the stomach, the spleen, and the liver. A gunshot-wound of the pancreas is almost certain to injure the left kidney, the stomach, or the

[1] See A. W. Mayo Robson, in "Lancet," April, 12, 1902.

vertebral column. It will be remembered that in the case of President McKinley the bullet passed through the stomach, damaged the left kidney, and injured the pancreas. Becker reported an isolated gunshot-wound of the pancreas, the only case on record (Stephen H. Watts). Garré ("Beiträge zür Klinische Chirurgie," xlvi, No. 1) collected 30 cases of subcutaneous rupture of the pancreas, and in only 8 of these cases was the pancreas alone damaged.

**Symptoms.**—When the pancreas is injured alone, hemorrhage is not usually severe; but if adjacent organs are also damaged, it is sure to be profuse. Hence when adjacent organs are damaged immediate symptoms of severe intra-abdominal hemorrhage appear; but profound collapse is not often present when the pancreas alone is injured. In fact, symptoms may not arise for a considerable length of time after injury of the pancreas. A diagnosis at this stage is impossible without exploratory operation. Wohlgemuth and Noguchi claim that within a few hours of a pancreatic injury there is an increase of diastase in the blood and urine ("Berlin. klin. Wochen.," xlix, 1912). If this observation is correct we have a very valuable diagnostic test. Severe injury of the pancreas is usually, but not invariably, fatal. After slight damage of the gland the patient may completely recover; but, as a rule, he partly recovers, and, after a number of weeks, a smooth enlargement, palpable in the epigastric region, is formed. When operation is performed this mass is found to be back of the stomach. It contains a quantity of fluid blood, clot, and pancreatic fluid. Such a fluid collection is in the lesser peritoneal cavity and is called a cyst, though it is not a true cyst of the pancreas. It is a pseudocyst. Robson and Moynihan, in their valuable treatise on "Diseases of the Pancreas," explain the formation of this collection of fluid as follows:

The injury lacerates the posterior layer of the lesser sac of the peritoneum and the pancreas, to which it is adherent. Blood and pancreatic fluid enter the lesser peritoneal sac. Peritonitis follows. The foramen of Winslow becomes blocked by adhesions; and the lesser peritoneal cavity, being now a closed sac, is distended by a serous exudate mixed with blood and pancreatic fluid. Collections of this character form very rapidly, and several pints may gather in a few days. Other results of injury to the pancreas are abscess, pancreatitis, and true cyst formation. A fistula may follow operation for rupture of the pancreas. Such a fistula is very troublesome, often refuses obstinately to heal, and the pancreatic fluid macerates the skin severely.

**Treatment.**—Operation is imperatively demanded, although the prospects are bad. Garré collected 8 cases, 3 were operated upon, and all died. He reported a successful case of his own (Loc. cit.). The pancreas was torn in two and the pieces were separated. The splenic vessels were uninjured. The two portions of gland were sutured together. This stopped the bleeding. Gauze-packing was introduced. Mikulicz ("Proceedings of Amer. Surg. Soc.") in 1903 collected 21 wounds and 24 crushes of the pancreas. Twelve of the wounds were due to bullets and 9 were stabs. Five of the 12 gunshot-wounds were operated upon, with 2 deaths. All unoperated upon died. The 9 patients who had been stabbed were all operated upon and only 1 died. In a gunshot-wound of the abdomen, when exploration leads the surgeon to surmise that the pancreas has been injured, this organ should be approached by dividing either the gastrocolic omentum, the transverse mesocolon, or the gastrohepatic omentum. Accessory injuries must be carefully noted, and if a bullet has penetrated the posterior wall of the stomach, the pancreas is almost certain to be damaged. One should remember that, as Park says, even after opening the abdomen it is difficult to explore the pancreas, especially in a stout person. If there is no evidence of posterior perforation of the stomach by a foreign body, one may

assume that the pancreas has escaped. When the pancreas is exposed, if it is found to be bleeding, the bleeding vessels should be ligated and the tear in the gland should be sutured with catgut, care being taken not to puncture the main duct of the gland. If this duct has been cut, it should be carefully sutured. In some cases of gunshot-wound it is necessary to resect a portion of the gland. At the termination of an operation upon the pancreas posterior drainage, preferably at the costovertebral angle, should always be obtained. It is necessary to carefully drain away all escaping pancreatic fluid, as it tends to cause necrosis of tissue with which it comes in contact.

In cases of crush with pancreatic injury the associated injury to other structures usually proves rapidly fatal, but in a less severe case the abdomen may be opened for exploration, and if this is done, the surgeon should proceed as previously directed.

The question of excising a lacerated portion of the pancreas is one of great interest. It is known that dogs have lived for some time after complete excision of the pancreas. Four-fifths of the pancreas can be removed from a dog without producing permanent glycosuria, but if more than this is removed the dog develops saccharine diabetes and eventually dies of it. In man, quite large-sized pieces of the gland have been removed and recovery has followed. Hence it is justifiable to excise a hopelessly damaged portion, bearing in mind Park's caution that the chief danger in excising a portion of the pancreas is injury to the splenic artery.

**Wounds of the Pancreas During Operations on the Stomach and Spleen.**—In the performance of gastrectomy, partial or complete, the pancreas will be injured if the growth or ulcer is adherent to it. Such an accident is held by most operators to greatly increase mortality. The Mayos report 448 resections of the stomach for benign and malignant disease. The average mortality was 10 per cent. In 8 per cent. of these cases the pancreas was injured, and the average mortality of such cases was only 11 per cent. (Wm. J. Mayo, in "Annals of Surgery," August, 1913). The injuries reported by the Mayos were superficial at the point the stomach adhered. In no case was the main duct opened. It was noticed in these cases that local peritonitis had caused the formation of a fibrous capsule. Bleeding was controlled by suture-ligatures of catgut. The pancreatic wound was not sutured. As stated on page 1085, if the pancreas is wounded during pylorectomy, the closed end of the duodenum is placed in the pancreatic wound and the anterior peritoneum and adventitious sheath is sutured to the anterior portion of the duodenum. If an ulcer of the posterior wall of the stomach is adherent to the pancreas, transgastric excision removes considerable pancreatic tissue. Such a wound is not sutured, but the gap is filled by a mobilized bit of gastrohepatic or gastrocolic omentum (Wm. J. Mayo, Ibid.). In 2 of my cases of splenectomy I damaged the pancreas, tying off a bit of the tail with the splenic vessels. In 1 case leakage occurred and death followed in spite of anterior drainage. In the other case there was profuse drainage for several days, which was carried off by a posterior drain. This patient recovered. Both were cases of Banti's disease.

**Pancreatic Fistula.**—A fistula may follow a wound of the pancreas. It is a very troublesome condition, often refuses most obstinately to heal, and the pancreatic secretion causes maceration and violent irritation of the adjacent skin. The usual treatment is to keep the way open for easy drainage. Wohlgemuth's plan is promising ("Berliner klin. Wochen.," 1908, No. 8). He reported 5 successful cases. I have had 1. The treatment consists in feeding upon strict antidiabetic diet and in giving large doses of bicarbonate of soda before and after meals. By cutting off carbohydrates a powerful stimulus to the flow of pancreatic juice is removed. The bicarbonate of sodium lessens the acidity of the stomach contents. The more acid the contents which enter

the duodenum, the greater the flow of pancreatic juice; the less acid, the less the flow.

**Displacement of the Pancreas.**—In cases of splanchnoptosis the pancreas may become considerably displaced, though this condition cannot be recognized without opening the abdomen. It may be a portion of the pedicle of a movable spleen. So far, I know of no case in which fixation has been attempted, though, of course, theoretically it could be done. The pancreas has been found in umbilical herniæ. In 10 per cent. of diaphragmatic herniæ the pancreas constitutes part of the contents. Körte collected 8 cases in which the pancreas prolapsed through an abdominal wound. In several cases in which the pancreas prolapsed into an abdominal wound the protruding part has been excised. In other cases it has been restored.

**Pancreatitis** often leads to the production of jaundice; always to very rapid loss of weight; occasionally to the presence of fat and sugar in the urine; sometimes to the presence of fat in the stools, and frequently to the condition known as fat necrosis. Robson and Moynihan[1] point out that when there is no diarrhea and the stools contain undigested muscle-fiber, one may assume that there is a deficiency in pancreatic juice. When there is a blockage to the secretion from the pancreas, if salol is given by mouth, salicyluric acid does not appear in the urine. The test is made by putting 15 gr. of salol into gelatin capsules hardened with formalin (Sahli) and giving them with a roll and a cup of water. If pancreatic ferment is in the intestine, salicyluric acid appears in the urine in from three-quarters of an hour to one hour; if the ferment is absent from the intestine, salicyluric acid is not found in the urine because the salol is not split up and absorbed. The test for the acid is ferric chlorid, which, in the presence of the acid, turns the urine violet. The general cause of pancreatitis is infection. Often obstruction of the common duodenal outlet of the pancreatic duct and common bile-duct is followed by infection and suppuration of the pancreatic ducts and pancreatitis. Besides the general cause, which is infection, various exciting causes may be named, among which are gall-stones in the common duct and calculi in the pancreatic ducts, traumatism, cancer of the stomach or duodenum, catarrh of the stomach or duodenum, and many infectious diseases. It thus becomes evident that the infection may be by way of the blood; but, undoubtedly, in the vast majority of cases, the infection comes by way of the duct. One manner in which the disease may be produced was suggested by Halsted and Opie, of Baltimore: A stone becomes impacted in the duodenal outlet of the common duct and pancreatic duct, the pancreatic duct, where it emerges above the common duct, not being blocked. The bile and pancreatic juice are thus prevented from entering the duodenum, and the bile flows back into the pancreatic ducts. Swelling of the papillæ could act in the same way. So could a plug of mucus. It is thought that overacid gastric juice may enter the duct and produce pancreatitis. Pancreatitis is predisposed to by obesity and arteriosclerosis (Balch and Smith, "Publications of the Mass. Gen. Hosp.," Oct., 1911). Deaver is of the opinion that some infections reach the pancreas through the lymphatics ("Annals of Surgery," August, 1913).

That strange condition known as *fat-necrosis* is often present in pancreatitis. In fat-necrosis the fat is decomposed into fatty acids and glycerin. The glycerin is absorbed, but the fatty acids unite with calcium salts and remain in the tissues, forming patches of yellowish-white color and varying size. These patches are found in the fat beneath the peritoneum, in the omentum, and in the mesentery, and even in distant parts (for instance, the pericardium).[2] It is an undoubted fact that fat-necrosis is not uncommonly found after diseases and injuries of the pancreas; and many assume that it is produced by the entrance of the ferment of the pancreas into the

[1] Robson and Moynihan, on "Diseases of the Pancreas." [2] Ibid.

fatty tissue. How the ferment gets there is a matter of some doubt. In the case of a wound of the pancreas one can understand the flow of the secretion and its imbibition by adjacent parts; but in other cases one must assume that it has been absorbed by the lymphatics and distributed to more distant parts. When one reflects that in some conditions of the pancreas there is no fat-necrosis, while in others this condition arises, it is presumable that the pancreatic conditions associated with it are such as to permit the fat-splitting ferment to diffuse into neighboring tissues.

In pancreatic disease *hemorrhage* into that organ is common. The hemorrhage is not, of necessity, fatal, but frequently is so. Occasionally death takes place as the result of sudden pancreatic hemorrhage in a person apparently in excellent health. It is thought by Robson and Moynihan that during the existence of cancer of the pancreas there is a strong tendency to excessive hemorrhage after any operation. In 1 case of my own the patient bled to death after the performance of cholecystostomy for obstructive jaundice. The oozing of blood in this case was from the margins of the gall-bladder and the adjacent peritoneal surfaces. We, therefore, conclude that in certain conditions of the pancreas there is a tendency to local hemorrhage in that organ; and that there may also be a tendency to the development of a general hemorrhagic diathesis, the general hemorrhagic tendency being much increased if jaundice exists. During acute inflammation of the pancreas hemorrhage is almost certain to occur into that gland; in other varieties of inflammation hemorrhage may occur or may be absent. In degenerative lesions of the pancreas a material like unfermented pentose is frequently present in the urine. When the reaction for this material is obtained we speak of it as the *Cammidge reaction*, after its discoverer. (For Cammidge's improved method, see "Brit. Med. Jour.," May 19, 1906.) The Cammidge reaction is not by any means conclusive proof of organic pancreatic disease. It may be found in a great variety of other conditions (gall-stones, cholecystitis, gastric carcinoma, burns, etc.). In most patients who exhibit it there is arteriosclerosis and, of course, this condition might effect pancreatic secretion (Watson, "Brit. Med. Jour.," April 11, 1908). My own views coincide with those of Swan and Gilbride ("New York Med. Jour.," April 23, 1910). They believe a positive reaction indicates disturbed pancreatic function, but not, of necessity, organic disease.

**Forms of Pancreatitis.**—This disease is divided by Robson and Moynihan into the acute, the subacute, and the chronic forms; and they say that recorded cases demonstrate the fact that three distinct classes of inflammation may arise: (1) Cases that die within forty-eight hours of the beginning of the trouble. In this group hemorrhage is usually found, and if fat-necrosis is present, it is limited in area. (2) Those that live for some weeks after the beginning of the trouble. In these cases the pancreas may become necrotic or suppuration may occur. Fat-necrosis is usually widespread. (3) In the third class of cases long-continued inflammation or repeated attacks produce sclerosis of the pancreas.

**Acute Pancreatitis.**—In this condition the pancreatic secretion is infected and is blocked up in the ducts. It digests or ruptures the walls of the small ducts and diffuses through the gland, producing necrosis of gland tissue, exudation into gland tissue, necrosis of the blood-vessels, and, in consequence, hemorrhage (Balch and Smith, in "Publication of Mass. Gen. Hosp.," Oct., 1911). It is known that normal pancreatic juice will not digest living pancreas. It seems probable that trypsin is activated by bacteria and material from the duodenum (Polya, in "Pflüger's Archiv," cxxi, Heft 9 and 10). A part of the gland or the entire gland may be involved. Fat-necrosis occurs. The entire pancreas may become gangrenous.

The *symptoms* of this condition come on suddenly and consist of violent pain in the epigastric region, but seldom marked tenderness, usually vomiting, constipation, weakness of the circulation, slow or moderately rapid pulse, cold extremities, and collapse, with a great fall in blood-pressure. The temperature is normal or moderately elevated. Some maintain that collapse is due to trypsin; others, that it results from the absorption of toxic products from the gland. The pain is extremely violent and is intensified in paroxysms, and there is rigidity of the epigastrium. In some cases there is appreciable tenderness. The patient vomits the contents of the stomach and then bilious matter. Distention soon becomes distinct in the upper portion of the abdomen. The patient presents the appearance of one suffering from peritonitis. This condition is not unusually mistaken for intestinal obstruction, but in acute pancreatitis the constipation is not absolute; the patient passes gas, and may even have a bowel movement as the result of the administration of an enema. The condition is usually fatal within a few days, but in very rare instances recovery takes place. In acute pancreatitis from stone in the common duct there is no leukocytosis (Murphy). In some cases of pancreatitis from other causes there is high leukocytosis.

The *diagnosis* cannot be made with certainty and is merely an inference. Reginald Fitz told us that the existence of this disease should be suspected when a person previously in good health, or who has complained only of occasional attacks of digestive disorder, is suddenly seized with severe pain in the epigastric region, followed by vomiting and collapse; and when, within twenty-four hours or more, there appears a circumscribed swelling in the epigastrium which is resistant or tympanitic. Visible oil in the stools, a Cammidge reaction, or sugar in the urine add probability to a diagnosis of pancreatitis. When an exploratory incision is made in the abdomen, if fat-necrosis is detected, the diagnosis becomes certain. The peritoneal cavity may contain thin, bloody fluid.

*Treatment.*—Operation was suggested by Naunyn in 1903. The exploratory operation is carried out in front, and the earlier it is made the better. Robson operates at once, even in shock. It is quite true that the patient might, if let alone, pass through the acute stage, and that a local abscess might then form, the treatment of which would be obvious. But the danger of waiting is too great to justify delay, and if suppuration should occur it might not remain local, but might spread widely in the retroperitoneal tissues. When observation after exploratory incision into the greater cavity of the peritoneum suggests the existence of acute pancreatitis, the infected area should be exposed, preferably above the stomach, through the gastrohepatic ligament. The pancreas should be incised, hemorrhage should be arrested by ligation or packing, the gauze pack emerges above the lesser curvature, an incision should be made at the costovertebral angle, and posterior drainage should be made from the lesser peritoneal cavity. One should follow the rule laid down by Rosswell Park, and explore in every case in which the disease is suspected to exist. Of Körte's 16 cases operated on during the first week 11 recovered ("Annals of Surgery," 1911, vol. lv). Of Balch and Smith's cases ("Publication of Mass. Gen. Hosp.," Oct., 1911), 11 were operated upon within three days, and 3 recovered. The two authors just quoted state that the Massachusetts General Hospital records for twenty-one years show only 1 victim who recovered without operation.

**Subacute pancreatitis** comes on suddenly, with violent pain, vomiting, and constipation, but there is far less exhaustion and weakness than in the acute form. The vomiting is less marked and the swelling in the epigastric region is not so rapid. The symptoms are similar to those of the acute form, but not so violent nor so rapidly progressive. The temperature frequently rises higher than in the acute form, and it may become irregular or chills may occur.

In many cases the patient seems to grow better after a time, the violent pain abating, though distinct pain may remain; but he does not gather strength and continues to lose flesh, and there is usually albumin and there may be sugar in the urine. In rare instances fat is found in the urine. In subacute pancreatitis abscess is prone to form. The abscess may make a distinct swelling in front, and may lead to the development of a subphrenic or of a perirenal abscess. In rare cases an abscess of the pancreas tracks its way for a long distance in the subperitoneal tissue; occasionally it opens into the stomach or bowel. Cases of subacute pancreatitis usually die, but occasionally recover after a long illness.

*Treatment.*—Exploratory incision. Expose the pancreas, preferably by dividing the gastrohepatic ligament; determine its condition; remove purulent matter and necrotic areas; arrest hemorrhage with packing, and insert posterior drainage at the costovertebral angle. Leave the anterior wound open for the emergence of the gauze packing.[1]

Wm. J. Mayo[2] reports a successful operation for subacute pancreatitis. The patient was a man of fifty-two years, who, seven days before Mayo saw him, had developed violent pain in the epigastrium, collapse, distention, and other signs of intestinal obstruction; but some slight movements had taken place from the bowels as the result of medication. On admission, the abdomen was tympanitic. An ill-defined mass the size of a fist could be palpated to the right of and above the umbilicus. The pulse was 120 and very weak; the temperature between 101° and 102° F.; and there were slight jaundice, restlessness, and hiccup. A diagnosis of gangrenous cholecystitis was made. The abdomen was opened, and the omentum was found to be studded with thick, adherent, infiltrated round spots, the size of a pea or larger. There were some similar spots in the mesentery, and the peritoneal cavity contained bloody fluid. On palpation the pancreas felt like a pudding in a tight sac, and on aspiration a little blood was obtained. The gall-bladder was opened, a stone was removed, and some pus was evacuated. Drainage was inserted into the gall-bladder, and eighteen days later there was an enormous flow of bloody fluid, containing bile and pancreatic juice, from the drainage-tube. The patient recovered. This plan of treatment—free drainage of the pancreas by the performance of cholecystostomy—is to be taken into consideration.

**Chronic Pancreatitis.**—There are many causes of chronic pancreatitis, viz., syphilis, alcohol, bacteremia, block of the common duct (stenosis, stone, etc.), extension of inflammation from the bile-ducts, and ascending infection from the duodenum. It usually results from disease of the bile-passages and is often associated with gall-stones. In 2200 operations performed by the Mayo brothers on the gall-bladder and bile-ducts, the pancreas was found diseased 141 times (6.4 per cent.).

In 168 cases of pancreatic disease on which they operated 81 per cent. were caused by or, at least, associated with gall-stones. In operations upon the common or hepatic ducts the pancreas was diseased in 18.6 per cent. of cases. It was diseased in 4.45 per cent. of cases of operation upon the gall-bladder. Chronic pancreatitis produces enlargement of the organ, and the enlarged area is hard and feels like a malignant growth. This condition is more common than the acute or subacute form. Robson and Moynihan have operated upon 30 cases. This disease is frequently associated with gall-stones or with stones in the pancreatic duct, and occasionally with ulcer of the stomach or of the duodenum. In some cases symptoms of the condition come on acutely. Pain, nausea, and vomiting occur, and jaundice develops rapidly, as it does after the

[1] Roswell Park, "Annals of Surgery," December, 15, 1901.
[2] "Jour. Am. Med. Assoc.," Jan. 11, 1902.

passage of a gall-stone. It is noted, however, that the pain is not in the region of the gall-bladder, but is in the middle of the epigastrium, and it passes to the left rather than to the right. The tenderness, too, is in the middle of the epigastrium and not in the gall-bladder region. There is either constipation or diarrhea. A series of these attacks may occur, the jaundice growing worse after each attack. In some cases, however, the condition comes on gradually and insidiously, the pain slowly developing, but no violent seizures taking place. There are rigidity of the rectus muscles, rapid loss of flesh, anemia, sometimes bronzed skin, usually vomiting, and considerable flatulence. The gall-bladder is enlarged and commonly palpable.

In some cases it is possible to palpate the inflammatory mass. There may be irregular fever and chills with episodes of subnormal temperature. None of the above indications are conclusive signs of disturbed pancreatic function. Signs of disturbed function of the gland are of great importance in making the diagnosis, and these signs are glycosuria and impaired power of digesting fats and proteins (Walko, in "Arch. f. Veranungskrankheiten," 1907, xiii). Fatty stools containing unsaponified neutral fat are very significant.

The jaundice in chronic pancreatitis results from compression of the duct by the hyperplastic mass, or blocking of the duct by a stone. Stenosis of the duodenum may occur. In jaundice from chronic pancreatitis capillary hemorrhage is particularly common (Robson). Mayo Robson attaches much importance to the Cammidge reaction. In the Jefferson Hospital we regard it as often of decided use, but we do not as yet attach as much importance to it as do some other clinicians. This reaction, when present, indicates degenerative disease of the pancreas. It is obtained when the urine contains a substance having the characteristics of unfermented pentose.

*Treatment.*—Exploratory incision, opening and draining the gall-bladder; or the performing of cholecystenterostomy.

**Pancreatic Calculi.**—When the pancreatic secretion is blocked, stones tend to form; and the blocking may be due to inflammation of the duct of Wirsung, or may result from chronic pancreatitis. The stones may be single or multiple.

**Symptoms.**—There is pain in the epigastric region, which usually comes on in paroxysms that resemble those due to gall-stones, though they are not so violent. Pain is accompanied by vomiting, exhaustion, and sometimes actual collapse, and may be followed by rigors. Portions of stone are sometimes recovered from the feces, and sugar is occasionally found in the urine. Fat has also been noted in the stools in some cases. Sometimes jaundice develops because the calculus presses upon the common duct. Pancreatic calculi are composed of lime salts and can be skiagraphed.

**Treatment.**—Pancreatic calculi have, in rare instances, been removed by operation; and this is the proper procedure when the diagnosis can be made. The diagnosis is, however, possible only after exploratory incision. As a rule, no operation is performed until a cyst results or an abscess forms; and when the cyst or abscess is opened fragments of stone may be found in the fluid, and stones may subsequently come away in the resulting fistula. My colleague, Prof. Nassau, removed successfully a pancreatic calculus.

**Pancreatic Cysts.**—Many forms of cyst may develop in the pancreas; the following are set forth by Robson and Moynihan: (1) Retention cysts; (2) proliferation cysts, including cystic adenoma and cystic epithelioma; (3) hydatid cysts; (4) congenital cysts; (5) hemorrhagic cysts; (6) pseudocysts. What we speak of as pseudocysts have already been considered in discussing effusions into the lesser peritoneal cavity. They result from lacerations of the pancreas (see page 1055). Retention cysts are due to blocking of the

pancreatic duct. Congenital cystic disease is extremely rare. Hemorrhagic cysts result from hemorrhage into the substance of the pancreas itself.

**Symptoms.**—Cysts are somewhat more common in men than in women. A cyst of the pancreas proper is more often met with in the head of the organ than in its body or tail. The cyst may be single or multiple. In its growth it either destroys the substance of the pancreas or it grows away from the pancreas and damages it but little. In some cases the cysts grow to a very large size; and Robson and Moynihan refer to a case in which the cyst attained the size of a man's head, and to another in which it was the size of a full-term pregnancy. A pancreatic cyst is smooth, round, elastic, and rather tense (Robson and Moynihan). The contained fluid varies greatly. As a rule, it is brownish-red in color; in 1 case upon which I operated it was clear yellow; in some cases it is milky, and in others it is nearly black. The fluid is always albuminous. Urea may be present, and in many cases pancreatic ferments are found. In most cases the cyst adheres so closely to the surrounding structures as to render extirpation practically impossible. A pancreatic cyst of considerable size causes epigastric discomfort, pain during digestion, and frequently vomiting. In some cases the pain is trivial, in others it is very violent. As a general rule, the patient is constipated, but sometimes diarrhea occurs, and the movements may even contain blood. If the tumor presses upon the common bile-duct, jaundice will develop. The patient loses flesh markedly and with considerable rapidity, and he becomes very weak. In rare instances fat is present in the stools, and in other unusual cases sugar is found in the urine. A test should always be made with salol, to see whether pancreatic ferment is present in the intestine (see page 1057). In the beginning the pancreatic cyst is behind the stomach; but it enlarges and, as a rule, pushes the stomach upward and to the right side, and the transverse colon downward. The cyst approaches the surface of the abdomen below the greater curvature of the stomach (Robson and Moynihan). The same authors tell us that in rare cases the cyst appears at the upper border of the stomach, and that in others it inserts itself between the layers of the transverse mesocolon. In a case upon which I operated it had worked its way through the subperitoneal tissue into the right loin, and was looked upon by Professor Montgomery and myself as a hydronephrosis. As a rule, the pancreatic cyst is immovable, but in rare instances it is movable. When a hand is placed in the loin and another on the abdomen, ballottement may be appreciated. If the distended stomach or colon overlies the tumor there will be a tympanitic percussion-note, but when the tumor reaches the abdominal wall there will be a dull percussion-note. On inquiring into the history of these cases it will be found frequently that there has been a severe injury to the upper abdomen.

**Treatment.**—Exploratory incision makes the condition clear. In the majority of cases the cyst is incised, emptied, and stitched to the wall of the abdomen. This operation may be done in two stages—first, exposing the cyst and fixing it to the abdominal wall; second, when adhesions have formed, opening it. As a rule, however, it is performed in one stage, the abdominal cavity being carefully protected by gauze. Some authors advocate exposing the cyst, opening and evacuating it through the abdominal wound, and draining through the loin. Complete extirpation is usually impossible because of the adherence of the cyst. If the cyst is movable, extirpation may be carried out, but the safest operation consists of incision and drainage.

**Tumors and Other Growths of the Pancreas.**—The pancreas may be affected with sarcoma, carcinoma, adenoma, tuberculous disease, or syphilis. Primary tumors are very rare. Billroth in 1884 removed an adenocarcinoma. Finney reported 1 case and collected 16 from literature which came to

operation ("Annals of Surgery," June, 1910). He says the diagnosis can only be made by exclusion and that in 25 per cent. of cases the mass is not fixed, but is movable. In Finney's series of cases there were 9 recoveries and 8 deaths. Wm. J. Mayo reports the successful removal of a cyst with sclerosed pancreatic tissue (Ibid., August, 1913).

**Treatment.**—Attempts have been made to remove tumors of the pancreas. After an exploratory incision has determined the condition the pancreas is exposed at the point at which the tumor projects. This is usually done by opening through the gastrocolic omentum. If the tumor is in the tail of the pancreas, however, the exposure may be effected in the flank. When the tumor has been exposed an attempt may be made to enucleate or resect it. Coffey ("Annals of Surgery," Jan., 1911) shows that ligation of the duct does not occlude it permanently. Tumors of the splenic portion of the pancreas have been removed. Total pancreatectomy or complete resection of the head of the gland should not be attempted.

In a large tumor of the head of the pancreas palliate the condition by cholecystenterostomy. Villar reports 13 cases of partial resection of the pancreas for tumors, with 5 recoveries from operation (French Surgical Congress of 1905). Finney's paper deals with 17 cases (see above).

## Injuries and Diseases of the Spleen

**Wounds of the Spleen.**—A considerable wound of the spleen causes great hemorrhage and, if surgical aid is not soon at hand, will almost inevitably produce death. It is caused by a bullet or a stab and, as a rule, other viscera are also damaged. Immediate operation is indicated.

**Rupture of the spleen** (Fig. 621) is unusual if the organ be healthy, but does occasionally occur from crushes. It is rarely found unassociated with other injuries. The spleen may be dislocated as well as ruptured. An enlarged spleen is particularly liable to rupture not only from a crush, but from a kick, a blow, or a fall. Rupture of the spleen produces pain and rigidity in the left hypochondriac region and the signs and symptoms of intra-abdominal hemorrhage. There is tenderness over the spleen, pain over the heart, and great shortness of breath. The bleeding is profuse, but sometimes slow. The splenic blood contains numerous leukocytes and clots rapidly, hence the bleeding may be arrested for a time, and if it should be the patient will not bleed to death rapidly and reaction will generally occur (Ballance). The blood in some cases clots so rapidly that it gathers in the left loin, and is not commonly diffused throughout the abdomen. It gives rise to an increasing area of dulness on percussion in the left flank, which, Ballance points out, seldom shifts when the position of the patient is shifted, as it does in bleeding from other intra-abdominal structures. In some cases, however, the blood remains fluid and spreads throughout the belly, and then there is rising dulness in each flank. The cases reported by Le Dentu and Mouchet shows that the blood may remain fluid ("Bull. de l'Academie de Med.," June 16, 1903). In some cases the signs of hemorrhage are late and they may even be deferred until the fourth day (Eisendrath, "Annals of Surgery," Dec., 1902). In some cases there is violent pain in the left shoulder (*Kehr's sign*). Exploratory incision will be required to recognize the condition positively. In Elder's table there are 52 uncomplicated cases, not a case was operated upon (operation was not the rule until 1890), and 84.6 per cent. died. Eisendrath[1] has collected 50 cases operated upon: 56 per cent. recovered and 44 per cent. died. Février[2] has collected 56 ruptures of the spleen. In 46 cases operation was performed and the mortality was 50 per cent. E. Berger ("Archiv. für klinische Chirurgie," Bd. 28, Heft 3)

[1] "Jour. Am. Med. Assoc.," Oct. 25, 1902. [2] "Rev. de Chir.," Nov., 1901.

collected 168 fatal cases of rupture of the spleen: 145 died during the first day and every one of them died from hemorrhage. After the first day 23 died. In 90 per cent. of the entire series hemorrhage caused death; in 10 per cent. infection was responsible for death. Vedova collected 194 cases of splenectomy for traumatic rupture, with 65 deaths, a mortality of 33.5 per cent. ("Practical Medicine Series," vol. ii, 1913). Hemorrhage is the great danger in ruptured spleen—hemorrhage from the parenchyma rather than from the great vessels. The parenchyma is friable and contains multitudes of capillaries and veins, there is no muscular tissue, divided vessels do not tend to contract, and the capsule is thin (the elder Senn, in "Jour. Am. Med. Assoc.," Nov. 21, 1903).

Fig. 621.—Fauntleroy's case of ruptured spleen. External surface.

**Treatment of Wounds and Rupture.**—The treatment is evident from the previous remarks. It is as follows: Open the abdomen immediately, the patient being surrounded with hot bottles and hot salt solution flowing into a vein. Explore the spleen and other viscera. If the spleen is damaged, we may do splenectomy (total or partial), may use the suture, the cautery, or the tampon, and any other visceral injuries are, of course, attended to.

The usual operation has been total splenectomy. In partial splenectomy only the injured part is excised and the wound margins are sutured.

The arrest of hemorrhage by suture is known as *splenorrhaphy*. Lamarchia, in 1896, was the first to perform this operation. The tear or wound is sutured with catgut and the suture line is covered with omentum. Berger collected 14 cases of suturing, with 2 deaths, but these were injuries of less severity than those requiring splenectomy. In some cases the tampon can be used. Berger collected 10 cases, with 1 death. Another method is to crush the splenic structure slowly with broad forcipressure forceps and suture the crushed margins with catgut. Senn followed this plan. George Ben Johnston (paper read before Johns Hopkins Med. Soc., March 2, 1908) has collected 150 cases of splenectomy for wounds or ruptures, with 99 recoveries and 51 deaths, a mortality of 34 per cent.

**Abscess of the spleen** is a rare condition which is usually metastatic in origin. It may follow typhoid, may develop during pyemia, or may result from injury. Chronic suppuration may be due to tuberculosis or actinomycosis. Pain is felt, and enlargement is noted in the splenic region, and the symptoms of pyemia exist. The abscess may become adherent to the belly wall, may become encapsulated, or may rupture into a viscus or the peritoneal cavity. Fluctuation can seldom be obtained. What is known as a tropical abscess (Fontoynant and Jourdrau, in "Archiv. Prov. de Chir.," No. 11, 1902) may develop during a malarial attack as a result of severe exertion. There are severe pain in the left hypochondrium, dyspnea, and dry tongue. There may or may not be fever. The pus may be sterile.

The **treatment** of abscess of the spleen consists in incising the abdomen at the outer edge of the left rectus muscle, suturing the spleen to the abdominal

wall, opening the abscess, and providing for drainage (Tédenat[1]). If the abscess is adherent to the abdominal wall, incise it directly. Splenectomy has been performed for abscess. In 9 recorded cases of splenectomy for abscess there was 1 death (George Ben Johnston, paper read before Johns Hopkins Med. Soc., Mar. 2, 1908).

**Enlargements and Tumors of the Spleen.**—(See Royale H. Fowler, in "Long Island Med. Jour.," July, 1911.) The spleen undergoes hypertrophy in the course of infectious disease, from amyloid disease, from malaria, from splenic anemia, from tuberculosis, from leukemia, and from Hodgkin's disease. Secondary cancer is seen after cancer of the stomach. Genuine primary tumors are extremely rare. Fibroma, enchondroma, hemangioma, lymphangioma, angioma, and sarcoma occasionally develop. Jepson and Albert reported a case of primary sarcoma of the spleen and collected 31 others from literature ("Annals of Surgery," July, 1904). Primary carcinoma can only arise from fetal inclusion. It is usually medullary and is sometimes melanotic. Secondary carcinoma and secondary sarcoma are more common. Echinoccocus cysts, dermoid cysts, lymph cysts, serous cysts, and blood cysts occasionally develop. The spleen alone may suffer from hydatid disease. The liver is apt to be also involved. "There is but a single recorded case of a dermoid cyst. It was reported by Andral in 1830" (Royale Hamilton Fowler, in "Surg., Gynec., and Obstet.," August, 1910).

Non-parasitic cysts may be unilocular or multilocular.

A blood cyst is usually preceded by injury or an infectious disease. A serous cyst may result from a hemorrhagic cyst, and a blood cyst may be due to hemorrhage into a serous cyst (Fowler, Ibid.). There are on record 12 cases of splenectomy for sarcoma: 9 recovered and 3 died (George Ben Johnston, Loc. cit.).

**Treatment.**—The condition may become clear only after exploratory laparotomy. For some tumors splenectomy is indicated. A hydatid cyst is treated as is a hydatid cyst of the liver (see page 1034). A blood cyst is sutured to the incision in the abdominal wall and is drained.

**Splenoptosis, or Wandering Spleen.**—The spleen may wander into any part of the general peritoneal cavity. This condition is seldom met with except in women. It is most common in women who have borne children. A wandering spleen may undergo atrophy, engorgement, or axial rotation (Sir J. Bland-Sutton). The spleen may be healthy or enlarged from malaria or leukemia. As a matter of fact, it is usually diseased. The organ when displaced drags upon the stomach, producing dilated stomach; it may interfere with the bile-duct, causing jaundice; it may cause intestinal obstruction by forming adhesions, or may cause uterine retroflexion or prolapse by passing into the pelvis.

Sir J. Bland-Sutton[2] says this condition may endanger life, as it may lead to rupture of the stomach, intestinal obstruction, splenic abscess, or splenic rupture. A wandering spleen can be identified by the fact that it has a notch upon its edge, and can be pushed about the abdomen. When the spleen wanders it may be missed from its normal situation. Always examine the blood in order to determine if leukemia or malaria exist.

**Treatment.**—Greiffenhagen advocates suturing the organ in place (*splenopexy*). Most surgeons prefer to perform splenectomy. In a case without leukemia the operation is very successful. Splenectomy for wandering spleen is rarely followed by serious blood changes or other trouble. The reason is that a wandering spleen is usually a diseased organ, having undergone hypertrophy or fibroid change, and other structures have taken on splenic function.

[1] "Rev. de Gynéc. et de Chir. Abd.," July, August, 1901.
[2] "Brit. Med. Jour.," Jan. 16, 1897.

Splenectomy should not be undertaken if leukemia exists. In such a case surgeons usually apply a support and employ medical treatment for the existing disease, but some endeavor to suture the organ in place. If the wandering spleen were enlarged by malaria, I would perform splenectomy. If the spleen were healthy I would surround it with gauze exactly as is done with the kidney in a case of movable kidney. If the spleen were enlarged by leukemia I would not operate at all.

## Operations Upon the Abdomen

**Abdominal Section** (*Celiotomy; Laparotomy*).—There are many different methods of opening the abdomen. The plan selected depends upon the nature and the situation of the disease, and upon the inclinations and the custom of the operator. The abdomen may be opened to attack a recognized seat of disease or injury or to determine what the disease or injury is and where it is situated. Abdominal section performed for the latter purpose is spoken of as exploratory section or exploratory incision.

An incision should not be unnecessarily lengthy, but it should be long enough to permit of thorough exploration and rapid and safe work. A very lengthy incision favors visceral prolapse and renders the patient liable to hernia. Again, a lengthy incision requires longer to suture than a short one, and so operation is prolonged. James E. Moore protests against too small incisions, which are often made as a matter of pride ("Jour. Am. Med. Assoc.," Sept. 16, 1911).

Of recent years exploratory operations have become extremely common, and many abdominal conditions would be unrecognized without such exploration, or would be recognized at so late a period as to be beyond the reach of surgery by the time the diagnosis had been made. This is notably true of the surgical diseases of the stomach. The wise surgeon will not be too radical in employing exploratory operations. The fact that he can explore with such comparative impunity does not release him from the obligation to endeavor by every proper method to make a diagnosis before resorting to operation. I fancy that of recent years the belief that it is almost waste of time to make prolonged efforts to diagnosticate many intra-abdominal troubles because the solution is so much easier by section, has become so common as to have led young and unskilled operators to perform section in cases in which the diagnosis might have been made without this procedure.

Before opening the abdominal cavity for exploratory purposes or to gain access to some area of abdominal or pelvic disease the patient is carefully prepared as for any other operation. In an appendicitis case the patient is moved with the utmost care and is prepared for operation most gently, because of the possible danger of rupturing an abscess. In an emergency case no prolonged or complicated method of cleansing can be employed. The abdomen and loins are scrubbed carefully with soap and water, special attention being given to the umbilicus; the pubic region is shaved, the soapsuds are washed away with sterile water, the surface is gently scrubbed with alcohol and then with a hot solution of corrosive sublimate (1 : 1000), and is covered with gauze wet with the sublimate solution. In emergencies the iodin method can be used after dry shaving (see page 68). As previously stated (see page 67), we no longer regard it as necessary to "prepare" the abdomen the day before. The patient can be prepared antiseptically the morning of the operation, or can be shaved just before etherization, and be cleaned when under ether. The instruments required depend upon the nature of the case. Have at hand an electric light and appliances for throwing salt solution into a vein. Always have the instruments, sponges, and pads counted twice by two people, write down the number, and have two people count them twice after operation. This rule is adopted so that no instrument, sponge, or pad

will be left in the abdomen. Some surgeons do not use abdominal pads and sponges when dry, believing that dry gauze injures the peritoneum and favors the subsequent development of adhesions (Sanger). Believers in this hold that pads and sponges should be wrung out in hot normal salt solution before being used. I find moist pads and sponges satisfactory, but moist packs cannot be satisfactorily adjusted. If dry packs are used, dry pads and sponges may as well be.

**Operation.**—An anesthetic is given. In some cases the patient is placed recumbent; in others, is put in the *position of Trendelenburg* (Fig. 622). In the Trendelenburg position the pelvis is elevated, the intestines fall toward the epigastrium, are removed from the necessity of being handled and from the danger of being bruised, the pelvis is thoroughly exposed, and pelvic work becomes easier and safer. This position should not be used if there is myocardial disease, as the increased pressure in and flow of blood from the inferior cava may cause fatal acute dilatation of the heart (Kraske, of Freiburg, in "Proceedings of German Surg. Congress," 1903). The position is of little use in very fat people (Trendelenburg), and in such subjects may cause intestinal obstruction (Kraske). When this position is employed, the table should be lowered as soon as possible, because gastric hemorrhage may occur (von Eiselsberg). The normal position should not be suddenly assumed, as this may cause intestinal obstruction, the omentum being mixed with coils of intestine, pulling the colon down (Pasteau, in "Bulletins and Mém. de la Soc. Anat. de Paris," July, 1905). The position should not be used in a pelvic abscess (König), as it may lead to a flow of pus from the pelvis into the far more dangerous regions above.

Volvulus or kinking of the ileum and of the large intestine have followed the use of the position. If the Trendelenburg position was employed, before closing the belly return the omentum to its proper position and spread it out (Lauenstein). In every abdominal operation the patient is to be carefully protected from cold, the extremities and the chest are covered with blankets, and sterilized sheets are placed well around the field of operation. The skin is sterilized anew immediately before operating. The surgeon steadies the skin of the belly with the fingers of his left hand, and, holding the knife free in the right hand, makes an incision. For purposes of exploration the incision is made about 2 inches in length, and it is lengthened if it is found necessary. The abdomen may be opened in the median line above or below the umbilicus. This incision is advantageous for operations on the pelvis, for general exploration, and for certain procedures upon the stomach, the intestines, and the left lobe of the liver. The closure of such an incision, however, lacks strength, as compared with the closure of an incision where strong muscles will overlie the scar through the peritoneum and the transversalis fascia. Incision through the semilunar line is practised by a number of operators. A favorite incision is through the rectus muscle. The fibers of this muscle are separated, the structures beneath it are divided, and, after the completion of the operation, the deeper structures are sutured and the parts of the separated muscle are allowed to fall together. The scar resulting from such an incision is well supported and solid, hence the likelihood of hernia developing is diminished. A favorite method with some is to open the sheath of the rectus muscle, retract the entire muscle aside, incise the posterior portion of the sheath and the structures back of it, and, when the operation has been completed, allow the entire muscle to come back into place, thus strengthening the deep-seated scar. When the abdominal trouble is in a region that

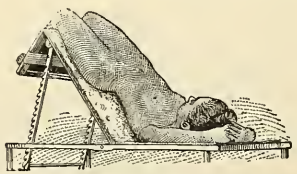


Fig. 622.—The Trendelenburg position.

admits of it, I almost invariably go through the rectus muscle or retract the entire muscle. Besides these methods, there are special incisions, suitable for particular cases: An incision along the costal margin, for reaching the gall-bladder; an incision shaped like the italic letter *f*, for the same purpose; special incisions for certain operations upon the stomach, for abdominal nephrectomy, etc. Some operators have even used a transverse incision in certain pelvic operations. (A full discussion of abdominal incisions will be found in an article by R. E. Farr, "The Journal Lancet," Nov. 1, 1912).

In an operation through the median line the first cut goes to the aponeurosis of the external oblique muscle. Clamp the vessels. Do not hunt for the linea alba below the umbilicus, but go right through or between the recti muscles. Above the umbilicus the linea alba is very distinct and the surgeon often cuts through it. Divide the transversalis fascia, beneath which is a little fat, and expose the peritoneum. The latter structure is recognized by its glistening appearance, by the ease with which it can be pinched up between the finger and thumb, and by the readiness with which its opposed surfaces may be made to glide over each other. On identifying the peritoneum, catch it at each side of the incision with forceps, raise a fold, nick it with a knife, and open the layer by scissors to the length of the external wound. To prevent stripping of the peritoneum a good plan is to anchor it to the belly wall with a stitch on each side of the incision. Through the wound thus made the abdomen and its contents are explored, the trouble located, and determination made as to whether or not further operation is advisable, and, if it is advisable, what form it shall take. It may be necessary to enlarge the wound. This is done by placing the index and middle fingers of the left hand in the belly, with their pulps against the peritoneum, in the line where the surgeon will cut, to serve as supports to the scissors and as guards to intraperitoneal structures. The scissors are introduced and the wound is enlarged upward or downward, going around the umbilicus if necessary. As soon as the incision is complete for work in the lower abdomen or pelvis it is a good plan to push a large pad into Douglas's pouch and leave it there until the operation is finished, when it must be removed. Slender adhesions are stripped off with the finger or are pushed off with gauze; firm adhesions are tied in two places and cut between the ligatures.

The *toilet of the peritoneum* is important after the operation is completed. Following a clean laparotomy, when but little blood has flowed into the cavity, flushing is not required; if much blood has flowed or if septic matter has passed into the peritoneal cavity, after removing the pad from Douglas's pouch flush the belly thoroughly with hot normal salt solution. In a clean case empty out most of the fluid, but let a pint or more remain in the abdomen. In flushing the abdomen bear in mind Monks's observations as to the mesentery. It is a sort of shelf. If we follow down the left side of it with the finger the finger must enter the left iliac fossa; if we follow down the right side of it the finger must enter the right iliac fossa. Hence in order to flush the right cavity carry the nozzle down the right side of the mesentery to its root, and in order to flush the left fossa carry it down the left side of the mesentery to the root (Monks, "Annals of Surgery," Oct., 1903). The retention of the saline fluid in the belly minimizes shock. It is absorbed with great rapidity after the operation if the patient is placed with his head lower than his feet, because in this position the saline fluid gravitates to the diaphragmatic region, where absorption is very active; in fact, in one hour the peritoneal cavity can absorb from 3 to 8 per cent. of the body weight. If there is widespread infection with stomach contents or feces, eviscerate, wipe out the peritoneum with pads soaked in hot normal salt solution, and wipe the intestines carefully, slowly returning them as they are wiped. Extravasated septic matter is apt to collect in the peritoneal

fossæ and between the liver and diaphragm, and these regions must be carefully wiped and irrigated. In cases of septic and purulent peritonitis, flushing, evisceration, and wiping with gauze are not advisable (see page 1024). In some cases it is desirable to drain through a lumbar incision. Rutherford Morison has pointed out that a lumbar opening into the right kidney pouch will drain a fossa which holds over a pint of fluid, and which, when the patient is recumbent, is the most dependent portion of the peritoneal cavity. In some cases a drainage-opening is made through the linea alba, through one side or on each side of the belly, or above the pubis or through the vagina. In septic cases it may be advisable to drain with several pieces of iodoform gauze instead of inserting tubes. After most laparotomies drainage is not needed, but it should be used when stomach contents were extravasated, and it must be used if feces or urine were extravasated, in certain recent septic cases, and when hemorrhage has been severe. We may drain by a rubber tube, strands of gauze, a cigarette drain, or a glass tube. If a glass tube is used, it is introduced at a lower angle of the wound and reaches the bottom of the pouch of Douglas. The tube is repeatedly emptied during the progress of the case by means of a syringe. Before closing the wound arrest hemorrhage and ask for the count of the instruments and pads in order to know that nothing foreign has been left in the belly.

It is highly important that an abdominal incision shall be accurately closed, for any failure of neat approximation will, in all probability, result in the formation of a hernia through the cicatrix. Various methods have been employed. Probably the majority of operators use layer sutures, sewing up the peritoneum with a continuous suture of catgut, and the aponeurotic layers with the same material or with chromicized catgut, and closing the skin with either interrupted sutures of silkworm-gut or a subcuticular stitch of catgut, silkworm-gut, or silver wire. Other operators close the peritoneum with a continuous suture of catgut, then pass silkworm-gut sutures through all the other structures, leaving them for the time untied: put in and tie layer sutures of catgut or of chromicized catgut, and then tie the silkworm-gut sutures. A layer suture makes a beautifully neat approximation, and is frequently quite satisfactory; but I have become persuaded that the dead space, so often left unobliterated when this method of suturing is employed—a space in which blood and inflammatory exudate may gather—is a danger to the future integrity of the wound. The combination of a dead space with catgut, a material that is always somewhat uncertain, is an unfortunate one from the surgical point of view. I have returned in many cases to the use of the through-and-through suture, applied according to the method of the late Dr. Joseph Price. This suture is inserted with the straight needle, is composed of silk or of silkworm-gut, is put in close to the margin of the skin, gathers up a great deal more muscle than skin, and then passes close to the margin of the cut peritoneum and transversalis fascia. When these sutures are adjusted the peritoneal edges are brought into accurate and firm apposition, the peritoneal surface is overlaid with abundant muscle, the skin edges are brought into neat approximation, and the formation of a dead space is rendered impossible. When passing the sutures have a gauze pad under the wound and be very careful not to include bowel or omentum in the stitches. It is necessary to tighten and tie most carefully to prevent omentum being caught in the loop of the stitch. After closing a laparotomy wound, dress with aseptic gauze and apply a flannel binder. In badly infected cases the wound is often kept open.

If a 2-inch incision has been closed without drainage and primary union has taken place, the patient can usually get out of bed in seven or eight days. A large incision offers greater danger of subsequent hernia, and the patient should be kept in bed for two or three weeks. If the wound has been kept open

for drainage, a prolonged retention in bed may be necessary. I get patients up at an earlier period than used to be my custom, but I do not get them up as do Kümmell and others in from one to three days (Kümmell, in "Zentralblatt f. Chirurgie," 1908). To get them up reasonably early lessens constipation, favors an early return of appetite and strength, and diminishes the risk of postoperative thrombosis and embolism and of bronchitis. We must bear in mind that if there is myocardial degeneration very early getting up may prove disastrous or even fatal, and that in septic diseases there is often myocardial degeneration (E. W. Foote, in "Progressive Medicine," June, 1909). In a case in which an incision of considerable length has been made, an abdominal support should be worn for a variable time. It limits the movements of cough, laughter, etc., and *reminds* the patient of the necessity of caution in lifting, hurrying, etc.

The **after-treatment** depends somewhat on the case, but certain general rules can be laid down. The late J. Greig Smith said many wise things, and among them this: "A golden rule in the treatment of cases of celiotomy is to let the patient alone. Everything approaching to meddlesomeness is to be condemned. The patient must not be upset by fussy applications of tentative therapeutics; when an emergency arises, it is to be met, promptly and decisively, by a method which has been approved trustworthy" ("Abdominal Surgery"). In many cases immediately after the operation the patient must be treated for shock by methods previously set forth. The treatment of vomiting resulting from the administration of an anesthetic is discussed on page 1202. If vomiting persists during the third or fourth day it may be due to acidosis, but is probably due to the development of inflammation which has caused intestinal paresis; and if it is so produced, medicine is practically useless. In this condition there is usually marked tympanitic distention, and vomiting is, in a sense, a relief. Nothing should be given by the mouth and the patient should be fed entirely by enemata. The insertion of a rectal tube and its retention for a considerable time may afford relief. Lying on the side is more comfortable than recumbency. Washing out the stomach from time to time gives great comfort and is often of real service.

In the average case of celiotomy, in which persistent vomiting does not occur, the question of feeding is of much importance. Usually, for the first twelve or twenty-four hours, nothing is given by the mouth but small quantities of hot water. The day after the operation, if everything is satisfactory, food is given to the patient. In many cases, however, food is not given by the stomach for forty-eight hours and the patient is fed by the rectum during the wait. He should not be given milk because it will not be easily digested, may lead to nausea, and causes flatulence. Peptonized milk, if the patient will take it, does not possess these hurtful qualities. At first albumin-water or liquid beef peptonoids should be given, and later meat-juice, beef-jelly, broth, etc. Food is given every third or fourth hour, and stimulants are administered if required. After the first twenty-four or forty-eight hours considerable quantities of plain water or Poland water should, if possible, be taken, to favor elimination by the kidneys. Hot coffee is not only a stimulant, but is an excellent diuretic. The urine is always scanty after an abdominal operation, and a normal daily amount is not voided for ten days or more. Solid food is not given for seven or eight days. The patient is apt to suffer greatly from thirst, in spite of the hot water given during the first twelve to twenty-four hours. He seldoms takes and should never have any great amount of hot water, and iced water and ice are inadmissible and tend to induce nausea and vomiting. Sucking ice or sipping water draws air into the stomach and causes distention. Thirst can be much mitigated by enemata of salt solution. J. Greig Smith recommended an enema composed of from 4 to 20 oz. of tepid

water and some brandy. Usually, after the first twenty-four hours, a sufficient amount of liquid can be given to keep the patient free from actual distress.

The bladder must be watched to see that retention does not occur. If retention occurs, a clean catheter must be used at regular intervals. If tympanitic distention occurs after forty-eight hours, a saline purgative should be given and it should be followed by an enema of turpentine. The rectal tube is frequently of signal service in such cases. If obstruction develops, it is treated as directed on page 989.

In any ordinary case after operation the bowels should be moved after forty-eight hours as a prophylactic measure against distention, peritonitis, and obstruction. From four to eight 1-dram doses of Epsom salts are given, in hot water, the solution having been filtered through gauze. The saline is followed by the administration of an enema consisting of soap, water, and $\frac{1}{2}$ oz. of castor oil. Should opium be given? Never as a routine, and not to secure sleep; but if the patient is in pain which not only harasses him, but causes him to turn and shift in torturing restlessness, one or possibly two hypodermatic injections each containing $\frac{1}{4}$ gr. of morphin can be given with confidence that the good will overbalance the harm.

**Operation for Appendicitis.**—Before operating try to locate the situation of the appendix, and the relation the area of infection bears to the ascending colon. The incision should be over the seat of disease. In the rare left-sided cases and in median cases the incision is median. In some cases in which the appendix is posterior the cut may be in the loin. In 1 case I opened a purulent collection through the rectum. In the vast majority of cases the incision is made in the right iliac region.

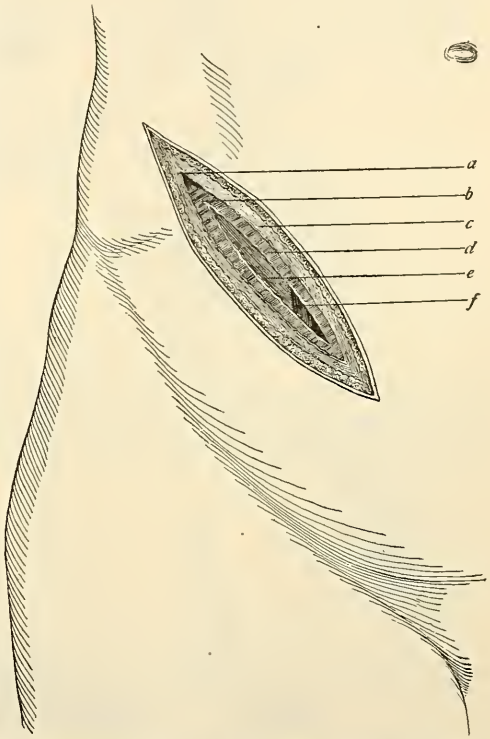



Fig. 623.—Resection of the vermiform appendix, incision through the abdominal wall: *a*, External oblique muscle; *b*, internal oblique muscle; *c*, aponeurosis of external oblique; *d*, aponeurosis of internal oblique; *e*, peritoneum; *f*, outer border of rectus abdominis muscle (under it the deep epigastric vessels) (Kocher).

In acute appendicitis, when there is not thought to be a distinct abscess, the incision usually made is 2 inches internal to the anterior superior iliac spine and perpendicular to a line drawn from the spine to the umbilicus (Fig. 623). The skin incision is usually 3 inches in length, the upper third of the incision being above the omphalospinous line; the incision in the peritoneum is about 2 inches in length, but if there are many adhesions, it may be necessary to make it much longer. The oblique incision may be carried out as advised by McBurney, the muscles being separated by blunt dissection. By this method very few nerve-fibers are divided, and hence the operation is not followed by marked muscular wasting, a condition which strongly predisposes to hernia.

Further, as Van Hook[1] points out, the oblique incision enables the surgeon to reach freely all the ordinary areas of appendix trouble, the wound is parallel with the lines of traction of the abdominal muscles, and does not tend to gape widely. In an acute case I make an oblique incision, but cut the muscles

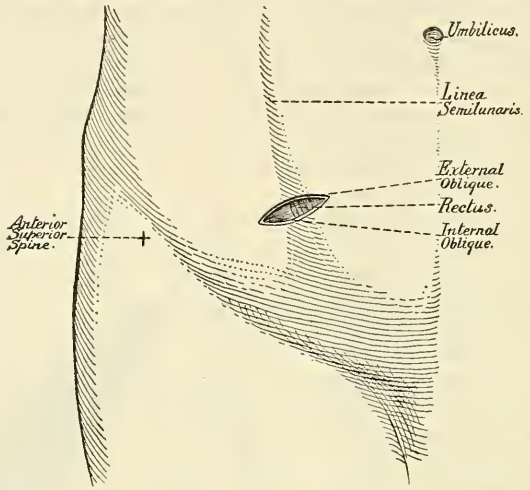



Fig. 624.—Davis's small transverse incision for simple cases.

(Fig. 623). In an interval case I separate the muscular fibers. Battle's incision at the outer edge of the rectus muscle is preferred by many surgeons. The anterior layer of the rectus sheath is opened longitudinally, the rectus is drawn

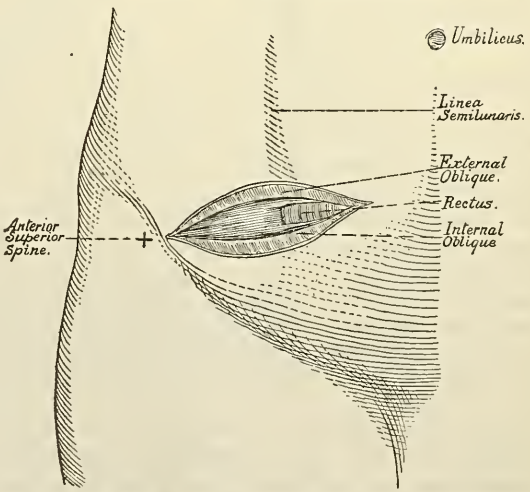



Fig. 625.—Davis's large transverse incision for difficult cases.

inward, and any existing portion of the posterior rectus sheath with the transversalis fascia and peritoneum is incised.

I have used *Davis's transverse incision* (Figs. 624 and 625) in many interval cases with entire satisfaction (Gwilym G. Davis, in "Annals of Surgery," Jan.,

[1] "Jour. Amer. Med. Assoc.," Feb. 20, 1897.

1906). This incision does not divide arteries, but it divides the deep muscles in the direction of the nerves, hence the nerves are not injured. The center of this incision is almost over the base of the appendix. Davis describes his incision as follows:

"*For easy cases* the incision is made directly transverse, $1\frac{1}{2}$ inches long. Its center is to be on the semilunar line on a level with the anterior superior spine. The aponeurosis of the external oblique is divided in the line of the skin incision, but obliquely to the direction of its fibers. The fibers of the internal oblique and transversalis muscles are parted—not cut—in the same line as the structures above. The peritoneum is then opened and the incision is carried inward, first through the anterior layer of the sheath of the rectus. A blunt retractor $\frac{3}{4}$ inch wide is then inserted and the muscle drawn toward the median line. This exposes the transversalis fascia and peritoneum posteriorly, which are then also divided. Thus is obtained a triangular opening with its base of $\frac{3}{4}$ inch and two sides of about 1 inch long, which is ample for simple cases.

"*For Difficult Cases.*—If the case is a difficult one, the outer end of the incision is prolonged to the anterior spine or even above and inwardly through the sheath of the rectus to within 1 inch of the median line. This will give an opening 4 to 5 inches long, according to the size of the patient, sufficiently large to insert the hand if necessary and through which the appendix can be extracted under almost all circumstances."

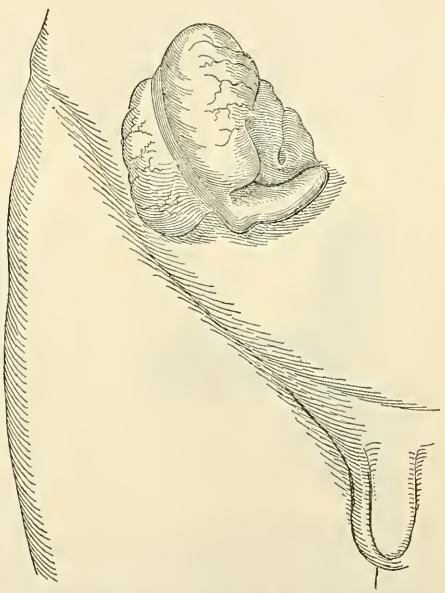


Fig. 626.—Radical operation for appendicitis (Kocher).

After opening the peritoneum examine very gently to detect the situation of the appendix, and if there are or are not adhesions. In a very recent case and in a very acute case there will probably be no adhesions unless there have been previous attacks. Surround the region of infection with packs of plain gauze, each strip being $2\frac{1}{2}$ inches wide, 15 inches long, and four layers in thickness. The edges of the wound should be lifted up by retractors and the strips inserted around the cut, between the parietal peritoneum and intestines and to a distance of 3 inches from the wound. Strips of gauze are passed, when possible, below the appendix to prevent entrance of infected material into the pelvis, and a piece is pushed upward toward the liver (Van Hook). Over the packing gauze, which it may be necessary to leave in place after the operation, other pads are packed. The appendix is sought for by finding the colon. The colon is found by following the parietal peritoneum with the finger. The course of the finger is first outward, next backward, and finally inward; the first obstruction it encounters is the colon. The fact that it is the colon can be confirmed by finding the longitudinal bands. The anterior longitudinal band leads directly to the appendix. Pass the finger down to the head of the colon, find the appendix, usually posterior and internal, and lift it and the head of the colon into the wound. In many cases it will be advisable to deliver the head of the colon from the belly (Fig. 626); in other cases this will not be

necessary, in some it will not be possible. If adhesions exist, they must be gently and carefully separated. Barker's method (Fig. 627) is a very satisfactory mode of removing the appendix. It is done as follows: Turn up a cuff of peritoneum, pull down the other coats, ligate at the base, cut through the tube, let the musculomucous stump retract, and tie or suture the peritoneal cuff over the stump. Another method, which is the one I usually employ, is as follows: Pass a ligature through the meso-appendix, as shown in Fig. 628, *A*, tie the ligature, and cut off the meso-appendix below the threads. Crush the stump of the appendix with strong straight hemostatic forceps. This divides the mucous membrane, submucous tissue and muscular coat, and leaves the peritoneal coat undivided. Remove the forceps. Surround the appendix with a catgut ligature and tie the ligature in the groove produced by the crushing. When the ligature is tied, peritoneum is brought against peritoneum. Cut off the appendix between the ligature and a clamp by the cautery, a knife, or scissors. Disinfect the stump of the appendix by pure carbolic acid. The stump beyond the ligature contains mucous membrane and muscle, which are lifted out with forceps and scissors. Suture the fringe of the meso-appendix, invert the stump into the wall of the colon, and suture a portion of the wall over it by inversion stitches. Figure 628 shows an older method still used by many. The meso-appendix is

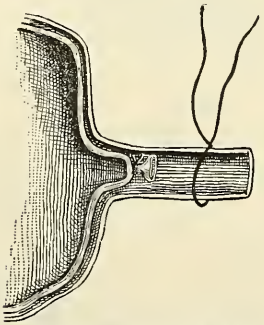


Fig. 627.—Barker's technic of operation for removal of the appendix.

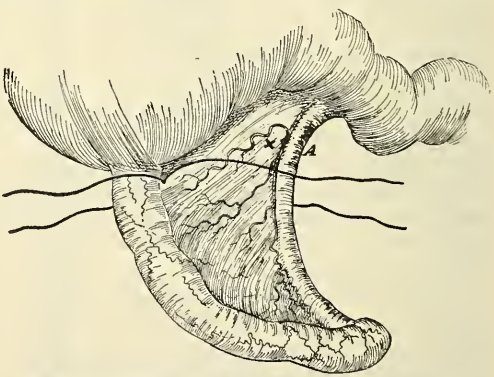


Fig. 628.—Ligation of appendix and meso-appendix.

tied off by one ligature, the appendix is not crushed, but is tied off by another ligature, and both structures are cut off below their respective ligatures. The stump is disinfected with pure carbolic acid or the cautery, inverted, and the fringe of the meso-appendix is sutured. This method does not entirely remove the appendix, but inverts glandular tissue into the wall of the bowel. The stump may not be completely asepticized by the carbolic acid and hence may lead to postoperative abscess, dense adhesions or fecal fistula, or the undestroyed lymphoid structure may cause further trouble, even persistent ill health (Joseph Price). Some remove the appendix by an elliptical incision around its base, and close the colon wound by Lembert sutures. This method, of course, removes the appendix completely. Dawbarn surrounds the appendix with a continuous Lembert purse-string suture of silk. This is inserted in the superficial layers of the cecum, $\frac{1}{2}$ inch from the appendix. The appendix is divided so as to leave a stump never shorter than $\frac{1}{2}$ inch. The lumen of the stump is gently stretched by inserting a pair of mouse-toothed forceps and opening the blades. The stump is then invaginated into the cecum, that is, it is turned "outside in." The sutures are tightened, and while this is being done, the mouse-tooth forceps used in effecting inversion are withdrawn. Finally, the sutures are tied (Robt. H. M. Dawbarn, in "Internat. Jour. of

Surg.," May, 1895). In this method the stump is not ligated and hemorrhage is liable to take place into the gut. Deaths from such hemorrhage are on record. The retained bit of appendix drains into the colon. I believe it is a mistake to trust to simple ligation and to fail to bury the stump left after appendectomy. If a surgeon follows this plan in any large number of cases he will now and then have a case in which the ligature slips and feces pass into the peritoneal cavity, or cases of temporary fecal fistula, or cases of intestinal obstruction from adhesion of some portion of the bowel to the exposed stump (Murat Willis, in "Annals of Surgery," July, 1908). I do not believe that a a buried stump increases postoperative pain.

If there is no pus and no extravasated feces, if the peritoneum is not seriously affected, if the appendix is not gangrenous or perforated, and if there is no pus within the appendix, remove the pads which were inserted last, irrigate with hot salt solution, remove the strips of gauze which were inserted first, and close the wound. If any of the above conditions were found, remove the infected pads, but leave in place the strips which were first inserted in order to limit infection and secure drainage. Pass sutures through wound edges, tie some of them and leave some untied until gauze is removed at a later period (Van Hook).

If an operation is performed in a distinct interval, pus is absent and the surgeon can proceed without apprehension. If there is any question of the presence of pus, surround the region with gauze, as suggested above, before breaking down adhesions and liberating the appendix. An interval operation should not be performed until three weeks after an attack. In an interval case McBurney proceeds as follows: He makes the skin incision in the direction of the fibers of the external oblique muscle, separates the fibers of this muscle by blunt dissection, retracts them, separates the fibers of the internal oblique and the transversalis muscles in the same way and retracts them, and opens the transversalis fascia and peritoneum. No muscle-fibers are cut and hernia is not apt to follow. Such a wound is closed as follows: a continuous catgut suture for the peritoneum, sutures of chromic gut for the transversalis fascia, the muscles are restored to place, the aponeurosis of the external oblique is sutured with chromic gut, and the skin is closed by interrupted sutures of fine silk or silk-worm gut or by a subcuticular stitch.

If an *abscess* is believed to exist, make an incision parallel with Poupart's ligament and over the area of dulness on percussion (Willard Parker's oblique incision). If the abscess is adherent to the anterior abdominal wall such an incision will not enter the free peritoneal cavity. If, after opening the abdomen, an abscess is thought to exist, although it is not adherent to the anterior abdominal wall, surround the abscess with gauze before opening it, as directed under acute appendicitis. The gauze is placed under the margins of the incision in the peritoneum all around the appendix area; a piece is carried toward the pelvis and another piece toward the liver. Overlay this gauze with gauze pads (Van Hook). Adhesions are broken through with the finger, and when pus appears it is at once wiped away. Remove the appendix in most cases, but not in all. If the appendix lies loose in the abscess cavity, if it is sloughed off or but loosely attached to the abscess wall, remove it. If the appendix is firmly fixed in the abscess wall and must be dug out of a mass of inflammatory material do not remove it. To remove it under these circumstances may rupture the wall and disseminate the pus into regions not protected by pads and gauze. Deaver and others tell us always to remove the appendix. I do not believe this to be a safe rule to follow. To insist on removing the appendix may cause death. When the appendix is left it usually sloughs away. It is true, a fecal fistula may result, but this is in the large bowel and usually heals spontaneously. Even if a fecal fistula forms and does not heal, the surgeon acted properly in not removing the appendix, be-

cause a fecal fistula may be remedied by another operation. It is rarely that secondary abscess forms, and there are not a great many cases recorded in which an appendix has subsequently given serious trouble when left after operation. In fact, in many cases the appendix is destroyed or obliterated by inflammation. In some cases, however, a secondary operation will be required because of a fecal fistula, a persistent sinus, or an acute inflammatory attack. When Deaver decides to remove such an appendix, he makes an incision in the median line of the abdomen, packs around the periphery of the abscess with gauze, opens the abdomen by another incision, removes the appendix, disinfects, inserts drainage, and then removes the surrounding gauze and closes the median incision. In every abscess case search for fecal concretions and remove them if found. Irrigation should not be employed in appendicular abscess. The force of the stream may break down barriers of lymph and spread infection. After the evacuation of the pus, whether the appendix was removed or not, take out the pads, but leave the long strands of gauze first placed to keep the wound open. Introduce iodoform gauze into the abscess cavity and insert a rubber tube, partially suture the wound, and dress with dry gauze. In forty-eight hours all the strands of gauze are removed and fresh pieces are inserted for drainage. After this period the gauze drain is changed daily. Morris maintains and proves that large pieces of gauze sometimes cause intestinal obstruction and iodoform gauze sometimes causes iodoform-poisoning, but the risk, it seems to me, should be taken. An interval case should be up and about in from ten days to two weeks after operation. An abscess case may require a much longer time for complete recovery. A fecal fistula sometimes results in cases in which the appendix was not removed, and occasionally forms when it was removed.

If on opening the abdomen pus is found, unlimited by adhesions but widespread in the peritoneal cavity, remove the appendix, and then bear in mind Murphy's wise counsel as to how to treat general peritonitis (see page 1025). Put a drainage-tube into the pelvis and one in the appendix region, place the patient in Fowler's position, and administer salt solution by continuous proctoclysis at a low pressure. The after-treatment of an ordinary appendix operation is that advised after celiotomy (see page 1070).

*Mortality After Operations for Appendicitis.*—The interval operation is practically without mortality. In over 1000 cases Treves had 2 deaths. In 446 cases of chronic appendicitis the Mayos had 1 death ("Report of St. Mary's Hospital for 1912"). In grave acute cases the mortality is large. In 100 consecutive cases of this character collected by Hearn and operated upon in the Jefferson Hospital by Keen, Hearn, and DaCosta, there were 8 deaths. As previously stated, Maurice H. Richardson reported a death-rate in such cases of 18 per cent. in 750 cases. Deaver reports from the German Hospital 144 cases, with a mortality of 17.8 per cent. He eliminates 1 death from diabetes, 1 from pneumonia, and 1 from phthisis, and estimates his personal mortality at 15.9 per cent. (Deaver and Ross, in "Jour. Am. Med. Assoc.," Oct. 5, 1901). In 124 cases (including all chronic cases and those acute cases in which the inflammation had not extended beyond the peritoneal coat) there was 1 death. In 347 cases of acute and suppurative appendicitis operated upon in the Mayo clinic in 1912 there were but 2 deaths ("Report of St. Mary's Hospital for 1912"). I fancy most of these cases came from a distance and hence the proportion of violent and rapid cases must have been less than is usually the rule in a great city In Burgess's 500 consecutive operations there were 40 deaths ("Brit. Med. Jour.," Feb. 24, 1912). When infection was limited to the appendix the mortality was .74 per cent (135 cases); when there was circumscribed abscess it was 4.64 per cent (213 cases); when there was diffuse spreading peritonitis it was 19.07 per cent. (152 cases). The usual causes of death are

intestinal obstruction, septic peritonitis, septic endocarditis, pylophlebitis, hepatic suppuration, metastatic abscesses, endocarditis, and gangrene of the bowel. In a further report from September 1, 1902, to September 1, 1903, Deaver reports 566 cases in the German Hospital, with an aggregate mortality of 5 per cent. In cases with diffuse peritonitis the mortality was 31 per cent. In abscess about a necrotic and perforated appendix it was 12 per cent. In early appendicitis or when disease was confined to the appendix it was 0.8 per cent. In 107 cases of circumscribed abscess in which Burgess removed the appendix the mortality was 1.86 per cent. In 106 cases in which he did not remove the appendix it was 7.54 per cent. ("Brit. Med. Jour.," Feb. 24, 1912).

**Appendicostomy (Weir's Operation).**—This operation was devised by Weir, of New York, in 1902. It consists in opening the abdomen, finding the appendix, fastening this structure to the skin, closing the rest of the wound, opening the appendix to see that it is patent, and applying a temporary ligature to prevent leaking. The temporary ligature is removed in a day or two, and a few days later the adherent and open appendix is used as a route for the introduction of irrigating fluids. The operation is of the greatest value in chronic ulcerative colitis, as it enables us to irrigate thoroughly the large bowel. Daily a large tube is passed into the rectum and a small tube into the appendix. The fecal matter is washed out of the bowel through the rectal tube by salt solution, and then a 1 : 5000 solution of silver nitrate or

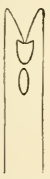


Fig. 629.—Eye of the calyx-eyed needle.

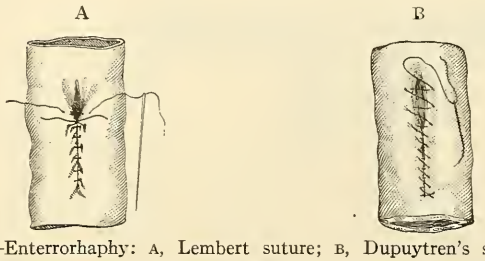



Fig. 630.—Enterrorhaphy: A, Lembert suture; B, Dupuytren's suture.

bismuth and starch water (1 dram to 1 oz.) is used to irrigate the colon. It is injected by way of the appendix and it runs out of the rectal tube. It is used for the same purpose in some cases of tuberculous rectal or anal fistulæ. A most extraordinary suggestion is that appendicostomy be performed in epileptics, so that the opening may be used to flush the bowel, a suggestion which I will not act upon. When the fistula exists, it does not leak to any appreciable degree. When we wish to close it we insert within the lumen of the tube the Paquelin cautery at a red heat. This destroys the mucous membrane and the fistula closes (Robt. Weir, in "Med. Record," August 9, 1902).

**Enterorrhaphy, or Suture of the Intestine.**—Surgical opinion has greatly altered in regard to this operation since the day when John Bell wrote his famous attack on Benjamin Bell. John Bell said: "If in all surgery there is a work of supererogation, it is this operation of sewing up a wounded gut." To-day we know that if in all surgery there is a proceeding of imperative necessity, it is the sewing up of a wound in the intestine. To perform this operation take fine sterile silk and thread a thin, round, straight, calyx-eyed needle with it (Fig. 629). The needle is very useful, as it can be threaded rapidly by pushing the calyx eye down upon the silk thread while the latter is kept taut. *Lembert's suture* (Figs. 630, A, 631, and 632) was devised in 1823. Lembert used it on animals, but never on man. It is inserted at right angles to the wound. It goes down to, but not through, the mucous membrane. It is formed by picking up a fold of the intestine ($\frac{1}{12}$ to $\frac{1}{8}$ inch wide) $\frac{1}{8}$ inch from

the edge on one side of the wound, passing the needle through, picking up a fold on the opposite side of the wound, and passing the needle through. On tying

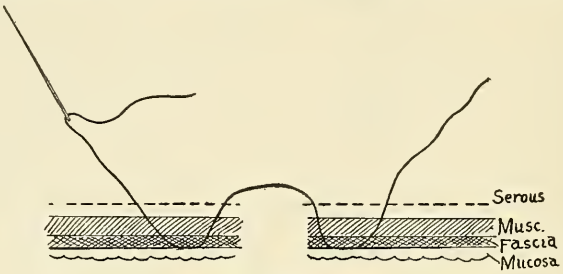



Fig. 631.—Lembert's suture.

the threads the serous membrane is inverted and peritoneum is brought into contact with peritoneum. For many years it was taught that this suture

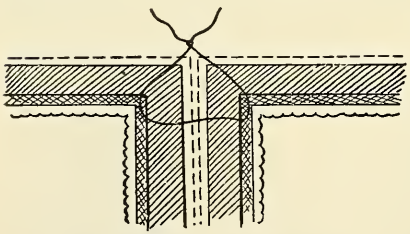


Fig. 632.—Lembert's suture closed.

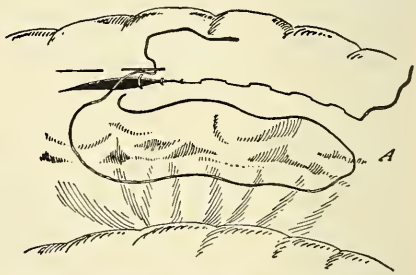



Fig. 633.—Cushing's right-angled suture (Senn).

should include only the serous coat, but Halsted, in 1887, showed that it must include the tough submucous coat. The submucous coat is strong and will hold a suture. The other coats are thin, tear easily, and will not hold a suture.

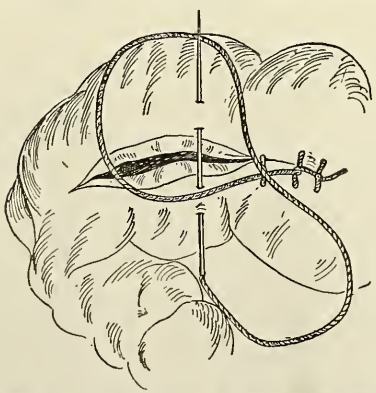


Fig. 634.—Ford's stitch, showing a Lembert insertion and the needle passed so as to tie a single knot by drawing it on through.

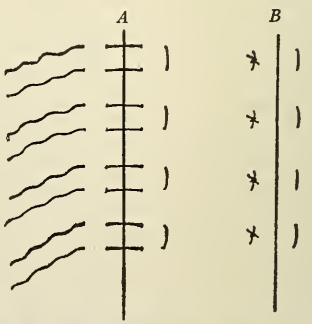



Fig. 635.—*A*, Halsted sutures untied; *B*, Halsted sutures tied and serous surface inverted.

So thin are the coats that a surgeon could not suture the serous coat alone were he to try. Sutures which include only the muscular and serous coats tear out easily. *Dupuytren's suture* (Fig. 630, B) is simply a continuous Lembert

## EXPLANATION OF PLATE 11.

### Intestinal suture, all knots inside (Connell).

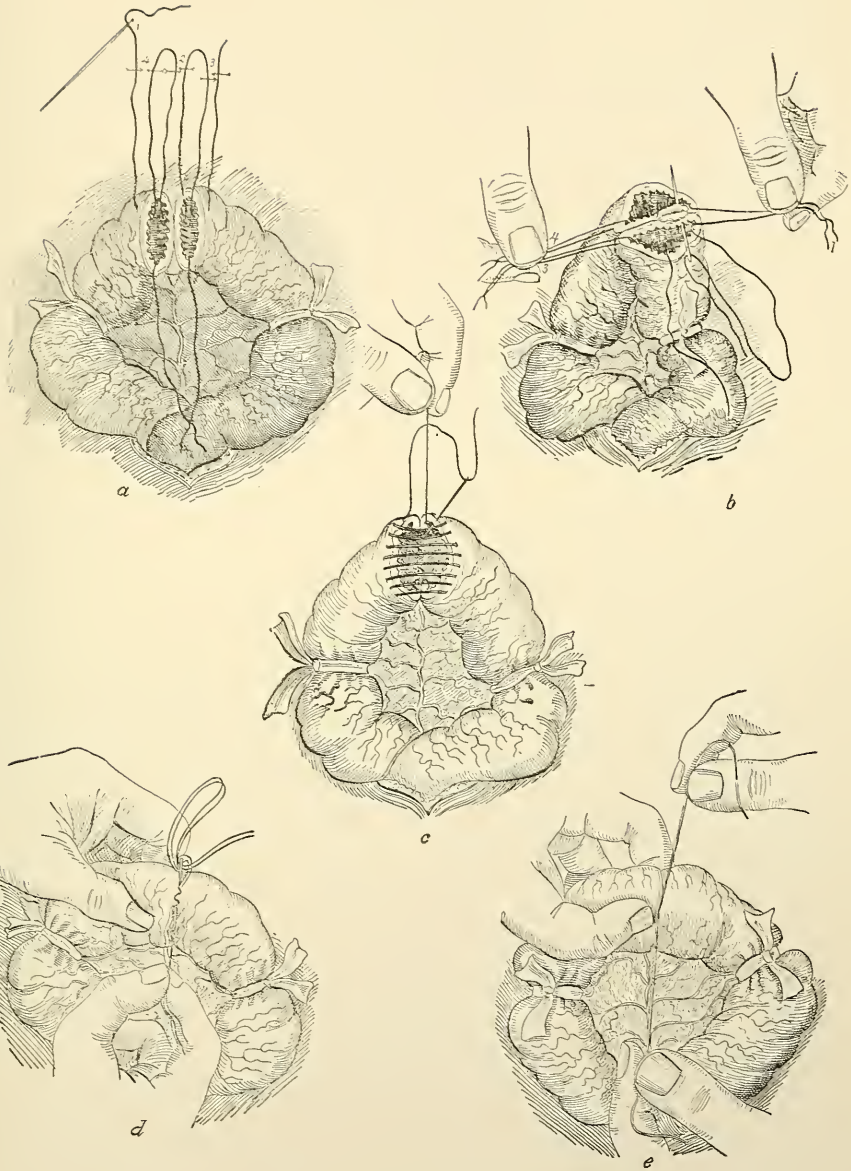
*a*, Suspending loops 2, 3, and 4 are made with one thread inserted at a point two thirds of the distance from mesenteric to convex border. The needle with suture is passed through the four walls of the cut ends, and that portion of suture within each lumen is drawn up to a sufficient length, then cut, and the contiguous threads tied at the points indicated by the arrows; thus having as a result four suspending loops dividing the circumference of each cut end into thirds. Instead of employing four suspending loops which divide the circumference of the bowel into thirds, we may use but two loops, and thus divide the circumference into halves; or, if available, the "holder" devised by Dr. E. H. Lee can be recommended highly, and will be found a most efficient aid in maintaining the cut edges in apposition. (The description of the instrument will be found in the "Annals of Surgery," January, 1901.)

*b*, Loop 2 has been cut away, and loop 1 takes its place in one hand of the assistant, with loops 3 and 4 held in the other hand, thereby bringing into apposition that portion of the walls to be included in the second third of the suture. The operator continues the suture to the points of insertion of loops 3 and 4, where again a back stitch is taken, to fix the suture and prevent a purse-string contraction of the same. The white elevation in the center of illustration, representing mesentery, shows that that portion of the intestinal wall not covered by peritoneum, at the mesenteric border, has been secured in the suture.

*c*, The needle, after having entered the lumen, is passed out again on the same side $\frac{1}{8}$ inch distant; then over to the opposite cut end, where it is inserted from without in, and again emerges from within out, on the same side. This step—the taking of a bite—is repeated alternately on opposing margins until the necessary number of stitches have been inserted. It will be observed that when the needle enters the lumen the last time, it makes what might be termed a half-stitch, as it does not return again *through* the wall; but having reached the point where the suture was commenced, the free end and the needle end will complete the last stitch, when tied, on the mucosa. The needle at this point is then brought out of the lumen at the angle of wound alongside of the free end of the suture. The cross-over stitches are next carefully drawn up, thus bringing into contact the opposing serous surfaces at every point except where the suture ends still protrude.

*d*, The eye-end of threaded needle is made to emerge alongside of the suture ends, and is then withdrawn a little, which causes its thread to form a loop, through which the assistant passes the ends of the suture. The operator next withdraws the threaded needle, at the same time bringing with it the suture ends, and they present externally at the point of withdrawal of the needle. The serous coats throughout the entire circumference are now in apposition, and the suture ends can be tied.

*e*, By slight traction on the suture ends the opposing mucous surfaces are brought in close contact; the suture ends are then tied firmly, and deep between the serous coats, thus tying the knot upon the mucous coat, and the ends then cut off short.


a
b
c
d
e

suture running obliquely across the wound. *Cushing's right-angled suture* (Fig. 633) is a continuous suture catching up the submucous coat and serving to invert the serous layer. Ford, of San Francisco, employs a continuous inversion suture, which is tied in a single knot each time it is drawn through (Fig. 634). *Halsted's mattress* or *quilt suture* is shown in Fig. 635. Each stitch picks up the submucous coat. Mattress sutures do not tear out easily, they appose evenly considerable surfaces, and do not constrict the tissue as much as Lembert stitches. The *Czerny-Lembert suture* is a suture passed through the serous membrane on one side of the wound, made to perforate the mucous membrane, and to emerge at a corresponding point of the serous membrane. A Lembert

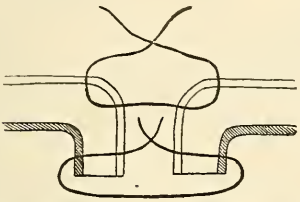


Fig. 636.—Czerny-Lembert suture.

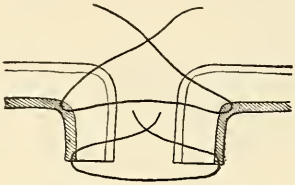


Fig. 637.—Czerny-Lembert suture as at present used.

suture is added (Fig. 636). As at present used, the Czerny suture is carried to, but not through, the mucous membrane (Fig. 637). *Gussenbauer's suture* is similar to the Czerny-Lembert suture, except that it applies the Czerny and the Lembert with one suture, and this suture does not pass through the mucous membrane (Fig. 638). In *Connell's suture* (F. Gregory Connell, in "Phila. Med. Jour.," Jan., 1899) the knots are placed within the lumen of the bowel (Plate 11). Connell's very useful and ingenious stitch seems to be a modification of a stitch described by Frederick Holme Wiggin ("Med. Record," Nov. 19, 1898). *Wölfler's suture* unites broad layers of the serous coat, the knots being tied internally (Fig. 639). Senn says that after suturing a large wound of the stom-

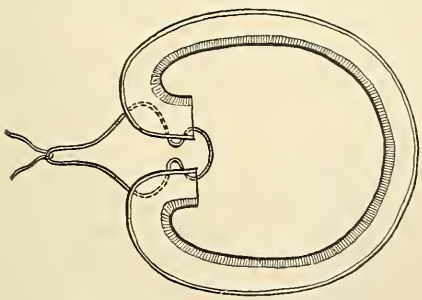


Fig. 638.—Gussenbauer's suture.

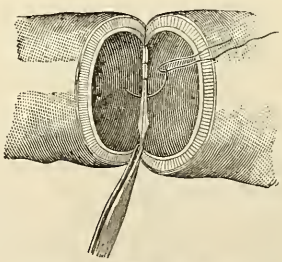


Fig. 639.—Wölfler's suture.

ach or of the intestine a strip of omentum ought to be laid over the wound and fastened by catgut sutures (*omental graft*). These grafts adhere and are a safeguard against leakage. (For other methods of enterorrhaphy see Intestinal Resection and Anastomosis.)

**Operations Upon the Stomach.**—A patient must be carefully prepared for an operation upon the stomach. The Johns Hopkins method, founded on the researches of Harvey Cushing regarding sterilization of the stomach, is to be used. During the two or three days immediately preceding operation clean the mouth and teeth several times during the day with a carbolic solution. Give only sterile water and sterile liquid food by the mouth,

and for twelve hours before operation give no food whatever. During the two or three days before operation wash out the stomach with boiled water night and morning. I do not wash immediately before operation, as it sometimes leads to annoying vomiting and thus may interfere with anesthetization. After operation give no food whatever for thirty-six hours. Small quantities of hot water are allowed as soon as the patient recovers from ether. During the first twenty-four hours give an enema of hot salt solution and coffee every five hours and then alternate nutritive enemata with salt enemata. After thirty-six or forty-eight hours usually begin to give food by the mouth—at first small doses of albumin-water, and, if this is tolerated, broth and milk. Solid food should not be given for two weeks.

If the patient is advanced in emaciation and much exhausted we should not wait for thirty-six hours to feed him, but should give milk and broth as soon as the patient recovers from ether. The bowels should be moved by enema the day after the operation. If the enema fails, calomel is given (3 or 4 gr.).

**Digital Dilatation of Pylorus for Cicatrical Stenosis (Loreta's Operation, or Pylorodiosis).**—Place the patient recumbent and administer ether. Make a vertical incision in the linea alba or through the right rectus muscle. The median incision begins 1 inch below the ensiform cartilage. The cut in either case should be 5 inches in length. When the peritoneum has been opened the stomach is drawn out of the wound, any adherent omentum is separated, and the pylorus is carefully examined. The stomach, after being surrounded with gauze pads, is opened near the center of its anterior surface, "but rather nearer to its pyloric end" (Jacobson).

Insert the index-finger through the stomach wound and into the pylorus, and follow that with the middle finger. The pylorus can be well dilated by separating the fingers. If the stenosis is so tight as to prevent the entry of a finger, first introduce a pair of hemostatic forceps and open the blades a little when they are within the lumen of the constricted area. The wound in the stomach is closed by a continuous silk suture of the mucous membrane and two layers of Halsted sutures, to invert and approximate the peritoneal surfaces. After closure of the stomach wound the abdominal wound is sutured.

Divulsion by the fingers or by an instrument is no longer practised, because experience has shown that the constriction is sure to return.

**Pyloroplasty (Heineke-Mikulicz Operation).**—The first operation was performed by Heineke in 1886. Early in 1887 Mikulicz, not knowing of Heineke's antecedent operation, did the same thing. Open the abdomen in the middle line or, better, through the right rectus muscle. Draw up the pylorus as well as possible, and pack warm moist gauze pads around it; make an incision through the stricture and in a direction corresponding to the long axis of the stomach and bowel (Fig. 640). Catch an aneurysm-needle under the upper margin of the incision and draw it up, and an aneurysm-needle under the lower margin and draw it down. The effect of traction is to convert the longitudinal wound into a transverse wound. The sutures are applied so as to maintain the wound in a vertical line (Fig. 641). The mucous membrane is sutured with a continuous suture of silk, and interrupted Lembert or Halsted sutures of silk close the peritoneal and muscular coats (Figs. 641 and 642). Do not drain. A. W. Mayo Robson inserts a bone bobbin and then applies the sutures. The operation of pyloroplasty shows a mortality about the same as or slightly less than gastro-enterostomy. In some cases it is a very satisfactory procedure, but there are objections to it, and in 30 per cent. of cases it fails to give relief (Wm. J. Mayo). The outlet is not at the most dependent part of the stomach, hence the stomach may not empty itself. Further, as Finney points out, it cannot be performed if there are firm adhesions or active ulceration, and the scar may contract and give rise to stenosis. Again, it is

difficult to suture the wound so as certainly to provide against leakage. The Mayos reported 21 pyloroplasties without a death, but 7 cases required sec-

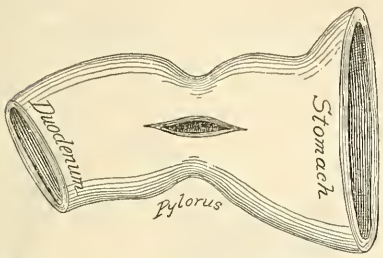



Fig. 640.—Heineke-Mikulicz's pyloroplasty: The incision.

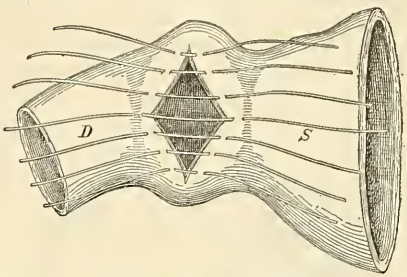



Fig. 641.—Heineke-Mikulicz's pyloroplasty: The axis of the incision is changed by traction from horizontal to vertical; sutures in position; only one of the two rows of sutures is shown.

ondary operations ("Annals of Surgery," Nov., 1905). Pyloroplasty has been abandoned by many surgeons; J. Rutherford Morison still advocates it. Finney has devised an operation to correct the objections to pyloroplasty.

**Gastroduodenostomy by Finney's Method.**—This operation is usually called a method of pyloroplasty, but it is rather a gastroduodenostomy. The operation was described in the "John's Hopkins Hospital Bulletin," July, 1902, and was then called pyloroplasty. It is performed as follows: Thoroughly free the first portion of the duodenum and the pyloric end of the stomach by dividing the posterior layer of peritoneum 1 inch to the right side of the duodenum. This step is known as mobilization. Insert three retractor sutures (Fig. 643) and draw upon them. Suture together, as far

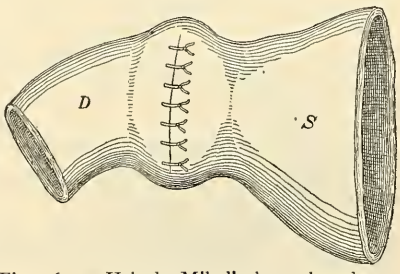



Fig. 642.—Heineke-Mikulicz's pyloroplasty: After tying the sutures.

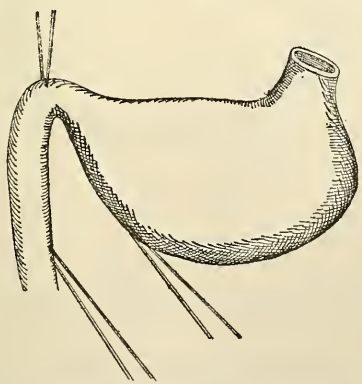


Fig. 643.—Finney's pyloroplasty: The retractor sutures.

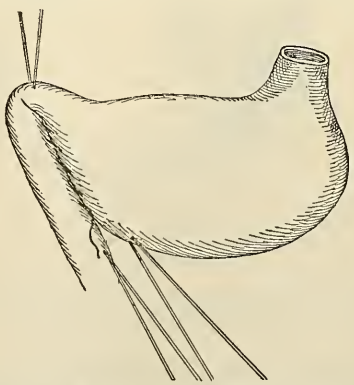


Fig. 644.—Finney's pyloroplasty: Suture of greater curvature of stomach to duodenum.

posterior as possible, the peritoneal surface of the duodenum and the peritoneal surface of the stomach along its greater curvature (Fig. 644). Then

insert an anterior row of mattress sutures, but do not tie them as yet (Fig. 645). Make a horseshoe-shaped incision (Fig. 646); arrest bleeding; excise as much scar-tissue as possible on either side of the incision, and trim off the redundant mucous membrane. Insert a continuous catgut suture on the posterior side of the incision and carry it through all the coats (Fig. 647). Straighten out

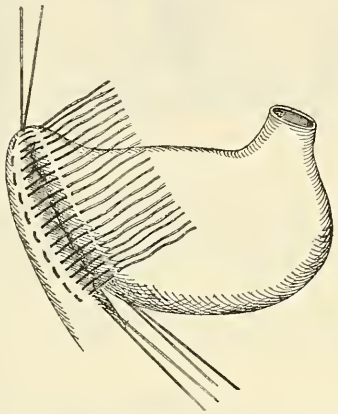


Fig. 645.—Finney's pyloroplasty: Shows the three retractor sutures, the posterior line of sutures tied and the anterior line of sutures untied.

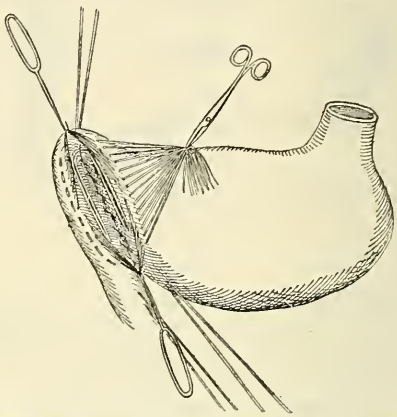


Fig. 646.—Finney's pyloroplasty: The anterior sutures gathered and lifted.

the anterior sutures and tie them (Fig. 648). The Mayos reported 58 Finney operations, with 4 deaths and 2 secondary operations (Wm. J. Mayo, in "Annals of Surgery," Nov., 1905).

The mortality is greater than after gastro-enterostomy, due probably to the necessity of separating adhesions and setting the duodenum free. The opera-

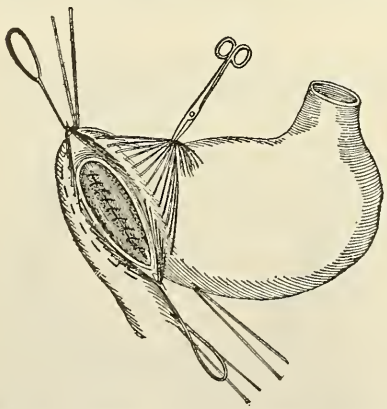


Fig. 647.—Finney's pyloroplasty: The continuous posterior catgut suture.

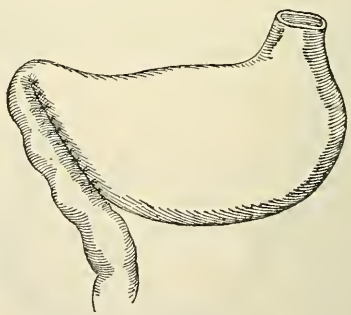


Fig. 648.—Finney's pyloroplasty completed by tying the anterior sutures.

tion should be restricted to cases in which adhesions are not widespread and firm and in which the gastrohepatic omentum is of fair length. In properly selected cases it is a very valuable operation.

**Pylorectomy (Excision of the Pylorus).**—The removal of a portion of the stomach is a partial gastrectomy, and pylorectomy is a partial gastrectomy in which the pylorus and also a portion of duodenum are removed.

The experiments of Gussenbauer and von Winiwarter on dogs in 1876 led them to suggest the operation. It was first performed by Péan in 1879. It was next performed by Rydygier in 1880. Billroth did the first successful pylorectomy in 1881. The operation is seldom performed for anything but cancer, but sometimes is done for pyloric ulcer and its results. In many cases of pyloric cancer the abdomen is opened only after a palpable tumor is detected, and when a palpable tumor is detectable it is usually too late to perform pylorectomy.[1] The lesson is to explore suspected cases earlier than has been our custom.

I agree with Hemmeter that stenotic symptoms, even when no tumor is palpable, call for exploratory laparotomy; if the stomach is dilated, if there is cachexia, if there is no free hydrochloric acid in the gastric juice, if there is an excess of lactic acid in the gastric juice, if the patient is at or beyond forty years of age, when there is vomiting of blood, when the Oppler bacillus is present, when blood examination shows a diminution in red corpuscles and hemoglobin, and also shows that there is no increase in white corpuscles after a full meal. After the abdomen has been opened the stomach is examined, and if a tumor exists, the surgeon must decide between the performance of pylorectomy and gastro-enterostomy. If the tumor is not very extensive, if there is no glandular involvement or only involvement which can be removed, and if adhesions are not extensive, pylorectomy is chosen; otherwise gastro-enterostomy is selected.

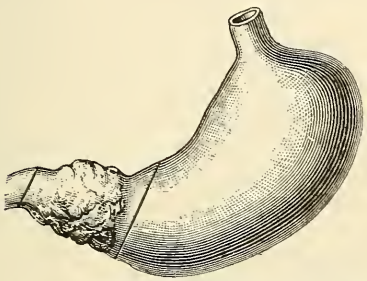


Fig. 649.—Billroth's method of pylorectomy.

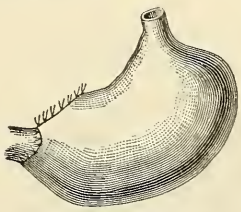


Fig. 650.—Pylorectomy.

Until very lately the mortality from pylorectomy was estimated to be 25 per cent., even in favorable cases. In 9 complete pylorectomies, with closure of both the stomach and duodenal ends, communication being reëstablished by the performance of gastrojejunostomy, Mayo reported 1 death, and in 14 pylorectomies and partial gastrectomies he reported 2 deaths, or 14 per cent. (Wm. J. Mayo, in "Annals of Surgery," Aug., 1902) (see page 1085). Prepare the patient for pylorectomy as for any stomach operation. The best incision through the abdominal wall is a vertical one in or near the median line. A small incision is first made to permit of exploration, and if the growth is found to be removable, the incision is enlarged. In some cases it will be found necessary to divide the rectus muscle by a transverse cut.

**Method of the Mayos.**—This is the best operation. The Billroth method, which was long employed, does not remove enough of the stomach if there is malignant disease, the opening left in the stomach is much larger than the duodenal opening, and in suturing so as to make the two openings of equal size an angle is left which is apt to leak. Billroth's operation is shown in Figs. 649 and 650. In the Mayo method, after the stomach has been exposed the gastric artery is ligated close to the stomach, the lesser omentum is tied in several segments close to the liver and then divided, and the pyloric artery is tied. Two clamps are applied to the duodenum 1 inch apart, and the duodenum is divided by means of the cautery (Fig. 651).

The right end of the duodenum is closed by means of a continuous catgut

[1] Keen's "Cartwright Lectures for 1898."

suture, the clamp is removed, and the closed end of the duodenum is inverted by a purse-string suture (Fig. 652). A hand is passed from above back of the stomach and lifts the great omentum forward. The right gastro-epiploic artery is tied close to the stomach. The left gastro-epiploic artery is tied distinctly to the left of any enlarged glands in the great omentum. The great omentum is tied in several segments. The great omentum is divided, leaving any enlarged glands attached to the portion of the stomach it is the intention to remove. The stomach is to be divided to the left of all lymphatic glands into which the cancerous region drains. The clamps are applied as shown in Fig. 651. The stomach is divided between the clamps by a cautery, and as the division is being carried out the stump is caught here and there by hemostatic forceps to prevent it slipping through the clamps. Slipping is disastrous and will cause leaking and entrance of air into the stomach, and entrance of air is apt to be followed by pulmonary difficulty. A row of locking stitches is passed through all the coats of the stump. The stitches are tied and a second row is passed and tied (Fig. 652). The clamp is removed and the stump is buried by Cushing's right-angled suture or Dupuytren's suture. A gastrojejunostomy is then performed to the posterior wall of the portion of stomach which remains.

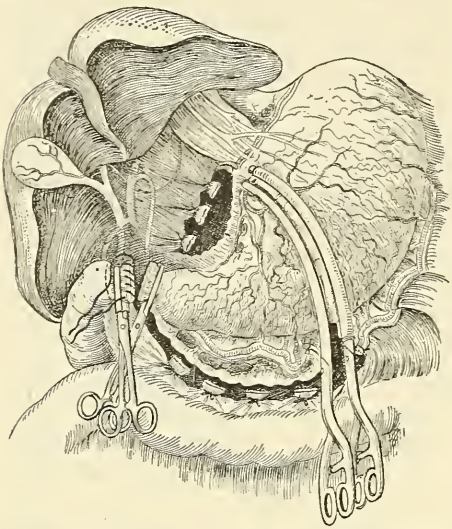


Fig. 651.—Pylorectomy by the Mayo method: Clamps applied, duodenum divided, and continuous catgut stitch introduced (Mayo).

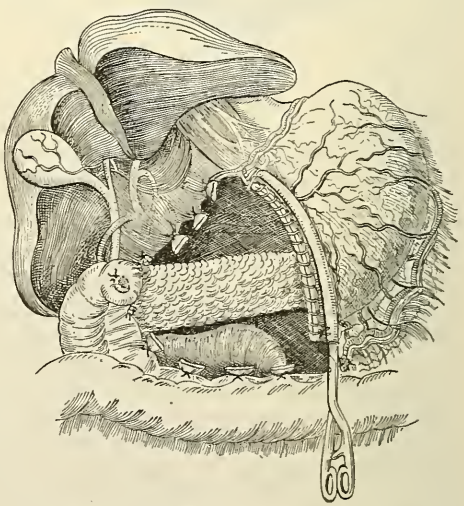


Fig. 652.—Pylorectomy by the Mayo method: End of divided duodenum buried by a purse-string suture. Row of lock stitches inserted in stomach stump (Mayo).

Such a patient is usually much dehydrated, and if he is, salt solution should be given intravenously during the operation, and an enema of warm salt solution should be administered every six hours for several days after the operation. Active stimulation is usually necessary and 8 oz. of coffee should be given by rectum at the completion of the operation. The patient must be placed erect or semi-erect in bed as soon as the effects of the ether pass away. Twelve hours after operation begin to give small amounts of hot water by the mouth. Nourish by the rectum from four to six days, when fluid food may be given by the mouth, starting with small doses of albumin-water, and,

if this is tolerated, giving dessertspoonful doses of peptonized milk every hour. During 1912 the Mayos did 46 pylorectomies for cancer, with 5 deaths ("Report of St. Mary's Hospital"). They did 13 for ulcers and benign tumors without a death (Ibid.). If during the operation the pancreas is wounded the closed end of the duodenum is applied directly to the pancreatic wound, as Willy Meyer suggested ("Trans. of Am. Surg. Assoc.," 1910). "The anterior peritoneum and adventitious sheath of the pancreas is then sutured to the anterior surface of the duodenum." This plan prevents leakage from the duodenum and pancreas (Wm. J. Mayo, in "Annals of Surgery," August, 1913).

**Total Gastrectomy.**—The entire stomach was first removed by Conner, of Cincinnati, in 1883. The first successful operation was performed by Schlatter, of Zürich, in 1897. Total gastrectomy will rarely be required, but in certain unusual cases it will be proper to perform it. In some cases the duodenal end can be sutured to the divided esophagus; in others it will be necessary to close the end of the divided first portion of the duodenum, and anastomose the esophagus to the jejunum.

The cases suitable for total gastrectomy are those in which the entire viscus, or almost the entire viscus, is cancerous, the stomach being still freely movable, and the glands not so much implicated as to forbid attempts at removal. It is a remarkable fact, first demonstrated in Schlatter's case, that an individual can digest food very well without a stomach. This statement is true only if the stomach function has been gradually abolished by disease. During this period the functions of the stomach have been assumed to a greater or less degree by other parts. In a recent injury of the stomach complete removal would almost certainly be followed by death, as in such a case other parts would have had no chance to learn how to assume gastric duties. The reported cases of total gastrectomy show 10 deaths out of 27 cases, but, as Robson truly says, if all cases were reported, the mortality would probably be found to be 50 per cent. Trinkler ("Archiv. für klin. Chir.," Berlin, xcvi, No. 2) reports 1 case (which died on the eighth day) and gathers 25 from literature; 13 recovered. When the duodenum was stitched to the esophagus the mortality was 57.1 per cent. When the jejunum was stitched to the esophagus the mortality was 28.5 per cent.

I have done the operation once. The patient died on the third day.

**Gastrotomy.**—This term is used to designate the operation of opening the stomach for the accomplishment of some purpose, and immediately closing the incision in the gastric wall when that purpose is accomplished. Gastrotomy may be performed to permit of the removal of foreign bodies, of exploration of the stomach and its extremities, of divulsion of the pyloric orifice, of the treatment of bleeding of an esophageal stricture or a stricture of the cardiac orifice of the stomach, or of the removal of a foreign body lodged in the esophagus. The first case on record was in 1602, when Florian Mathias, of Brandenburg, removed from the stomach of a juggler a knife which had been accidentally swallowed. When Evelyn was in Leyden in 1641 he saw a knife which had been removed by gastrotomy. He says (Evelyn's Diary): "I was showed the knife newly taken out of a drunken Dutchman's guts by an incision in his side, after it had slipped from his fingers into his stomach. The picture of the surgeon and his patient, both living, were there."

The patient is prepared as for pylorectomy. The incision may be vertical in the middle line or identical with the incision for pylorectomy. If a large foreign body can be felt, the incision is made directly over it. When the peritoneal cavity is opened, the surgeon decides as to the point where the stomach is to be incised, and draws this portion out through the wound, packing gauze pads under and around it. The stomach is opened by means

of scissors, the cut being at a right angle to the long axis of the viscus (Jacobson). Bleeding vessels are ligated with catgut. The purpose for which the stomach was opened is now to be carried out, the interior of the stomach and the surface of the extruded portion are irrigated with hot salt solution, the mucous membrane is sutured with a continuous suture of silk, and two rows of Halsted sutures are inserted. The abdominal wound is closed, drainage being employed for twenty-four hours.

**Gastrostomy** is the making of a permanent gastric fistula, through which opening the patient can be fed. Gastrostomy was first proposed by Egebert in 1837 and was first performed by Sédillot in 1849. In 1875 Sydney Jones operated upon the twenty-ninth case and obtained the first recovery (Keen). Up to 1884 the estimated mortality was 80 per cent. At present the mortality in malignant cases is from 20 to 25 per cent., and in non-malig-

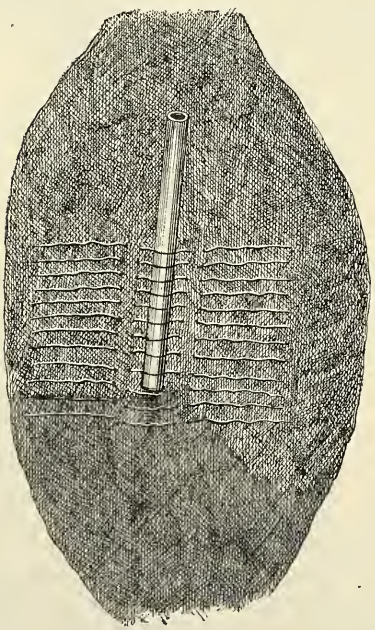


Fig. 653.—Witzel's method of gastrostomy, showing application of sutures in wall of stomach, embedding tube obliquely therein.

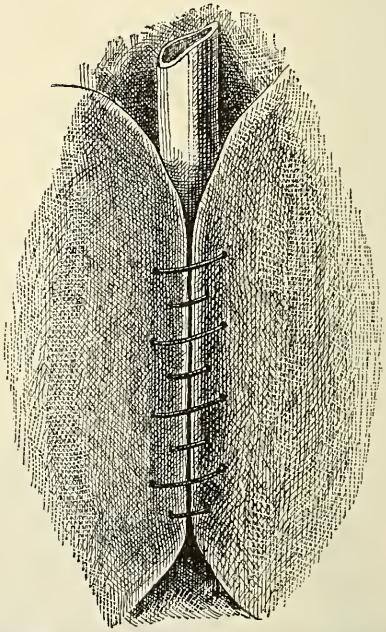


Fig. 654.—Sutures tied, completely embedding tube obliquely therein.

nant cases from 8 to 10 per cent. Gastrostomy is employed in cases of esophageal obstruction or obstruction of the cardiac end of the stomach. In many cases of malignant disease the operation is performed too late, and if performed when the patient is greatly emaciated and exhausted the operation has, of course, a high mortality. An early operation is far safer and confers the maximum of relief. The operation should be performed, as Mikulicz advises, when the patient is steadily losing weight and there is beginning to be difficulty in swallowing semisolids or liquids. The surgeon must endeavor to perform an operation which will not permit of leakage. Prepare the patient as for any stomach operation, except as to washing out the stomach, which is usually impossible.

*Witzel's Method.*—This operation was first practised in 1891. Make an incision 4 inches in length, running to the left from the middle line, just below the border of the ribs. After opening the peritoneal cavity seize the stomach.

bring it out of the wound, and pack gauze around it. Introduce a rubber tube into the stomach and enfold it by a double row of Lembert sutures (Figs. 653, 654). This tube should be 5 inches long and of the same diameter as a No. 25 French bougie. The opening is made in the stomach toward the

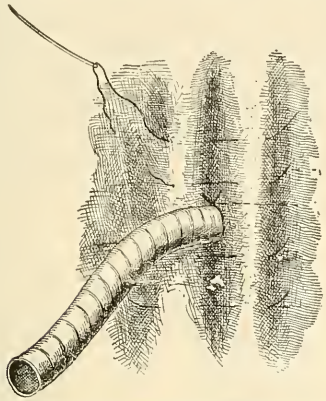


Fig. 655.—Kader's method of gastrostomy: Tube in place and first row of sutures inserted.

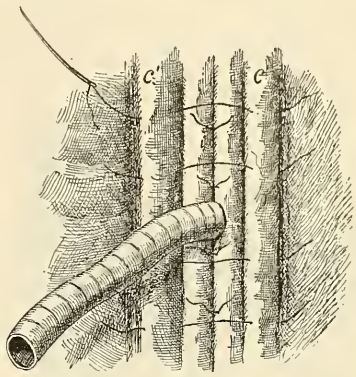



Fig. 656.—Kader's method of gastrostomy: First row of sutures tied and second row inserted.

cardiac extremity, the tube is placed parallel with the belly wound, and the outer end of the tube emerges in the median line. The tube is retained in place by a catgut stitch carried through the tube and the stomach wall. The stomach is returned and is stitched by three sutures to the abdominal wall. The abdominal incision is sutured and a clamp is placed on the tube. When the patient is fed, a funnel is slipped into the tube, the clamp is removed, and liquid food is poured into the funnel. After the wound heals it is not necessary to retain the tube permanently. It is passed when the patient desires food.

*Kader's Method.*—This operation was devised in 1896. It is a modification of Witzel's method. A small incision is made in the stomach and a tube is introduced and fastened to the stomach by one catgut stitch. Four Lembert sutures are passed so as to form a fold on each side of the tube and turn the stomach wall inward around the tube (Fig. 655). Lembert sutures are inserted in the furrow on each side of the tube. Two more folds are formed over the first two (Figs. 656 and 657). The stomach wall is stitched to the parietal peritoneum and sheath of the rectus muscle (Willy Meyer).

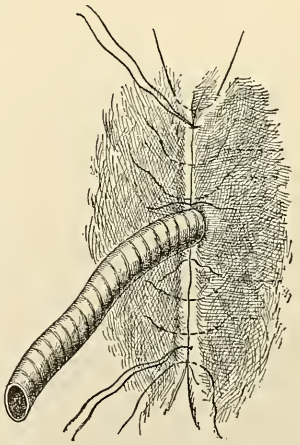


Fig. 657.—Kader's method of gastrostomy: Second row of sutures tied.

*The Ssabanajew-Frank Method.*—This operation is preferred by many surgeons. I usually employ it if the stomach is not so shrunken as to render the pulling out of a sufficient cone impossible. It was first performed by Ssabanajew in 1890 and was performed independently by Frank in 1893. Fenger's incision is made (a curved incision at the margin of the costal cartilages of the left side). A cone of the stomach is pulled out of the wound and is passed under a bridge of skin which has been prepared for it. The stomach is fixed above the margin of the ribs and opened (Figs. 658, 659).

Von Hacker makes the gastric fistula through the left rectus muscles, and Hahn between two of the rib cartilages (Willy Meyer).

*The Younger Senn's Method.*—Emanuel Senn devised the following method: A cone of the stomach is pulled out of the abdominal wound, and this cone is puckered by the insertion of two drawing-string sutures of chromicized catgut. A cuff of gastrocolic omentum is sutured by silk around the neck of the puckered cone. The stomach is sutured to the belly wall with silk, the sutures including the omental cuff, the serous and muscular coats of the stomach, and the structures of the belly wall, except the skin. The skin is partly sutured. The stomach may be opened at any time.

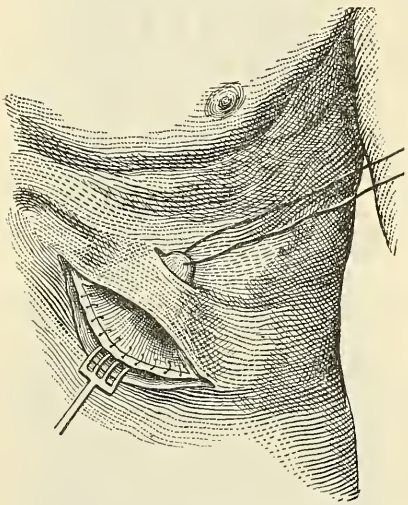


Fig. 658.

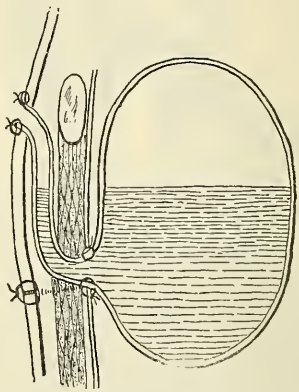


Fig. 659.

Figs. 658, 659.—The Ssabanajew-Frank method of gastrostomy in carcinoma of the esophagus.

**Gastro-enterostomy** or **gastrojejunostomy** is the establishment of a permanent fistula between the stomach and the small intestine, in order to side-track the pylorus. The operation is performed for cancer of the pylorus, for non-cancerous stenosis of the pylorus, in some cases of ulcer of the stomach, and for tetany. Anterior gastro-enterostomy was proposed by Nicoladoni in 1881 and was first performed by Wölfler the same year. In Wölfler's early operations the jejunum was so placed that the proximal end was to the right of the anastomosis opening. Hence peristalsis in the stomach was from left to right and in the jejunum from right to left, and this was supposed to be responsible for the common occurrence of regurgitant vomiting. It was sought to prevent this by altering the direction of the loop (Lücke), then by entero-anastomosis (Braun), and finally by posterior anastomosis. Posterior gastro-enterostomy was first proposed by Courvoisier in 1883. His suggestion was that the posterior surface of the stomach be reached through the transverse mesocolon. His plan necessitated a transverse division of the mesocolon, but it was found that this impaired the blood-supply of a part of the colon and might lead to gangrene. Von Hacker, in 1885, devised an improved posterior operation. As a matter of fact, the transverse mesocolon has a marginal artery, unlike other parts of the colon, and the danger of gangrene from a transverse incision is probably not very great. In the earlier operations by the posterior method a long loop was used and results were not notably better than after the anterior operation. In 1890 Czerny and Peterson advised the making of the jejunal opening close to the duodenojejunal flexure. The results from this operation are vastly better than from the

anterior operation. Posterior gastro-enterostomy has been signally improved in technic by the Mayos, Moynihan, and others. In the earlier operations of anterior gastro-enterostomy the mortality was 40 per cent. In non-malignant conditions the mortality after gastro-enterostomy is now very low (under 3 per cent.), the hyperacidity of the gastric juice disappears, and the functions of the stomach are restored. In malignant cases the mortality is about 20 per cent., but even in such cases, if operation is done early, life may be prolonged and made comfortable for months. Wm. J. Mayo makes the following report upon 421 cases of gastrojejunostomy: "Benign, 307 cases, 19 deaths (6.18 per cent.). In the last 140 there were 4 deaths, a mortality of 2.85 per cent.; the last 80 gave but 1 death. Malignant, 114 cases, with 21 deaths (18.5 per cent.). Of these 114 cases, 63 were in connection with pylorectomy and partial gastrectomy, with 8 deaths (12.6 per cent.). The very unfavorable cases of cancer obstruction were subjected to gastro-enterostomy, so that this operation gives a higher mortality than radical excision. In the last 40 gastrojejunostomies for malignant disease the mortality was 8 per cent. In the 421 gastrojejunostomies there were 21 reoperated cases (5 per cent.)" ("Annals of Surgery," Nov., 1905). During 1912 the Mayos did 40 gastro-enterostomies for chronic ulcer of the stomach, with 2 deaths, and 31 for cancer of the pylorus, with 2 deaths. They did the operation 187 times for chronic and subacute ulcer of the duodenum with 1 death ("Report of St. Mary's Hospital for 1912"). In about 5 per cent. of cases of gastro-enterostomy for benign disease secondary operation has been required. In Krönlein's clinic, 51 cases of malignant disease subjected to gastro-enterostomy showed an average duration of life of 192 days; 470 days after operation 17 cases were living. The causes of death, according to Wm. J. Mayo, are: exhaustion, exhaustion with vomiting, pneumonia, and detachment of the anastomosed intestine.

**Treatment After Gastro-enterostomy.**—On returning the patient to bed at once establish continuous proctoclysis with one-half strength salt solution, the reservoir being only 6 inches above the level of the bed. As soon as the patient is out of ether place him semi-erect. Mayo begins in from sixteen to twenty hours to administer by the mouth 1 oz. of hot water every hour, and if it is well tolerated the amount is quickly increased, and in thirty-six hours liquid food is given, and if tolerated, is continued.

**Complications Following Gastro-enterostomy.**—Among them are *lung complications*. These are not due to the anesthetic, for they tend to occur even when local anesthesia has been employed. They are not due to the epigastric incision interfering with cough and expectoration, for they are not nearly so common after operations upon the gall-bladder (Wm. J. Mayo). Mayo says that the latest theory is that some of the venous blood returning from the stomach does not pass through the liver, and infected emboli are deposited in the lungs. The *suture line* may *leak* after gastro-enterostomy because of imperfect suturing, or the anastomosed intestine may become *detached;* 20 per cent. of the deaths among Mayo's cases resulted from this cause. *Contraction* of the *anastomosis opening* may gradually take place. This has been held by some to be particularly common in cases of dilated stomach, shrinking of the stomach being the efficient cause, but evidence upon this point is not conclusive. In cases in which the pylorus is not obstructed shrinking often occurs, but it rarely takes place when the pylorus is obstructed. In some cases after operation a *spur* forms in the jejunum because of angulation; in other cases adhesions produce obstruction; and in rare instances ulceration takes place in the jejunum. The most common complication after gastro-enterostomy is *persistent vomiting*, which may or may not be expressive of the formation of a vicious circle.

*Peptic Ulcer of the Jejunum.*—The first case was reported by Braun in 1899.

The first English case was reported by Mayo Robson in 1903. Herbert J. Paterson reported a case and collected reports of 61 other cases (A. W. Mayo Robson, "Brit. Med. Jour.," Jan. 6, 1912). F. Gregory Connell has collected 38 cases and reported 1 of his own, 39 in all ("Surg., Gynec., and Obstet.," Jan., 1908). He points out that in many of the reported cases acute perforation occurred. Most of the reported cases suffered from non-malignant trouble and had hyperacid gastric juice. It very seldom occurs after operations for cancer. Most of the reported cases happened after the anterior operation and when the anastomosis was very near to the pylorus. It has happened, however, in 9 cases after the posterior operation, and cases have been reported following both the anterior and posterior methods associated with entero-anastomosis. It is probable that more cases seem to follow the anterior method because until late years it has been the operation commonly performed. In most of the reported cases the ulcer was single; in 3 out of 24 cases it was multiple. It is usually in the distal loop, but may be in the proximal loop. It may be situated at the anastomosis level, a little way below it, or even 5 or 6 inches below it. The ulcer may appear a few days after the operation, weeks after, months after, or even years after. The condition results from hyperacid gastric juice passing directly into the jejunum before it has been neutralized by admixture with bile and pancreatic juice. It is possible that the condition is predisposed to by a twist in the jejunum and by such a small anastomosis opening that hyperacidity was not corrected.

There may be no symptoms at all until there is a severe hemorrhage or perforation, or symptoms similar to those which called for the gastro-enterostomy may return. Connell's table shows that acute perforation took place in 14 of the 39 reported cases. Pain is to the left of the umbilicus, comes on two or three hours after eating, and is relieved by food. There may be tenderness and may be rigidity of left rectus muscle (Robson, Loc. cit.). In chronic cases treatment is first medical; if this fails, operation is indicated. If on opening the abdomen it is found that the original pyloric or duodenal ulcer has healed, but there is an ulcer in the jejunum, separate the bowel from the stomach, close the stomach opening, excise the jejunal ulcer, and close the wound in the bowel. If there is stenosis of the pylorus or duodenum we must have a gastro-enterostomy, so we make another after closing the original opening (Robson). Very extensive ulceration may call for resection. If the patient is greatly weakened or if the ulceration is extensive, jejunostomy (see page 1105) may be done (Robson). In perforation, operation must be immediate.

*The Vicious Circle and Regurgitation.*—Vomiting may occur after the performance of gastro-enterostomy. It may soon cease, may be productive of disastrous consequences, and may be expressive of an existing complication of great gravity. In some cases of gastro-enterostomy vomiting arises because the anastomosis has been made high up on the anterior gastric wall and the stomach is not drained. In other cases ether induces vomiting, and the mechanical efforts force the contents of the duodenum and even of the jejunum into the stomach. The true "vicious circle" is a condition in which the contents of the stomach pass through the anastomosis opening into the duodenal side of the loop of intestine, mix with the duodenal secretions, and return to the stomach (Fowler, in "Annals of Surgery," Nov., 1902). The following conditions are often classified under the same head, but each is called by Fowler a regurgitation or reflex: (1) When the duodenal secretions pass back into the stomach through a permeable pylorus (as in cases of gastroptosis, non-cancerous pyloric stenosis, and gastric dilatation); (2) when the duodenal secretions enter the stomach through the anastomosis opening; (3) when the contents of the jejunum pass into the stomach, because of efforts at vomiting or as a result of reversed peristalsis. In some cases the contents of the jeju-

num may pass into the afferent loop of intestine and distend it. It was long thought that the vicious circle was due purely to bile passing into the stomach from the proximal (afferent) loop of the jejunum. Dastre's experiments on dogs show that bile in the stomach does not impair either digestive power or the general health, and Moynihan has confirmed these experiments by clinical observation. It has been held that the cause of this condition is the making of an anastomosis with a long loop between the duodenojejunal flexure and the anastomotic opening into the jejunum, the loop being unable to propel its contents downward and tending by its weight to produce a bend or kink at the seat of anastomosis. This is a very doubtful theory.

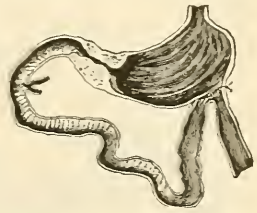


Fig. 660.—Billroth's method of gastro-enterostomy.

Persistent vomiting may be due to spur formation, which deviates stomach contents into the duodenal side of the loop. It is in some cases due to kinking or twisting of the distal loop; in others, to failure of peristalsis in the proximal loop; in still others, to contraction of the opening in the stomach wall (Chlumsky on "Gastro-enterostomy" in the Breslau Clinic; article by Charles L. Gibson, in "Annals of Surgery," Aug., 1908). I cordially agree with the statement of Herbert J. Paterson, viz.: "Most, if not all, fatal cases of regurgitant vomiting are due to mechanical obstruction at the afferent opening" ("The Hunterian Lectures," delivered before the Royal College of Surgeons of England, Feb. 19, 21, and 23, 1906). In order to lessen the danger of vomiting after gastro-enterostomy, do the operation under a local anesthetic whenever possible; and in order to prevent regurgitant vomiting take every care in the operation to prevent the formation of a spur on the mesenteric border of the jejunum (Herbert J. Paterson, Ibid.).

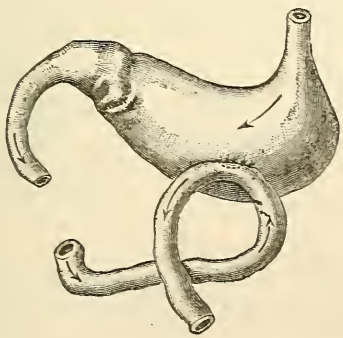


Fig. 661.—Gastro-enterostomy (after Lücke).

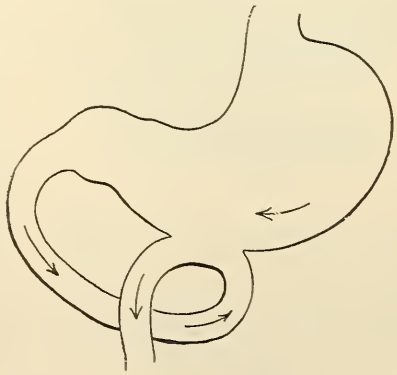


Fig. 662.—Wölfler-Lücke method of gastro-enterostomy.

After Billroth's operation (Fig. 660) and in all the earlier methods the contents of the duodenum certainly pass into the stomach, mix with the stomach contents, and usually, but not always, pass into the efferent loop. In all these operations there is great danger of the development of a vicious circle.

Lücke devised an operation with the idea of preventing such a complication. In the Lücke operation the direction of peristalsis in the efferent loop is the same as in the stomach (Fig. 662). McGraw points out that the crossing of the loop which is effected is dangerous. The Wölfler-Lücke operation is shown in Fig. 661. Wölfler also devised the operation pictured in Fig. 663. Von Hacker's posterior operation is thought by some to be less apt

than the anterior method to be followed by the vicious circle (Fig. 664). Kocher devised an operation in which a valve is formed, but, as Fowler points out, this valve does not prevent filling of the duodenum and imbibition of the material by the stomach; and, further, that the valve does not work when the parts become cicatricial (see Fig. 668).

The combination of gastro-enterostomy with entero-anastomosis does tend to prevent the vicious circle. This operation is shown in Figs. 665 and 666.

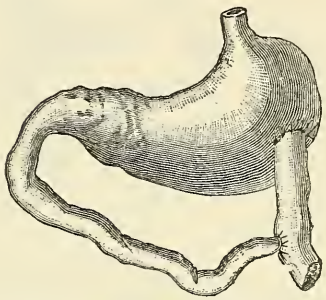


Fig. 663.—Implantation of duodenum into jejunum and jejunum into stomach (after Wölfler).

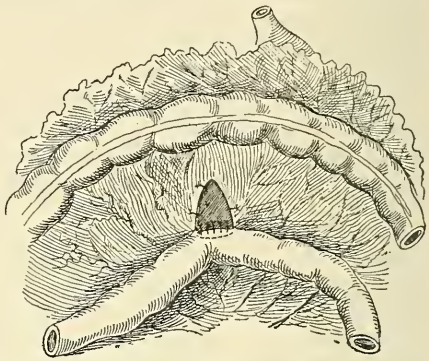


Fig. 664.—Von Hacker's posterior gastro-enterostomy.

I do not believe it should ever be a primary operation. It permits acid gastric juice to flow directly into the jejunum and keeps away the bile which would normally protect mucous membrane. Hence such an operation exposes the patient to the danger of jejunal ulceration. Another defect in such an operation is that there is still a communication between the stomach and the efferent loop. Fowler's operation (see Fig. 672) closes the jejunum and corrects the defect inherent in Braun's and in Jaboulay's operation. Other operators close

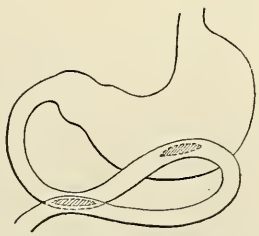


Fig. 665.—Jaboulay's method of gastro-enterostomy.

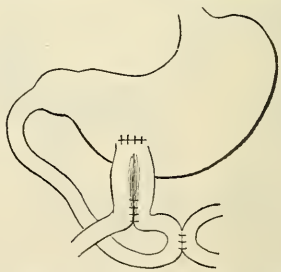


Fig. 666.—Braun's method of gastro-enterostomy.

the pylorus. McGraw's operation (see Figs. 669 and 670, which show entero-anastomosis) tends to prevent the formation of a vicious circle. It seems certain that the danger of the formation of a vicious circle is greatest after a long-loop anterior operation and least after a short-loop posterior operation. The shorter the loop, the less the danger, hence the latter is the operation of choice. The safest operation of all is the short-loop operation of Moynihan or Scudder (see page 1099) or the "no-loop" operation of the Mayos (see page 1101).

*Treatment of Persistent Vomiting After Gastro-enterostomy.*—If vomiting persists in spite of gastric lavage and rectal feeding following the operation of gastro-enterostomy with a long loop without entero-anastomosis, open the

abdomen again and perform anastomosis between the afferent and efferent loops of intestine. This was suggested by Braun in 1892, and both he and Jaboulay performed it in the same year. The operation has saved lives. In a short-loop operation we should assume that the jejunum has been twisted, should open the abdomen, and endeavor to correct the condition. Herbert L. Paterson ("Hunterian Lectures," before the Royal College of Surgeons of England, Feb. 19, 21, 22, 1906) points out that slight cases of regurgitant vomiting not immediately following an operation may be due purely to constipation, and may be recovered from if care is taken to secure daily a free bowel movement. In Paterson's opinion constipation causes reversed peristalsis, and as both the duodenal "siphon-trap and the pyloric sphincter are put out of service," regurgitation takes place from the efferent loop into the stomach.

**Anterior Gastro-enterostomy.** — *Senn's Method.* — A median incision is made through the abdominal wall, from below the xiphoid cartilage to the umbilicus. An opening is made in the lower part of the anterior wall of the stomach in the direction of the long axis of the viscus, and its edges are stitched by a continuous catgut suture. The contents of the jejunum are forced along to below the point where an incision is to be made. The duodenal loop of jejunum should be from 12 to 14 inches in length. A rubber tube is fastened around the bowel above this point, and another below it; an incision is made in the long axis of the bowel, and the margins of the wound are sutured in the same manner as the stomach wound. Bone plates are introduced into the stomach and intestine, and the ligatures are tied as in intestinal anastomosis. Catgut rings or rubber rings may be used.

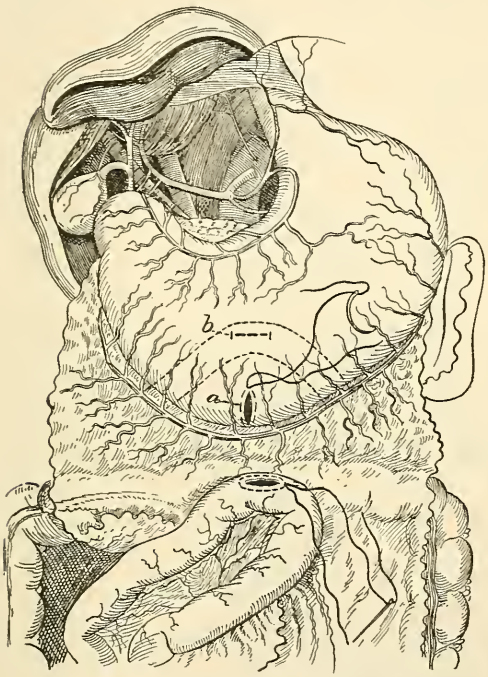



Fig. 667.—Mayo's method of anterior gastro-enterostomy, showing proper and improper locations of openings: *a*, Proper position, leaving no pouch; *b*, usual position, forming intragastric pouch ("Annals of Surgery").

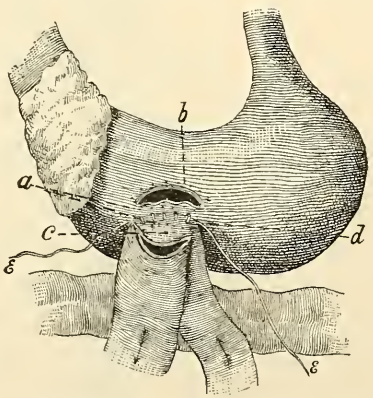



Fig. 668.—Kocher's method of gastro-enterostomy: *a*, Places of posterior annular suture through entire wall of stomach and intestine; *b*, places of anterior annular suture through the entire wall; *c*, valve at the jejunum by arch-formed incision; *d*, posterior annular suture of the serosa; *e*, thread ends for continuing anterior suture of the serosa.

*Mayo's Anterior Method* (Fig. 667).—Open the abdomen, and pick up the small intestine and find a point of jejunum about 14 inches from the point at which it emerges from under the mesocolon. Effect the union to the inferior

border of the stomach close to the greater curvature and at the lowest portion of the stomach pouch. When the anastomosis is completed the stomach pouch is funnel shaped. The usual custom has been to place the opening higher on the anterior wall. It sometimes led to the formation of a pouch on the anterior wall, did not drain the stomach, and caused vomiting. After the performance of gastro-enterostomy the edges of the omentum are caught upon each side of the anastomosis and are sutured to each other and to the stomach wall 1 inch above the opening. The edges are then united to each other in a downward direction for about 3 inches so as to form an apron over the anastomosis, yet not connected with it. Catgut is used for suturing. If leakage occurs, the omentum is adjacent and "available." If it does not occur, the omentum soon returns to its normal position (Wm. J. Mayo, "Annals of Surg.," Aug., 1902).

*Kocher's Method* (Fig. 668).—After opening the abdomen lift up the omentum, pull up a loop of intestine, and find the point where the jejunum

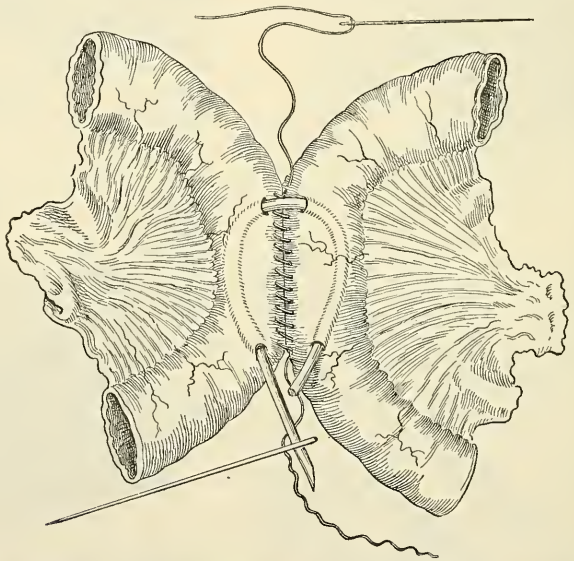


Fig. 669.—McGraw's method of lateral anastomosis: The elastic ligature is introduced (Walker). Gastro-enterostomy is done by the same plan.

appears from under the mesocolon. Select a loop 16 inches from the origin of the jejunum and prepare to attach it to the stomach. Wölfler believed that the intestine should be applied to the stomach in such a manner that the direction of peristalsis in the bowel would correspond to the direction of the stomach-tide. This can be accomplished by having the proximal portion of gut to the left, and the distal portion to the right. The operation is to be so performed that after its completion the stomach contents pass into the distal portion of the gut, and intestinal contents do not tend to enter the stomach (see Fig. 662). In order to accomplish this Kocher hangs the intestine to the stomach wall in such a manner that the proximal portion of the loop is posterior and ascending, and the distal portion is anterior and descending. The bowel is hung to the stomach by a continuous serous suture of silk, the ends of which are left long. The intestine is opened by a curved incision, the convexity of which is downward. The stomach is opened so that the convexity of the cut is upward. The valve-like portion of the bowel wall is sutured to the stomach below the incision in that viscus. The two openings are well approximated by sutures.

**Operation by McGraw's Elastic Ligature** (Figs. 669–671).—The elastic ligature was introduced by Silvestri in 1862, and was first used in intestinal anastomosis by the same surgeon. McGraw perfected the operation in 1891.

Fig. 670.—McGraw's method of lateral anastomosis: One tie of the elastic ligature with a strong silk ligature underneath ready to fasten the elastic ligature where it is drawn taut (Walker).

(See Dudley Tait, in "Annals of Surgery," Feb., 1906.) The operation may be anterior or posterior. The intestine and stomach are sutured together by

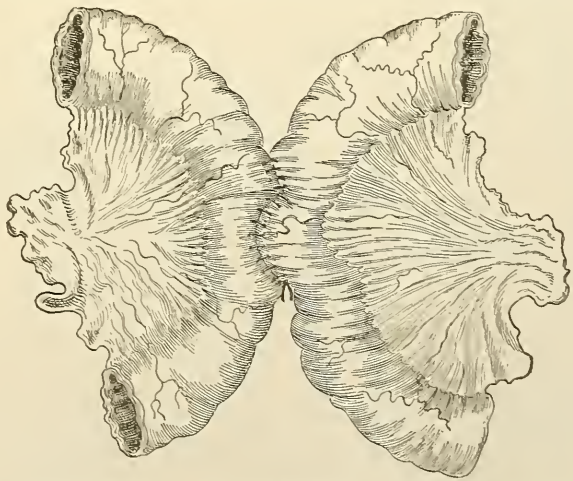


Fig. 671.—McGraw's method of lateral anastomosis: The operation completed (Walker).

Lembert stitches. The elastic cord, which is 3 to 5 mm. in diameter, is passed through the stomach and then the bowel, in the long axis of each, and is tightly tied, and the knot is fastened with a silk thread. Another row of Lembert

sutures buries the elastic cord from sight. The cord cuts through in from forty-eight to seventy-two hours and makes the anastomosis. Thus the danger of infection is greatly lessened, for when the anastomosis opening is formed it is completely encompassed by firm adhesions. Further, the danger of the formation of a vicious circle is greatly lessened, because there is no communication between the stomach and bowel for between forty-eight and seventy-two hours, the period in which vomiting of the type previously described is most apt to occur. The method is not suitable for absolute pyloric occlusion. In this condition it is imperative to give nourishment early, and, again, an ordinary gastro-enterostomy allays auto-intoxication and this operation cannot until the ligature cuts through. It is particularly valuable in the performance of lateral intestinal anastomosis. The cuts show the operation of lateral anastomosis of intestine, but gastro-enterostomy is performed in the same manner.

**Jaboulay's Gastroduodenostomy.**—This operation was devised by Jaboulay in 1892. It aims to obviate some of the objections to pyloroplasty and at the same time to retain the advantages this operation possesses over gastro-jejunostomy. Jaboulay's gastroduodenostomy has never become popular with surgeons, and Finney's method is much more satisfactory (see page 1081).

**Posterior Gastro-enterostomy** (see page 1088 and Fig. 664).—In a thin subject with a long mesocolon posterior gastro-enterostomy is to be chosen, but if the mesentery is short or contains much fat, or if the vascular loop coming from the superior mesenteric artery, and which supplies the transverse colon with blood, is small, so that on opening the posterior layer of the gastrocolic omentum it would be close to the artery, the anterior operation is employed (Wm. J. Mayo, in "Annals of Surgery," Aug., 1902). If a Murphy button is used, the posterior operation is selected. Posterior gastro-enterostomy is commonly performed as follows: After the abdomen has been opened the stomach and omentum are raised; a portion of the upper jejunum is seized, emptied, and a site selected for the clamp. This site must be within 5 inches of the flexure. If there is a broad mesocolic band preventing a near approach to the flexure the band must be divided. A clamp is applied on the side opposite the mesenteric attachment. A spot is selected on the transverse mesocolon where there are no vessels, and an opening is made through the mesocolon with a blunt instrument. The posterior wall of the stomach is pulled into the opening and sutured to its edges. This prevents downward displacement of the stomach and obstruction of the loop of gut. A portion of the posterior wall of the stomach is pulled out into a cone and clamped. Openings are made and the sutures applied as directed on page 1088. Regurgitation is less common after posterior than after anterior gastro-enterostomy. In 250 posterior operations in Czerny's clinic there was not one case of regurgitant vomiting; 170 cases were button operations and 45 were by sutures alone (Peterson). Von Hacker had one instance of regurgitation in 60 posterior operations.

**Operation by the Murphy Button.**—Gastro-enterostomy may be quickly performed by the use of a large-sized Murphy button. Murphy says that in some reported cases the button has slipped back into the stomach, but this accident can be prevented by the use of an oblong button and by making the anastomosis on the posterior stomach wall. The same surgeon advises us to scarify the peritoneum in order to hasten union, and says supporting sutures about the button are not required, except when considerable tension exists. There is no question that an anastomosis on the anterior wall, accomplished by a Murphy button, can be speedily performed. Anastomosis on the posterior wall cannot be performed so speedily, and it sacrifices to some extent the great advantage of the button operation—that is, speed. In spite of the reported cases we can positively assert that the danger of the button producing grave trouble is slight. In some cases it drops into the stomach and remains there, but seems to do no

harm. In other cases it takes a long time to pass. In 1 of the author's cases it did not pass until the eighty-sixth day. In one of Keen's cases it has been retained for years. If it does not pass in four weeks, the rectum should be explored by the finger from time to time to see if it is lodged in that region. The *x*-rays will determine whether the button is in transit. If the wall of the stomach is thick, the incision should be made in the stomach wall before the suture is passed, and this suture should pick up only a small portion of the stomach wall, otherwise the button may be retained in place for a very long time (Wm. J. Mayo). "In many cases in which the button passes, vomiting with symptoms of obstruction may appear during the second or third week while it is in transit. Gastric lavage and rectal feeding for a day or two cause these symptoms to subside" (Wm. J. Mayo, in "Annals of Surgery," Aug., 1902). Mayo long ago maintained that the suture operation is as good as the button operation, and that the results are about the same. Mikulicz says that in the suture operation entero-anastomosis is necessary, but not in the button operation, because the button, while in place, prevents angulation. The last-named surgeon uses the button in malignant cases and the suture in benign cases. Czerny is an advocate of the button. Every button should be tested before it is used. Mayo finds nearly 20 per cent. of buttons imperfect and dangerous.

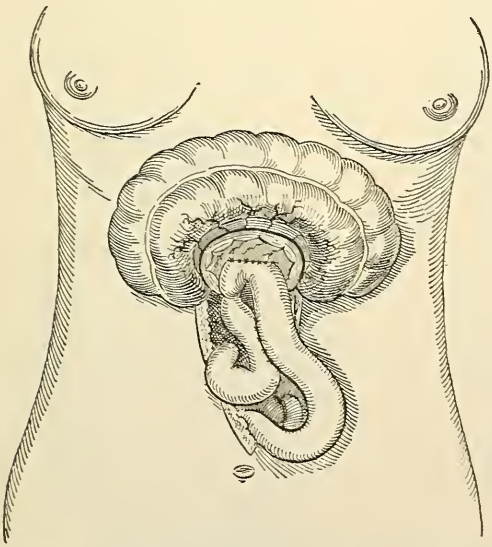


Fig. 672.—Fowler's method of gastro-enterostomy.

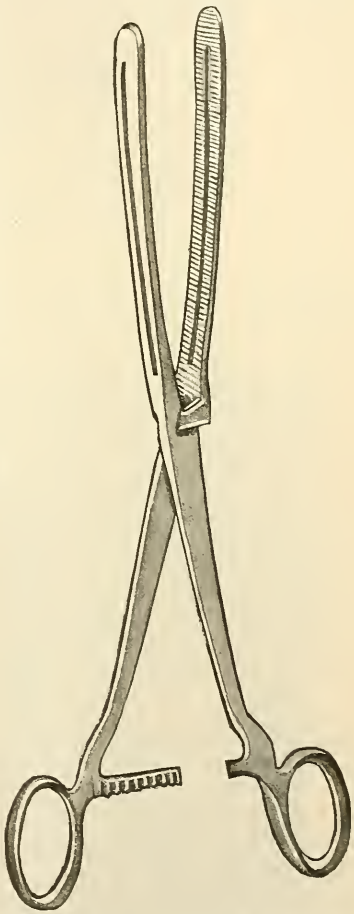


Fig. 673.—Moynihan's clamp for gastric and intestinal operations (made by Down Brothers, London).

**Fowler's Method** (Fig. 672).—Anastomose the posterior wall of the stomach to the jejunum and do an entero-anastomosis between the afferent and efferent loops of jejunum. Pass a No. 20 silver wire two or three times around the afferent loop of jejunum and draw it sufficiently tight to occlude the lumen without strangulating the wall of the gut. The ends are twisted, cut short,

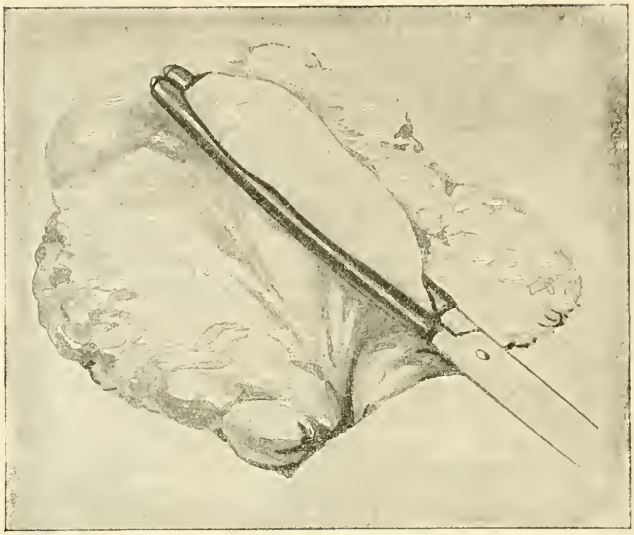


Fig. 674.—Moynihan's method of gastro-enterostomy: The oblique application of the clamp to the stomach (Moynihan).

rolled into a flat coil, the cut ends being in the coil. (See Geo. Ryerson Fowler on the "Circulus Vitiosus" following gastro-enterostomy, "Annals of Surgery,"

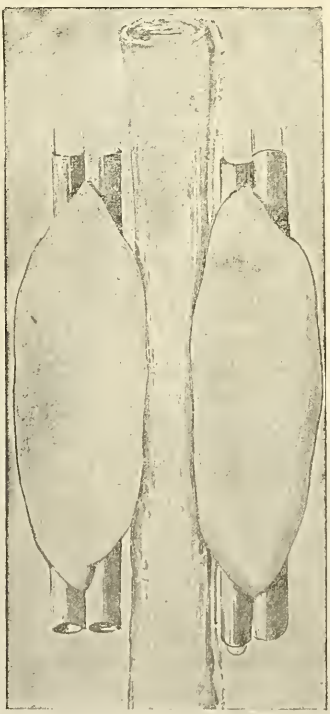


Fig. 675.—Moynihan's method of gastro-enterostomy: The strip of gauze between the clamps (Moynihan).

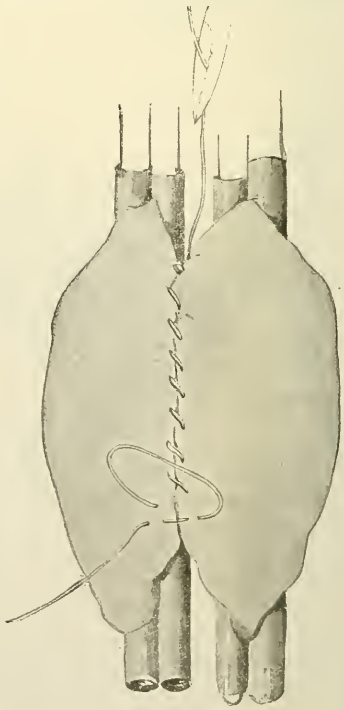


Fig. 676.—Moynihan's method of gastro-enterostomy: The first layer of serous suture (Moynihan).

Nov., 1902). This operation positively prevents the entrance of material from the duodenal loop into the stomach and also drains that loop.

**Moynihan's Method.**—This plan I have employed repeatedly. It is easy, rapid, and clean: Make a 4-inch incision 1 inch to the right of the middle line and above the umbilicus. Open the anterior sheath of the rectus and separate it from the front of the muscle as far as the middle line. Draw the entire muscle outward, open the posterior portion of the sheath, and then open the belly. Inspect and feel the entire stomach. Lift the omentum and transverse colon out of the abdomen and make the mesocolon taut by raising the stomach and colon with the left hand. Find "a bloodless spot in the arch of the middle colic artery," pick up a bit of the under surface of the mesocolon by a pair of hemostatic forceps, lift it from the posterior stomach

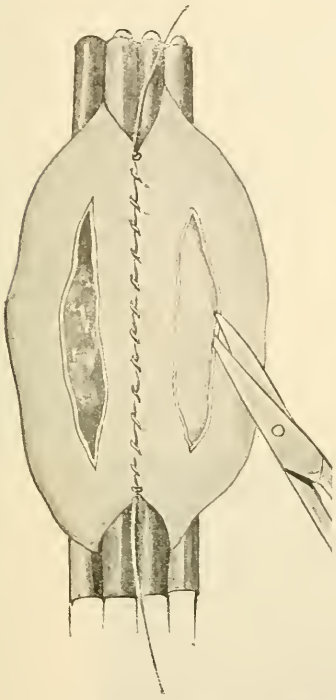


Fig. 677.—Moynihan's method of gastro-enterostomy: Removal of the ellipse of mucous membrane (Moynihan).

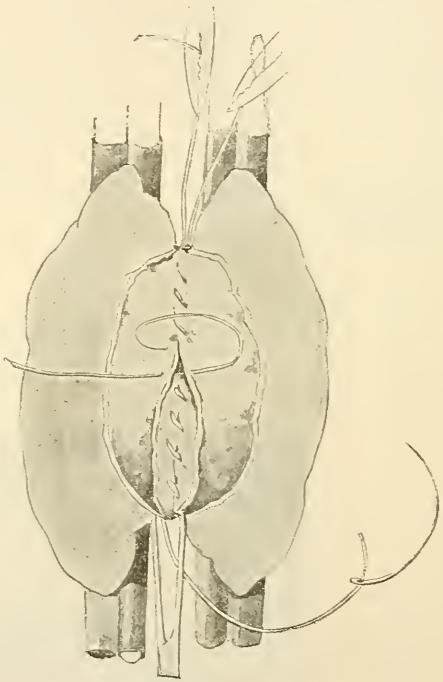


Fig. 678.—Moynihan's method of gastro-enterostomy: The inner suture continued (Moynihan).

wall, and open the lesser sac of peritoneum by the scissors. Enlarge the opening by dilatation or tearing until it admits three fingers. Inspect and feel the posterior stomach wall. Place the stomach in its natural position, mark with the thumb the lowest part of the posterior stomach wall, and again turn the viscus over. From the spot marked by the thumb a fold is raised. The fold is oblique and its upper end is to approach the cardia and lesser curvature. A stomach clamp (Fig. 673) having a rubber tube bent over each blade is applied obliquely so as to grasp the base of this fold. In applying the clamp the tip should point to the right shoulder and the handle of the outer side of the left hip, and the lowest portion of the stomach is grasped in the tip of the blade of the clamp (Fig. 674). The clamp is now put in a horizontal position. The duodenojejunal flexure is found by the finger, the jejunum is identified and its natural position is noted. The jejunum is picked up

and "drawn tight" and a spot is noted which reaches the greater curvature of the stomach when the jejunum is in its natural position. The point noted is 5 inches from the flexure and the anastomosis is made to the jejunum above the spot. The clamp is applied in the side of the gut opposite the mesentery. The surgeon must be sure that the jejunum is not twisted around its longitudinal axis. If it is, the clamps are not rightly applied, and they must be placed so that after the anastomosis the jejunum lies in its natural position without a twist. The clamped gut is placed by the side of the clamped stomach, a bit of gauze being put between them (Fig. 675). The stomach (except the clamped portion), the omentum, and transverse colon are returned to the abdomen and the clamps are surrounded by gauze. Each clamp holds a fold $3\frac{1}{2}$ to 4 inches in length. Pagenstecher's celluloid thread is used for suturing. The first line of sutures is passed as shown in Fig. 676. In front of these sutures an incision is made into the stomach and another into the jejunum, the serous and muscular coats being first divided, and an ellipse of mucous membrane being removed (Fig. 677). The next row of sutures is inserted as shown in Fig. 678. When this row is completed the clamps are removed and the long suture of the first row is picked up again and the operation is completed (Fig. 679). Finally, the edges of the mesocolic opening are sutured to the jejunum. The parts are cleansed with salt solution,

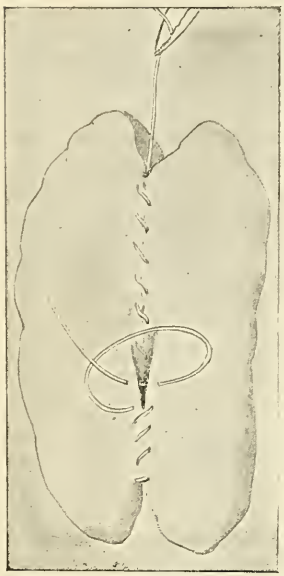


Fig. 679.—Moynihan's method of gastro-enterostomy: The serous suture resumed (Moynihan).

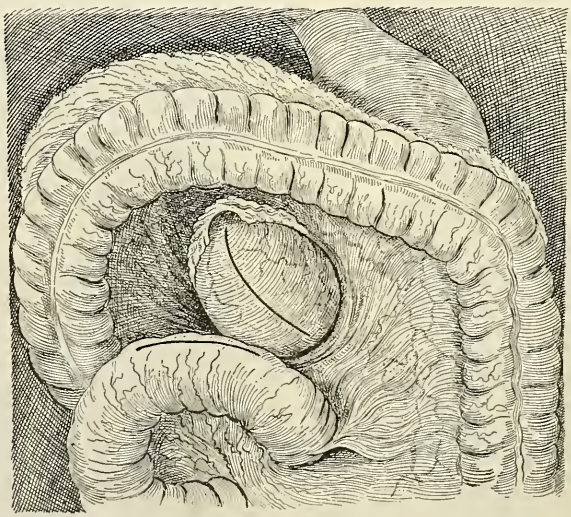


Fig. 680.—Mayo's method of gastro-enterostomy: Showing posterior wall of the stomach drawn through a rent in the transverse mesocolon. Note slight separation of gastrocolic omentum from its attachment to the stomach, permitting anterior wall of stomach to appear, and insuring drainage at lowermost level. Black lines mark site of proposed anastomosis; the jejunum shows at its origin.

the suture line is inspected, the parts are returned to the belly, and the abdomen is closed. (See Moynihan's "Abdominal Operations.")

**The No-loop Operation of the Mayos.**—(Figs. 680–682).—It is this operation I usually perform. By it the gastric opening, which is placed in the line advised by Moynihan, extends $\frac{1}{4}$ or $\frac{1}{2}$ inch into the anterior wall

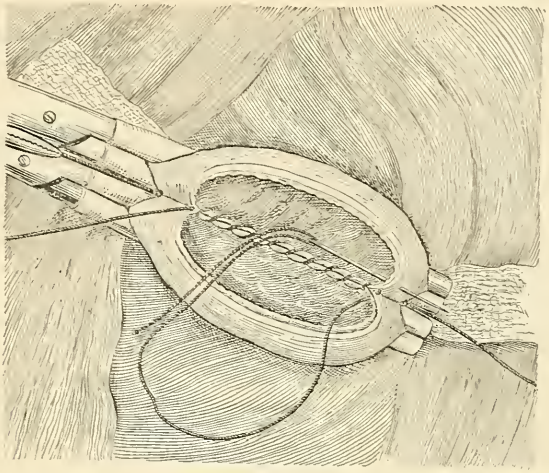


Fig. 681.—Mayo's method of gastro-enterostomy: Forceps in place and anastomosis half completed by suture.

of the stomach, and thus the lowest part of the opening will be the lowest part of the stomach (Fig. 680). The incision in the intestine begins from 1 to 3 inches from the origin of the jejunum, the measure being made on the anterior surface (Fig. 680).

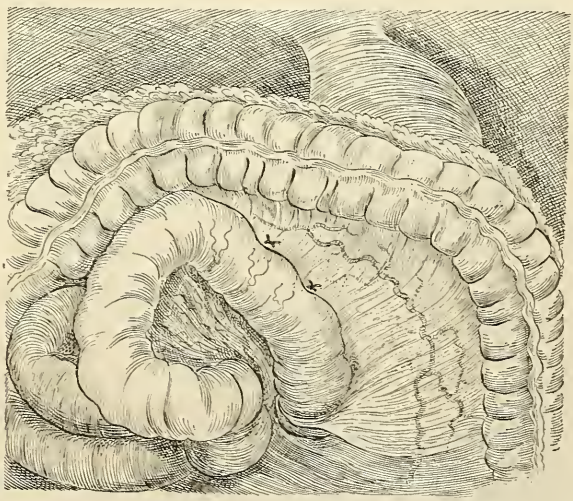


Fig. 682.—Mayo's method of gastro-enterostomy: Completed operation from behind. Margin of torn mesocolon attached by several interrupted sutures to line of union.

The object is to get as short a piece of jejunum as can be attached without tension. The operation is described as follows (Wm. J. Mayo, in "Annals of Surgery," Nov., 1905):

"(*a*) The abdominal incision is made 4 inches in length, ¾ inch to the right of the middle line, the fibers of the rectus muscle being separated. The lower end of the external wound lies opposite the umbilicus. This opening also enables inspection of the duodenum and gall-bladder and is reliable against hernia when closed.

"(*b*) The transverse colon is pulled out and the mesocolon made taut by traction upward and to the right, in this manner bringing the jejunum into view at its origin.

"(*c*) About 3 to 4 inches of the jejunum opposite the mesentery are drawn into a slightly curved clamp. The handles of the clamps should be to the right, to enable a short grasp on the intestine. Three-fourths of the circumference of the bowel is pulled through; the posterior border is not included, to prevent entanglement of the suture with the redundant posterior mucous membrane. The holding clamps are applied sufficiently tight to check hemorrhage and prevent extravasation of intestinal contents.

"(*d*) The ligament of Treitz is a short muscular mesentery covered by a variable peritoneal fold (too variable for a reliable landmark) extending upward from the origin of the jejunum on to the mesocolon. This peritoneal fold lies at the base of the arterial loop of the middle colic artery which supplies the transverse colon. The mesocolon is opened within the vascular loop and the posterior inferior border of the stomach pushed through. A small separation of the greater omental attachment to the stomach enables the anterior gastric wall to be drawn out posteriorly. The posterior gastric wall is drawn into a clamp, with the handles to the right, in such a manner as to just expose the anterior wall at the base.

"(*e*) The two clamps are laid side by side and the field carefully protected by moist gauze pads. With fine celluloidal linen thread on a straight needle the intestine is sutured to the stomach from left to right by a Cushing suture at least 2½ inches.

"(*f*) The stomach and intestine are incised ⅙ inch in front of the suture line and the redundant mucous membrane excised flush with the retracted peritoneal and muscular coats. With a No. 1 chromic catgut on a straight needle the posterior cut margins of the entire thickness of the gastric and jejunal wall are united by a buttonhole suture from right to left; at the extreme left the suture changes to one which passes through all the coats, of each side alternately, from the peritoneal to the mucous, then directly back on the same side from the mucous to the peritoneal. This acts as a hemostatic suture, and also turns the peritoneal coats into apposition. It passes around the anterior surface and is tied to the original end, which has been left long for the purpose. If silk or linen is used for this suture it may hang *in situ*, suppurating for months.

"(*g*) The clamps are now removed and the linen thread continued around until it is tied to the original end, firmly catching the blood-vessels in sight along the suture line. The parts are carefully cleansed and inspected. If necessary, a suture or two is applied to accurately coapt or to check the oozing.

"(*h*) The margins of the incised mesocolon are now united to the suture line by 3 or 4 interrupted sutures, and the parts returned into the abdomen." In this operation the greatest care must be taken to avoid twisting the gut around its longitudinal axis.

**Gastro-anastomosis** or **gastrogastrostomy** is an operation performed for hour-glass contraction of the stomach, a condition which occasionally ensues on the healing of an ulcer. In this operation an anastomosis is effected between the pyloric and cardiac pouches. It was devised and practised by Wölfler in 1894. I have performed it twice with success. Watson folds the two stomachs over each other, using the narrow isthmus as a hinge;

sutures the pouches together and leaves the ends of the sutures long. He incises the anterior wall of the anterior stomach in order to obtain access to the double septum between the two pouches. He makes an anastomosis opening through the double septum, sutures the edges, and closes the wound in the anterior wall of the anterior stomach. Wölfler made a vertical cut in each pouch and united these openings to make an anastomosis. The best plan is to apply clamps on each side of the isthmus and operate as we would for gastro-enterostomy (Fig. 684).

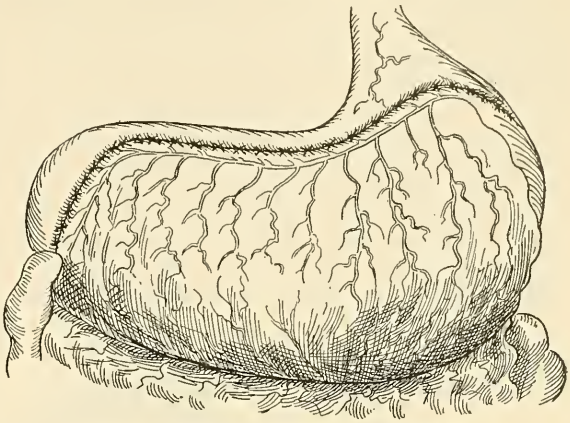


Fig. 683.—Bircher's method of gastroplication.

**Gastroplication (Brandt's Operation of Stomach-reefing for Dilated Stomach).**—Apply sutures in the anterior wall so as to form reefs, then tear through the great omentum and apply sutures in the posterior wall.

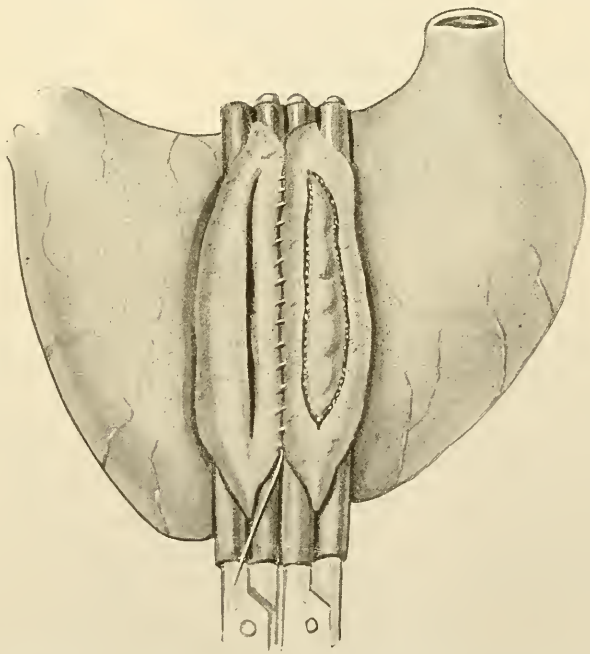


Fig. 684.—Hour-glass stomach. The application of clamps and the method of suture in gastrogastrostomy. The details are the same as in the operation of gastro-enterostomy (Moynihan).

The sutures pass through the serous and muscular coats. A continuous suture may be used on the anterior wall and another on the posterior wall, or numerous interrupted sutures may be inserted. This operation is of ques-

tionable value, and must never be used if stenosis of the pylorus exists, and stenosis of the pylorus is the most common cause of gastric dilatation.

Bircher's method of gastroplication is shown in Fig. 683.

**Gastropexy (Duret's Operation for Gastroptosis).**—It has been shown by Duret that dyspepsia of a peculiarly severe type may be produced by prolapse or downward displacement of the stomach. In this condition he advised the following operation: Perform a median laparotomy, but do not incise the peritoneum in the upper portion of the wound. Expose the stomach and fix it by means of a silk suture to the undivided but exposed peritoneum. The suture should be parallel to the lesser curvature and near the pylorus should be horizontal.[1] Rovsing, too, fixes the stomach to the abdominal wall. So do Hartmann and Eve. The operations of Duret, Rovsing, Hartmann, and Eve, of London, fix and distort the stomach. This seems to me an objectional procedure and liable to be followed by pain. To fix an organ which

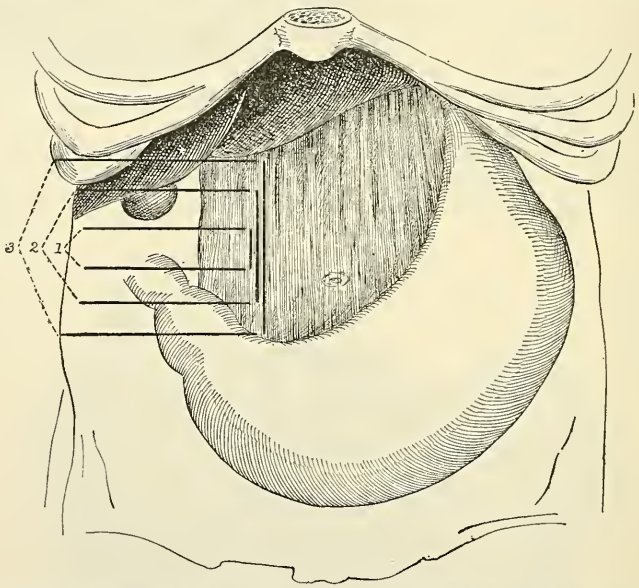



Fig. 685.—Beyea's operation for gastroptosis: 1, Position of one suture of first row; 2, one suture of second row; 3, one suture of third row. Others of each row introduced at intervals to and including the gastrophrenic ligament.

undergoes active peristalsis must surely be productive of difficulty. Byron Davis advises the suturing of the gastrohepatic omentum near its attachment to the lesser curvature to the stomach wall as high as possible. Beyea has devised an operation which is free from the objections which may be urged against Duret's operation. Sometimes gastro-enterostomy is also performed.

**Beyea's Operation for Gastroptosis.**—Insert three rows of interrupted silk sutures through the gastrohepatic omentum and the gastrophrenic ligament. Each suture is passed from above downward and the row begins at the right and passes to the left (Fig. 685). When the sutures are tied, a fold or plication is formed in the ligaments, the supports of the stomach are shortened, and the viscus is elevated to a normal position without any disturbance of its physiological mobility ("Univ. of Penna. Med. Bull.," Feb., 1903).

**Ransohoff's Omentopexy for Gastroptosis.**—Ransohoff ("Medical Communications of the Mass. Med. Soc.," 1912, vol. xxiii) points out that

[1] "Rev. de Chir.," June, 1896.

Beyea's operation is insufficient because only the right and left borders of the gastrohepatic omentum act as supports. He uses the omentum to raise and fix the stomach, and at the same time raises and fixes the transverse colon. He makes spaces between the fascia and peritoneum as advised by Coffey and sutures the omentum into these spaces. If necessary he also does gastroplication, or coloplication, or reefs the mesocolon or separates bands and adhesions, or shortens the round ligament of the liver.

**Duodenostomy and Jejunostomy.**—It has been suggested that one of the above operations should be performed in a case of pyloric obstruction in which neither pylorectomy nor gastro-enterostomy is feasible. Duodenostomy is said by some to be an easy operation because of the mobility of the pylorus and first part of the duodenum, and to be not only easier, but safer, than jejunostomy, because it makes the fistula above the opening of the common bile-duct ("Bull. et Mém. de la Soc. de Chir. de Paris," No. 39, 1901). Cackove advocates the operation in some cases of gastric ulcer with repeated hemorrhages and some cases of gastric cancer. In the latter cases he asserts that the mortality is about the same as from gastro-enterostomy and the prolongation of life is greater ("Arch. f. klin. Chir.," Bd. lxv, Heft 2). Hartmann's case of duodenostomy lived two months. The operation was performed for extreme cicatricial stenosis of the pylorus due to swallowing hydrochloric acid.

Jacobson disapproves of both procedures, and objects particularly to duodenostomy, because it involves a portion of the intestine which is difficult to deal with, and because important fluids escape constantly from the fistula.[1]

If duodenostomy is performed, it should be done in the same manner as gastrostomy by Witzel's method. I regard jejunostomy as an operation which is occasionally justifiable and as preferable to duodenostomy. As performed to-day there is little danger of leakage, even if the tube slips out. This operation puts the stomach at absolute rest. It is employed in very extensive ulceration, in multiple ulcers, and in some cases of cancer in which gastro-enterostomy is impossible or is contra-indicated, because the fistula would be in or too near the malignant growth. Wm. J. Mayo ("Am. Jour. Med. Sciences," April, 1912) regards the operation "as an active competitor of gastrostomy in cases of esophageal and cardiac obstruction." Mayo-Robson ("Brit. Med. Jour.," Jan. 6, 1912) advocates the operation. Mayo makes an epigastric incision, picks up the jejunum, makes a little opening at a point from 12 to 16 inches from the jejunal origin, introduces a rubber catheter (No. 9 English) "down stream" for about 3 inches, catches it to the wall of the bowel by one suture of chromic gut, infolds it for 1 inch or more as in Witzel's gastrostomy, using mattress sutures of linen, anchors the gut to the peritoneum by two or three lines of linen sutures at the lower angle of the incision, and closes the abdominal wound (Wm. J. Mayo, Loc. cit.). Food can be given at any time.

If the tube slips out it must be put back at once, as the tract might close in a few hours. When the operation has been done for cancer the fistula is permanently maintained. If for ulcer, it can be allowed to close or can be closed surgically, if it persists, as soon as the ulceration heals. Billon ("Archiv. prov. de Chir.") has collected 127 cases. The direct mortality was 29 per cent. Sixty-four of the patients lived but three months or less. A single case lived beyond a year. This is not a very gratifying showing.

Maydl does a much more formidable operation (divides the jejunum, attaches the upper end to a far-away portion of gut, and attaches the lower end in the abdominal wound). He has operated on 25 cases, with 4 deaths.

**Enterectomy, or Resection of the Intestine with Approximation by Circular Enterorrhaphy.**—How much of the intestine can be removed without the patient dying from lack of nutrition? The question is not settled.

[1] Jacobson's "Operations of Surgery."

It has been stated that the removal from an adult of more than $6\frac{2}{3}$ feet produces nutritional disturbance, and that a child tolerates the removal of a piece relatively larger better than does an adult. Senn was of the opinion that excision of more than one-third of the intestine makes inanition inevitable. Certain it is that great lengths have been successfully removed, and the patients have not only lived, but have been well nourished. Ruggi removed 11 feet, Witall removed 10 feet 8 inches, Von Eiselsberg, 11 feet 8 inches, and Obulinski, 12 feet 2 inches. Brougham removed 11 feet 2 inches for mesenteric thrombosis and the patient recovered. Childe successfully removed 9 feet 6 inches of small intestine for embolism of the mesenteric artery. Hayes removed 8 feet $4\frac{1}{2}$ inches from a boy ten years of age, and the patient was well eight months later. Dressman reported 26 cases in each of which more than 3 feet 3 inches had been removed (Alexander Blaney, in "Brit. Med. Jour.," Nov. 16, 1901). Blaney adds 7 cases from literature, and tells us that in 9 of the 33 cases death occurred soon after operation.

Alexander Blaney, in the previously quoted article, reviews the subject of the resection of great lengths of intestine. He tells us that how much remains after a resection is important, but uncertain. It is uncertain because, as Treves has shown, the length of the intestine varies from 15 feet 6 inches to 31 feet 10 inches.

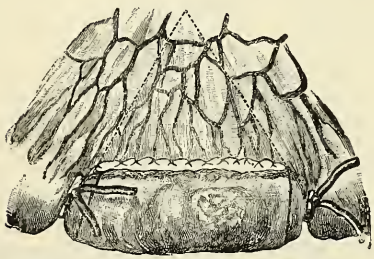


Fig. 686.—Excision of bowel: first step (Esmarch and Kowalzig).

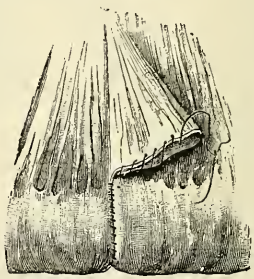


Fig. 687.—Excision of bowel with enterorrhaphy and stitching of the redundant mesentery: second step (Esmarch and Kowalzig).

Could end-to-end anastomosis be done as safely as lateral anastomosis it would usually be the preferred method. The great danger is infection from soiling. "Occasionally, the circular suture is the only feasible one" (Halsted, "Jour. of Exper. Med.," No. 3, 1912).

Resection of the jejunum is much more dangerous than resection of an equal length of ileum. Resection of the ileum is more dangerous than resection of the colon. If resection is employed, all diseased or injured bowel must be removed irrespective of ultimate bad consequences (Blaney). The operation is performed as follows: After opening the abdomen isolate the loop of intestine we intend to resect. Push a rubber tube through the mesentery close to the bowel, above the seat of operation, and pass a rubber tube through the mesentery below the seat of operation. Instead of tubes, strips of iodoform gauze may be used to encircle the bowel. Empty this segment of bowel by squeezing and stroking, tighten the rubber tubes, and clamp them to keep the bowel empty (Fig. 686). The diseased intestine is resected, each incision being carried through a healthy segment, and care being taken that the cuts are so arranged that at each end a blood-vessel from the mesentery reaches the edge of the cut bowel. Otherwise repair can scarcely occur. The lumen of each end of the divided gut is irrigated with salt solution. The divided surfaces are approximated by a double row of sutures—a continuous suture for the mucous membrane, and Lembert's, Dupuytren's, Cushing's suture, or Halsted's sutures

—to effect inversion. Thoroughly satisfactory approximation can be effected by one row of Halsted mattress sutures (Fig. 688). If a redundant fold of mesentery is left, it can be stitched at its raw edge (Fig. 687). Many surgeons remove a V-shaped piece of mesentery, tie the divided mesenteric vessels (Fig. 686), and introduce sutures so that no mesenteric vessel will be constricted (Fig. 688). The tubes are removed, and the wound is cleansed, closed, and dressed.

Senn effects invagination by means of a bone ring (Fig. 689).

If the two segments of bowel are unequal in size, it was formerly the custom to cut the narrow part of the bowel obliquely and the larger part transversely. To meet this complication Billroth devised *lateral implantation* (see Fig. 717). Suppose the cecum has been resected: its lower end is closed by Lembert sutures, an opening is made in the long axis of the periphery of the colon opposite the attachment of the mesocolon, and the end of the ileum is sutured into this incision. This is called *end-to-side approximation*, or implantation. It is used in the sigmoid, in the cecum, and in any intestinal segment in which the circulation is deficient. Eugene A. Smith ("Amer. Med.," May 10, 1902) sums up the advantages of end-to-side approximation as follows: The strain of peristalsis is less than in end-to-end union; the circulation of each end of the

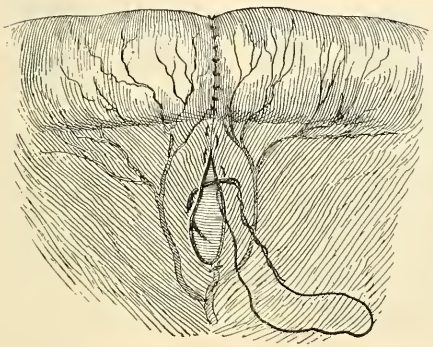


Fig. 688.—Suture of the mesentery after circular enterorrhaphy (Halsted).

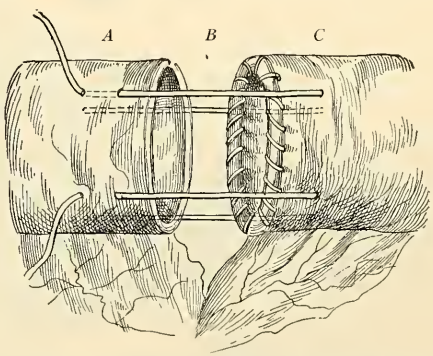



Fig. 689.—Senn's modification of Jobert's invagination method: *A*, Upper end lined with ring; *B*, invagination sutures in place; *C*, lower end.

bowel and the parts of bowel adjacent is better; each cut edge of mesentery is free to recover its circulation, and there is no dead space at the mesenteric border to lead to leakage.

Senn advised the insertion of an anastomosis ring in the ileum, the invagination of the colon as the ring is pulled into place, and firm suturing of the line of junction. By Senn's method the ileum may be implanted into the end of the colon or into a slit in the wall of the large bowel after the end of the colon has been closed. In some cases, in which one portion of bowel is larger than the other, lateral anastomosis is the preferable method. For a full week after an intestinal resection the patient is fed chiefly by nutrient enemata. During the first twenty-four hours nothing is given by the stomach but small amounts of hot water, and for the next six days only water and a little liquid food is allowed to be swallowed.

The use of *Murphy's button* (Fig. 691) permits of rapid approximation after resection (Fig. 690). This button closely approximates the portions of the intestine within its bite, rapid adhesion taking place. The diaphragm of tissue undergoes pressure-atrophy and liberates the button, which is passed per anum. It is claimed that the button-opening contracts but slightly. For end-to-end or side-to-side approximation of the small intestine a No. 3 button is used.

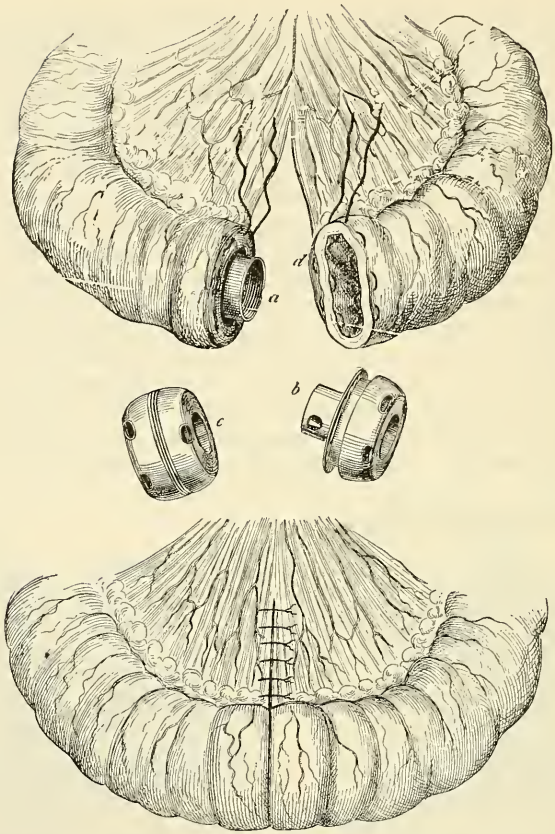



Fig. 690.—Resection of intestine: *a*, *b*, The two halves of the button; *c*, the two portions clamped together; *d*, introduction of the sutures for holding each half of the button in place. The lower figure shows the completed union of the intestine by the Murphy button; the slit in the mesentery has been closed by linear union (after Zuckerkandl).

For similar operations on the large intestine a No. 4 button is employed (Murphy). After the resection one-half of a button is inserted into each segment, and is held in place by a purse-string suture of silk which passes through all the coats (Fig. 690). The redundant mucous membrane is tucked in or clipped off, so that it will not be interposed between the serous surfaces. The serous surfaces are scratched with a needle and the halves of the button are locked (Fig. 690). It is not necessary to surround the margin of junction with sutures. Murphy says that liquid nourishment should be given as soon as the patient has recovered from the effect of the ether, and that the bowels should be moved at an early period, and frequent evacuations should be maintained. If the

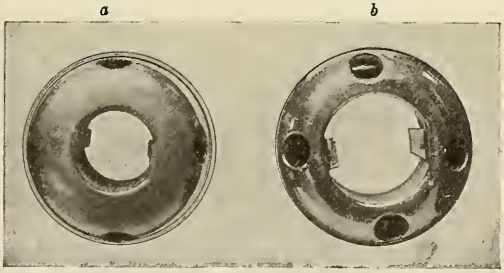



Fig. 691.—Comparison of old (*a*) and new (*b*) Murphy buttons.

button does not pass in four weeks, examine the rectum for it.[1] The situation of the button can be ascertained by the *x*-rays. An objection to the button is that it introduces a foreign body which must pass per rectum to complete the operation successfully. It may not pass, but trouble does not of necessity follow. In some cases its retention does lead to trouble, and intestinal obstruc-

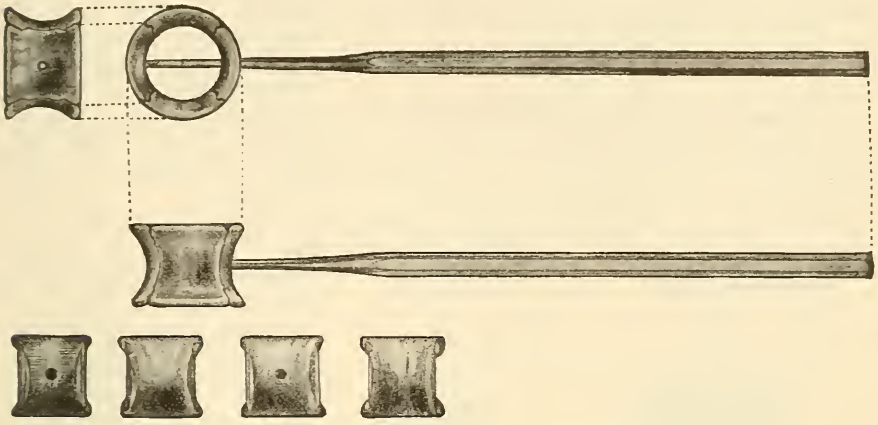


Fig. 692.—The segmented ring of Harrington and Gould.

tion ensues. If the caliber of the button blocks before dislodgment, obstruction follows, hence the rule to give saline purgatives the day after the operation.

Some surgeons have sought to make a button which would come apart and be absorbed after it had accomplished its purpose. One of these appliances is Frank's coupler, which is made of bone, the compression being

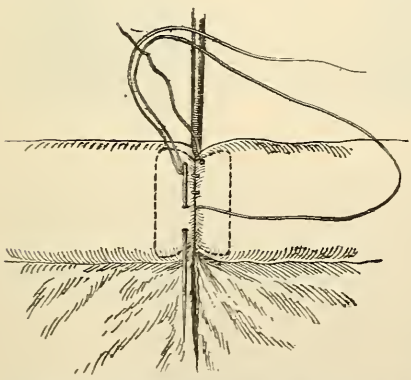


Fig. 693.—End-to-end union with aid of segmented ring. Continuous stitch beginning at one side of the handle (Harrington and Gould).

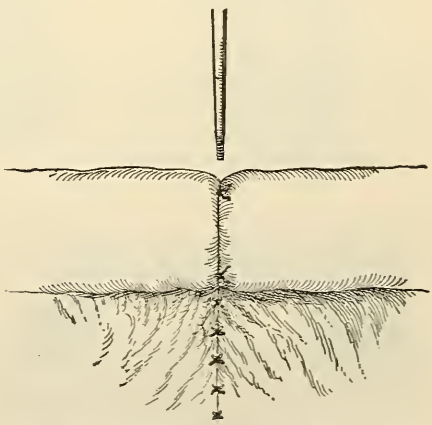


Fig. 694.—End-to-end union with aid of segmented ring. Handle unscrewed, suture completed (Harrington and Gould).

furnished by rubber. In this apparatus, however, the amount of pressure obtained is always uncertain and the rubber is apt to wear out. The button gives a lower mortality than the suture operation, and some surgeons now use it who once condemned it. Czerny is a strong advocate of the button.

[1] John B. Murphy, in "Med. News," Feb. 9, 1895.

Harrington and Gould use a *segmented aluminum ring*. This ring collapses into small segments after the anastomosis has been effected. By its use the authors believe that the operation is made more rapidly and safely ("Annals of Surgery," Nov., 1904). During the suturing the ring is held by means of a handle, which, after the anastomosis has been effected, is removed. The ring in the handle is shown in Fig. 692 and the operation in Figs. 693, 694.

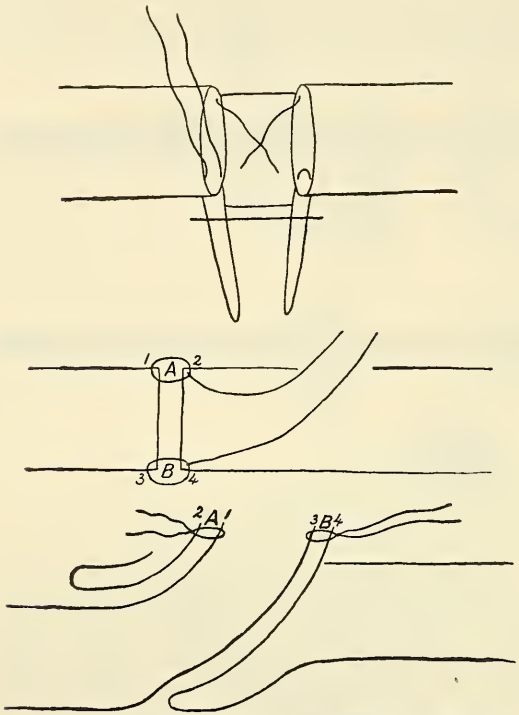



Fig. 695.—Maunsell's method of anastomosis (after Wiggin).

Maunsell has devised a most ingenious method of circular enterorrhaphy. The two portions of bowel are attached by two fixation sutures which penetrate all the coats (Fig. 695). An incision $1\frac{1}{2}$ inches in length is made through the wall of the proximal segment of gut, about 1 inch from its edge. The fixation sutures are brought through this opening, traction is made upon them, the distal portion of the bowel is invaginated into the proximal portion, and the ends emerge from the opening, their peritoneal surface being in contact (Fig. 695). Sutures of silk are passed through both sides of the area of invagination, the threads are caught up in the center, cut, and tied on each side. The fixation sutures are cut off. The invagination is reduced by traction. The longitudinal cut is closed by Lembert sutures.

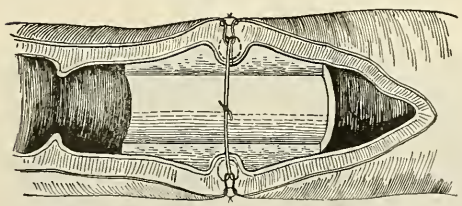


Fig. 696.—Robson's decalcified bone bobbin.

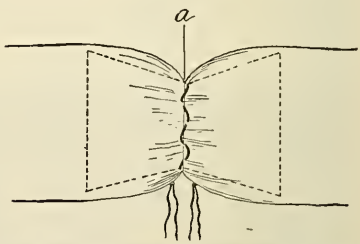



Fig. 697.—Allingham's decalcified bone bobbin.

A. W. Mayo-Robson performs circular enterorrhaphy and brings the ends of the gut together over a bobbin of decalcified bone (Fig. 696). Allingham uses a bone bobbin the shape of two cones joined at their apices. The bobbin is decalcified, except an area at the center (Fig. 697, *a*). Kocher performs circular enterorrhaphy as follows: A fixation suture is introduced

through the bowel at the mesenteric attachment and another is inserted at an opposite point. The intestinal ends are approximated by a continuous silk suture, which passes through all of the coats, but which includes more of the serous than of the mucous coat. The suture-line is overlaid by a continuous Lembert suture which includes the serous and a portion of the muscular coat.

In doing an end-to-end approximation I prefer to use the clamp of Moynihan (see Fig. 673), as shown in Figs. 698–700. We thus are able to hold the parts and keep them clean, rapidly make an even and secure stitch line, and have no free-edged septum.

Some surgeons have used inflatable rubber cylinders in making an end-to-end anastomosis (Halsted, Downes, and others). The method was devised by Treves, but was subsequently abandoned by him. Halsted no longer uses the inflatable cylinders. Professor Halsted is at present developing a method which seems to be aseptic and to offer other advantages. He calls it "A

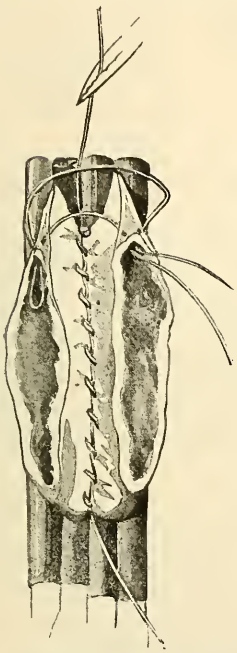


Fig. 698.—Moynihan's method of end-to-end anastomosis (Moynihan).

Fig. 699.—Moynihan's method of end-to-end anastomosis continued.

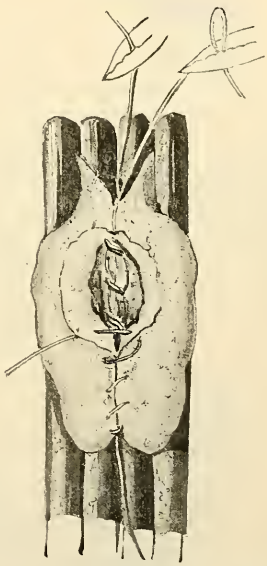


Fig. 700.—Moynihan's method of end-to-end anastomosis continued.

Bulkhead Suture of the Intestine" ("Jour. of Exper. Med.," No. 3, 1912). He reduces the wall of the bowel by crushing its submucous coat on each side of the loop to be excised; he then finally ligates and cuts through by a cautery. There is now a diaphragm on each end of the divided gut. He invaginates the gut by paper cylinders held in wooden mandrels, redivides it (thus cutting loose the diaphragm), and sutures it.

Connell has devised a method which places the knots in the lumen of the bowel (F. Gregory Connell, "Medicine," April, 1901). He maintains that the placing of the knots within the lumen of the gut has the following advantages: there is no foreign body; the suture passes away early; adhesions to neighboring organs are few; the serous approximation is perfect; the suture line is more secure; the septum is smaller and the danger of necrosis is less. The suture is shown in Plate 11.

Laplace has devised forceps which greatly facilitate suturing, which make it easy to obtain an even suture line, and which can be withdrawn after the suturing is finished, the small opening through which the instrument emerged

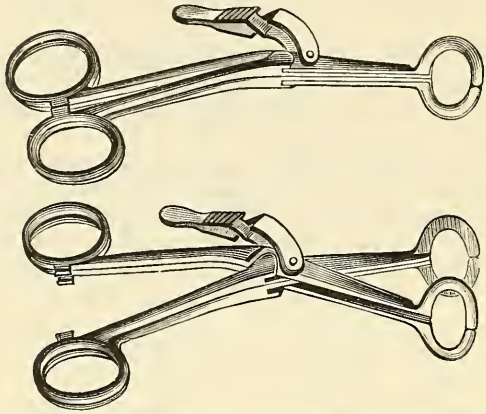


Fig. 701.—Laplace's forceps for intestinal anastomosis.

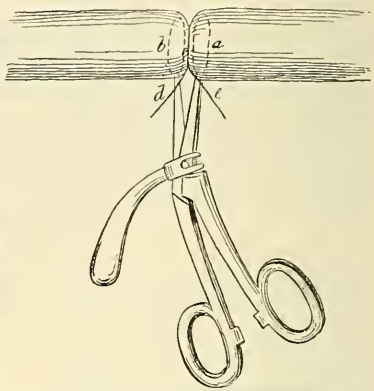



Fig. 702.—End-to-end anastomosis with the aid of Laplace's forceps.

being closed by a stitch (Figs. 701, 702). By aid of Laplace's forceps the operation can be neatly and rapidly performed, but a large diaphragm is formed, a considerable area is exposed to infection, the tissues of the diaphragm are bruised and may slough, the raw ends may grow together and cause obstruction, and it seems probable that considerable contraction will follow. Another objection is that an infected instrument is withdrawn from the bowel and may contaminate the peritoneum.

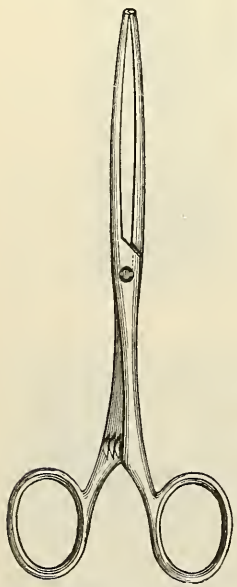


Fig. 703.—O'Hara's anastomosis forceps (about one-third original size).

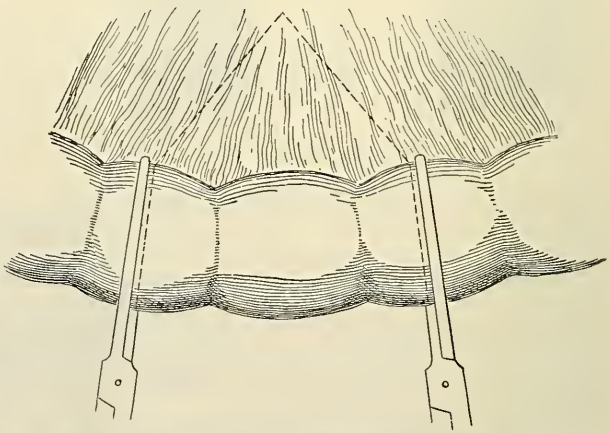


Fig. 704.—Showing the manner of placing forceps in resection of bowel; dotted lines show the incision to be made (O'Hara).

O'Hara's forceps (Fig. 703) permit of rapid and accurate suturing, but possess the same disadvantages as the Laplace forceps. In 1 case within my knowledge absolute obstruction from adhesion of the raw edges of the septum

followed its employment. Figures 704 and 705 show the use of O'Hara's forceps. Of the operations previously set forth, I prefer the clamp and suture as employed by Moynihan, the operation of Halsted by mattress sutures and without mechanical aids, and in some cases the operation with the Murphy button.

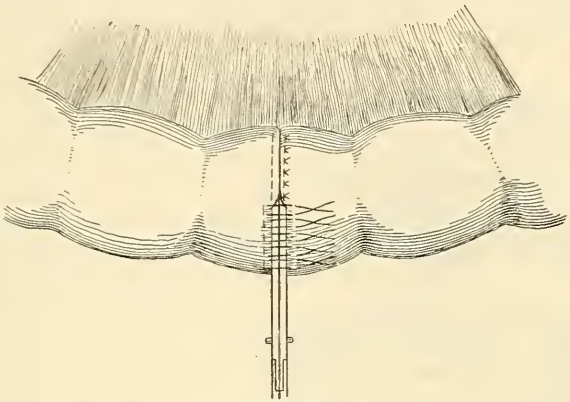


Fig. 705.—End-to-end anastomosis. Forceps brought together and held by serre-fine (not shown); sutures introduced, some of which are tied (O'Hara).

**Lateral Intestinal Anastomosis.**—Approximation may be effected by other methods than by end-to-end junction or by implantation. In fact, I prefer in most cases of resection to close each end of the divided gut and perform lateral anastomosis. It is a safer operation than end-to-end anastomosis and by it we can obtain as large an opening as we desire. Again, after lateral anastomosis the parts obtain a better blood-supply than after end-to-end suturing, because in the former operation the mesenteric vessels are not interfered with. Further, in lateral anastomosis there is little tendency to cicatricial contraction. Lateral anastomosis may be performed in some cases without a preliminary resection for the purpose of short-circuiting the fecal current, throwing a diseased portion of the bowel out of action, and thus avoiding obstruction (Fig. 706). This operation has the disadvantage that the diseased structure is not removed.

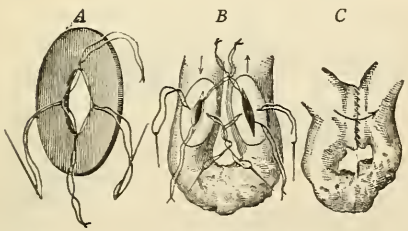



Fig. 706.—Senn's entero-anastomosis: A, Senn's bone plate; B, intestinal anastomosis; C, operation complete.

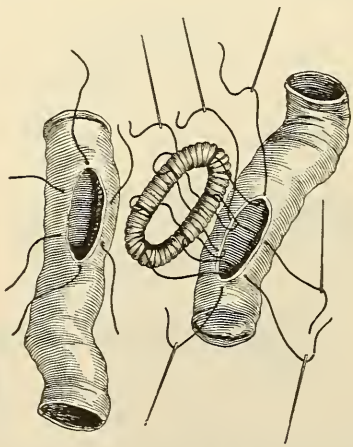


Fig. 707.—Method of passing the silk sutures in inserting the rings of Abbe.

**Operation with Rings.**—In this operation a portion of bowel above the obstruction and a loop below the obstruction are brought into the wound.

These segments are emptied, and are kept empty by fastening around them rubber tubes or iodoform strips. Two tubes are needed for each loop of bowel. Pack in gauze pads. Make an incision in one loop, in the long axis of the bowel, on the surface away from the mesentery; permit the contents to escape externally; irrigate this segment with saline solution, and introduce the bone plate of Senn (Fig. 706, A) or Abbe's catgut ring (Fig. 707). Calyx-eyed needles are used to pass the silk, and the threads of the ring are carried through the coats of the bowel and are gathered together in the bite of a pair of forceps. The other loop of intestine is treated in a similar manner. The two segments of intestine are so brought together that the two wounds are opposite each other, the posterior sutures being tied first, the upper next, then the lower, and finally the anterior threads. The ends of the threads are cut off, and the entire anastomosis is surrounded by a layer of Lembert or Halsted sutures or is encircled by Cushing's suture. Figure 706, B, shows an intestinal anastomosis partly finished, and Fig. 706, C, shows an anastomosis complete. Figure 707 shows the passing of the sutures when the catgut rings of Abbe are employed. After an intestinal resection each end can be closed and anastomosis effected as described above. Lateral anastomosis can be accomplished with a Murphy button, the intestine being prepared for the button as is shown in Fig. 708.

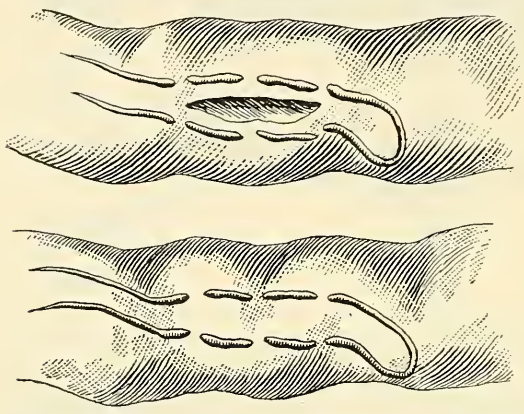


Fig. 708.—Showing relative size of incision and method of introducing sutures in lateral approximation with Murphy's button.

Abbe's method of anastomosis without mechanical aid is as follows: After resecting the bowel and mesentery and closing the ends of the bowel he places

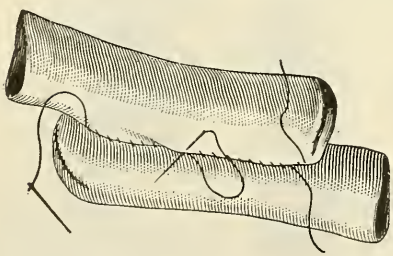


Fig. 709.—Suturing intestines in apposition before incision (Abbe).

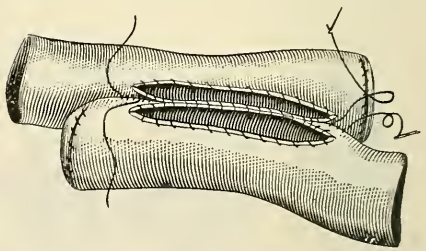


Fig. 710.—Showing the 4-inch incision and sewing of the edges (Abbe).

the extremities side by side and applies two rows of a Dupuytren suture, $\frac{1}{4}$ inch apart. These rows of sutures are 1 inch longer than the slit in the bowel will be (Fig. 709), the thread at the end of each row being left long. An incision is made in the bowel, $\frac{1}{4}$ inch from the sutures, both rows of threads being on the same side of the cut. This incision is 4 inches long. The other portion of the bowel is then incised in the same way. The adjacent cut edges are united by a whip-stitch which goes through all the coats, and the free cut edges are

stitched in the same manner (Fig. 710). The surgeon now utilizes the long threads of the first sutures, and brings the serous surfaces of the opposite sides together by means of Dupuytren's suture. Halsted performs anastomosis as follows: He places the two portions of bowel with their mesenteric borders in contact. Six quilted sutures of silk are introduced, tied, and cut off (Fig. 711, *a*). At each end of this row of sutures two quilted sutures are introduced,

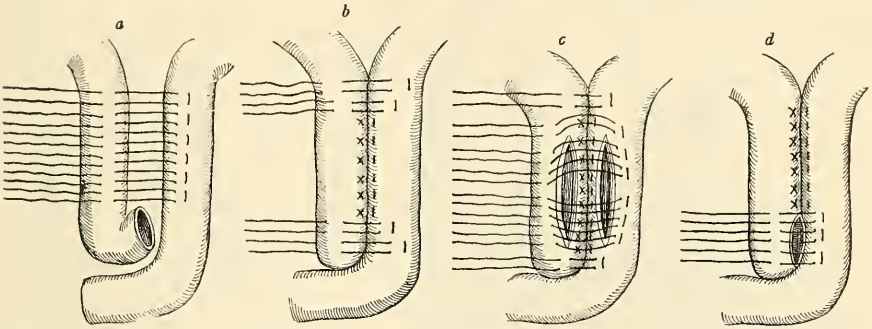



Fig. 711.—Halsted's operation for lateral anastomosis, showing four steps of same (Jessett, from Halsted).

tied, and cut (Fig. 711, *b*). A number of quilted sutures are introduced, as is shown in Fig. 711, *c*. The intestinal openings are made with scissors, and the sutures last introduced are tied and cut off (Fig. 711, *d*).

J. Shelton Horsley has suggested an ingenious method of intestinal anastomosis which secures for the sutured portion a greater diameter than that normal to the intestine.[1] After resection of the intestine and a V-shaped piece of

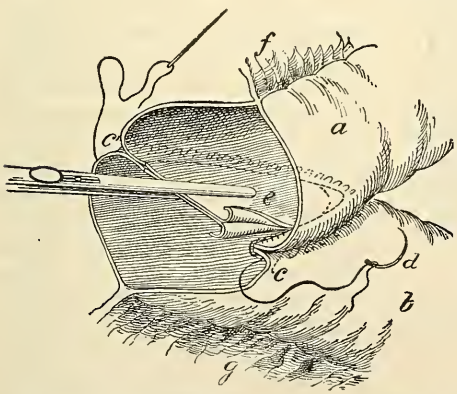



Fig. 712.—Represents the ends of the intestine in position and grasped by the artery forceps. The first row of sutures has been partially applied, the septum partly cut away, and the second row of overhand sutures begun: *a*, *b*, are the two ends of the intestine; *c*, *c'*, the first row of sutures (Cushing); *d*, the second row of sutures (overhand); *e*, the septum; *f* and *g*, the mesentery (J. Shelton Horsley).

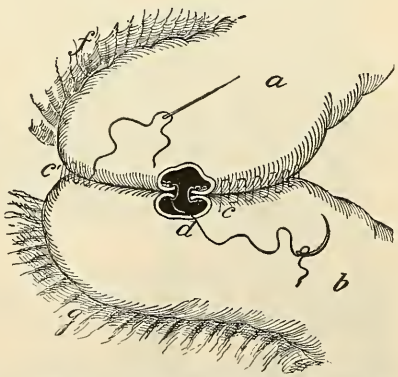



Fig. 713.—Operation nearly completed. The septum has been cut away, and the row of overhand sutures has been brought almost to its point of commencement. The cut also shows the first row of sutures (Cushing) as it should be continued after the overhand sutures are finished (J. Shelton Horsley).

mesentery, the ends of the bowel are placed side by side, the openings being in the same direction, and are clamped in place (Fig. 712). The first stitch approximates the two limbs of the bowel near the mesenteric attachment, is carried obliquely for about 2 inches to the border opposite the mesenteric

[1] "New York Polyclinic."

attachment, and continued over the other side (Fig. 712). The septum is cut away, a margin being left ⅓ inch wide. The edge of the shelf made by cutting the septum is sutured. When the suture reaches the end of the shelf, it is continued by invaginating about the rest of the resected ends (Fig. 713).

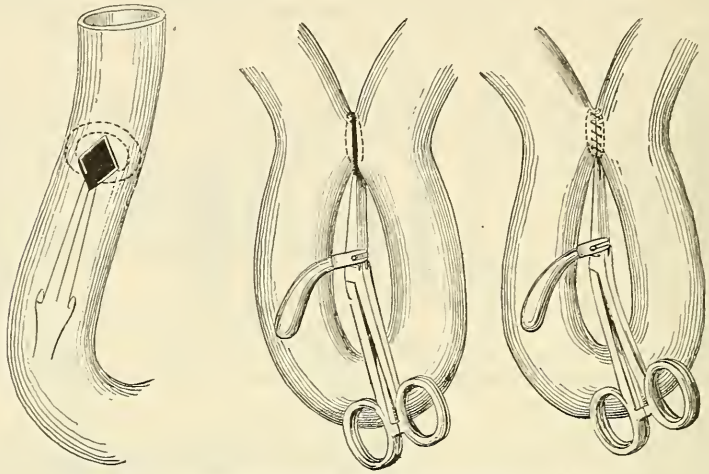


Fig. 714.—Lateral anastomosis with the aid of Laplace's forceps.

Bodine's method of intestinal anastomosis is referred to on page 1122. Laplace, of Philadelphia, has devised an operation in which temporary approximation is effected by means of forceps, the instrument being withdrawn before the abdomen is closed. Junction of two segments of intestine can be quickly and neatly effected by this method and the suture line is even and secure. The objections are that an infected instrument is withdrawn from the bowel and may contaminate the surface; that the septum is tightly squeezed and this septum may slough or may become infected, conditions which will be followed by infection of the suture line; and that contraction of the collar may ensue. The operation is more liable to be followed by leakage or by partial or complete obstruction than is the operation without forceps. Figures 714 and 715 illustrate the use of Laplace's forceps in lateral anastomosis. I usually perform lateral anastomosis with the assistance of Moynihan's clamps, the method being identical with the operation of gastro-enterostomy. Moynihan's operation is shown in Fig. 716.

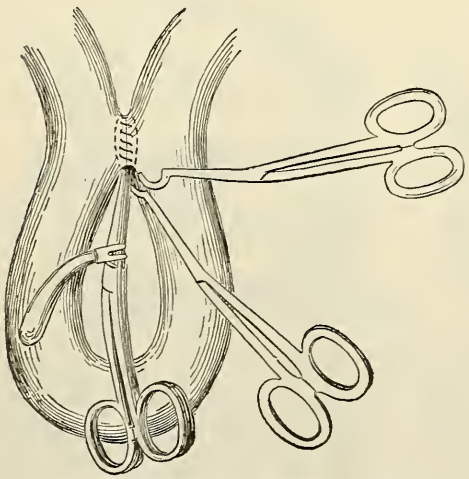


Fig. 715.—Withdrawal of Laplace's forceps.

**Consideration of Methods of Intestinal Approximation.**—At least 250 methods of uniting a divided intestine have been devised and the best method is a matter of dispute. The essentials of a good method are: rapidity of execu-

tion, the formation of an even and reliable line of junction, and the absence of any considerable permanent septum. The Murphy button can be applied with great rapidity, and rapid operation is of immense importance in intestinal work. The opening left by the Murphy button is small (too small, some surgeons think), but it does not strongly tend in most instances to contract because the tissue-diaphragm is separated by tissue-atrophy and not by inflammatory gangrene. The separation of the diaphragm is a most valuable feature. No other instrument thus cuts away the objectionable septum. Occasionally the opening made by the button contracts and gives trouble; occasionally the lumen of the button blocks with feces; occasionally the button is retained, this latter complication being especially frequent after anterior gastro-enterostomy. If the button is used, liquid food should be given soon after the effect of the anesthetic has passed off, and movement of the bowels should be obtained at an early period after operation and frequent evacuations should be maintained. The button gives better results in end-to-end approximation than in lateral anastomosis. Moynihan's forceps, Laplace's forceps, O'Hara's forceps, the decalcified bone plates of Senn, the catgut rings of Abbe, the segmented ring of Harrington, the catgut strands inside of rubber tubing of Brokaw, Chaput's button, Allingham's bone bobbin, Robson's bone bobbin, Frank's coupler, Clark's bobbin, tubes or plates of potato or carrot, and rings or plates of leather, all have their adherents. Of mechanical appliances, the best are Murphy's button, the bone ring, and Moynihan's forceps. Of recent years many surgeons have abandoned all mechanical aids, and have returned to closure by simple sutures. The ideal operation is without mechanical contrivances. But such devices are time-savers, and to lessen the time of operation will often save life. Further, Moynihan's forceps prevent fecal extravasation and consequent infection. What method to follow must be determined in each particular case by a study of the necessities of the situation. Nevertheless, it may be possible to formulate a few general rules: If the condition of the patient is excellent and the bowel is in a fairly healthy condition, well above and well below the seat of trouble, end-to-end approximation should be performed by circular enterorrhaphy with the aid of Moynihan's clamp, or each end can be closed after resection and a lateral anastomosis be effected with the aid of the clamp. If the condition of the patient is such as to make haste necessary, use a Murphy button. If the bowel below the seat of trouble is much contracted and haste is necessary, do not use a Murphy button, but

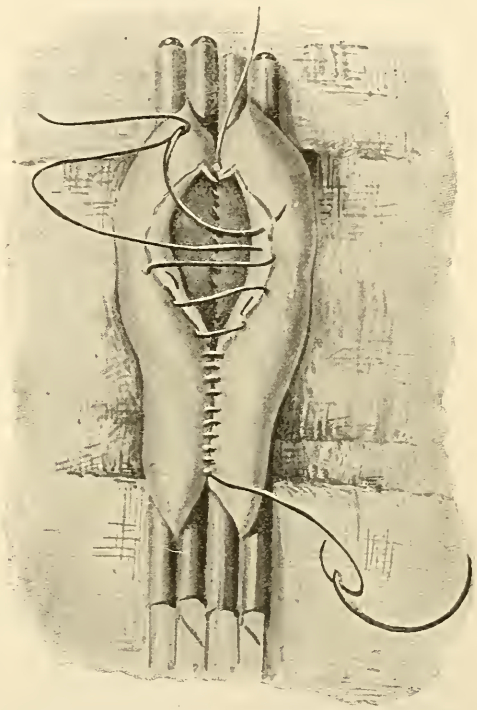


Fig. 716.—Moynihan's inner surfure in lateral anastomosis to show the infolding of the mucosa which results. A loop of the suture lies on the mucous surface (Moynihan).

use Senn's bone plate or Robson's bobbin. If haste is not imperatively necessary, do simple enterorrhaphy. If the surgeon is obliged to join a very much distended bowel to a very much contracted bowel, perform end-to-side approximation (implantation) with the bone plate of Senn or by simple suturing, or else effect side-to-side junction by the method of Abbe or of Moynihan.[1]

**Local Intestinal Exclusion.**—This operation was introduced by Salzer in 1891. It excludes the fecal current from a portion of the intestine. In complete exclusion the intestine is cut through above and below the diseased portion, and the ends of the healthy gut are united to each other or the end of one portion of gut is implanted into the side of the other. Both ends of the excluded portion may be fastened to the skin, making a double fistula (Von Eiselsberg); the distal end or the proximal end alone may be fastened to the skin, the other end being closed by sutures and replaced within the abdomen. Sometimes each end is closed and dropped back, and a fistula is made in the middle of the excluded portion to permit of drainage. Some operators close each end by suture and drop them back, and do not drain the excluded portion; and others aim at the same end by suturing together the two ends

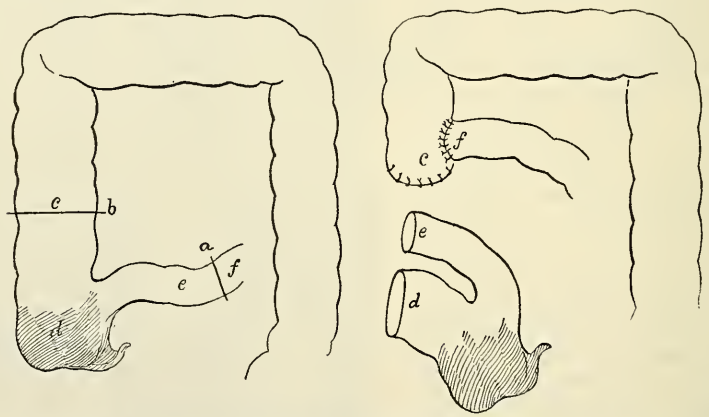



Fig. 717.—Operation of complete exclusion of the cecum: *a* and *b*, Lines of incision; *f* is implanted into *c*; *e* and *d* are sutured to the abdominal wall.

of the excluded part. It seems wisest to suture both ends, or at least one end to the skin (LeDentu, in "Rev. de Gyn. et de Chir.," Jan. and Feb., 1899). It is true this makes a permanent fistula, but if it is not done, the loop may become distended with secretion containing virulent bacteria, a condition which may lead to perforation and death. Exclusion is rarely performed upon the small intestine. It is best suited to the large intestine. If it is done at all, complete exclusion is the best operation (Fig. 717). Partial exclusion is rarely satisfactory. Exclusion has been performed instead of colostomy in cases of intestinal obstruction, but it is best suited to inflammatory areas or tumors, irremovable because of adhesions or some other cause. After the operation the diseased area may improve because of drainage and freedom from irritant fecal matter. In many cases it can be irrigated through the fistula. Sometimes the diseased part improves sufficiently after a time to permit of extirpation.

**Surgical Treatment of Ascites Resulting from Hepatic Cirrhosis (Epiplopexy; Talma's Operation).**—The portal system communicates with the vena cava by means of a number of small vessels. Normally, only an

[1] See the discussion of this subject by the late J. Greig Smith, in his "Abdominal Surgery."

insignificant amount of portal blood passes by this route to the general circulation. When cirrhosis obstructs the flow of blood through the liver, the radicles of communication between the portal system and the vena cava enlarge and an increased amount of blood is thus sent direct to the systemic circulation. Adhesions develop between the parietal peritoneum and some of the viscera and the collateral circulation is further increased. Thus, Nature seeks to prevent ascites. If, however, the obstruction to the passage of portal blood becomes so great that "the collateral circulation is no longer able to maintain an equilibrium in the blood-pressure in the portal radicles, the pressure thus rises to a point at which transudation takes place and ascites develops" (M. L. Harris, paper read before Chicago Medical Society, Feb., 1902). The theory above set forth is the "mechanical theory"; but, as Harris points out, increased portal tension is not the only factor concerned in the production of ascites, chronic inflammatory changes in the peritoneum being "materially instrumental" in maintaining ascites by lessening the absorbing power of the peritoneum. Influenced by the mechanical theory of causation, Talma, of Utrecht, devised an operation to cure ascites by establishing more free communication between the portal system and the systemic circulation. Drummond and Morison about the same time independently devised a like procedure.[1] This operation is called *epiplopexy*. In some cases the abdomen has been opened and the omentum sutured into the abdominal wound; in others, between the layers of the anterior abdominal wall. The results are slightly better when the omentum is sutured between the layers of the abdominal wall. The gall-bladder may be sutured to the abdominal wall as well as the omentum. The liver and spleen, under surface of the diaphragm, and parietal peritoneum about the liver and spleen are usually rubbed harshly with a piece of gauze. Drainage is not used by most operators. It does not appear to contribute any favorable chances and it exposes the patient to the danger of infection. Morison, however, advocates it, and makes suprapubic drainage, a glass tube being carried into the rectovesical or recto-uterine pouch ("Brit. Med. Jour.," Jan. 20, 1912).

The operation ought to be performed early, before the onset of chronic inflammation of the peritoneum. In a great majority of cases the operation proves futile, and not uncommonly death soon follows from complications or because the disease is very far advanced. In exceptional cases the operation proves of distinct benefit. The operation shows the least mortality and the greatest number of apparent cures when the liver is large; the greatest mortality and the fewest cures when the liver is contracted. The greatly lowered vital resistance of these patients is the imminent danger. Renal disease, cardiac disease, other grave complications, and the absence of sufficient functionating liver substance to maintain life contra-indicate operation (Greenough, in "Am. Jour. Med. Sciences," Dec., 1902).

Harris, in the paper previously quoted, collected 46 cases; 23 of these were instances of alcoholic cirrhosis; 30 per cent. were dead within fourteen days; 52 per cent. were dead within two months; 56 per cent. were dead within six months. Ascites had returned in all of those who died late. At the end of one year or longer 13 per cent. had recovered from ascites. The remaining 30 per cent. were either unimproved or were said to be improved with some ascites.

Of the group of mixed cases constituting the remainder of those Harris collected, 10 per cent. were dead in four days, 25 per cent. were dead in four months. In 40 per cent. no improvement took place. In 10 per cent. the report was too early to give any information. About 15 per cent. were free of ascites after one year or longer, and 5 per cent. were cured of intestinal

[1] "Brit. Med. Jour.," Sept. 19, 1896.

hemorrhage, ascites never having been present. Greenough collected 105 operations: 42 per cent. were improved; 58 per cent. were not improved; 29.5 per cent. died within thirty days. Two years after operation 9 cases were apparently in good health ("Am. Jour. Med. Sciences," Dec., 1902). One of Morison's cases was alive and well eleven years after operation; another was well for six years, when an attack of pneumonia lead to death; another patient remained well for two years, but died after an operation for ventral hernia (Morison, in "Brit. Med. Jour.," Jan. 20, 1912).

**Operation for Intussusception.**—Air distention and hydrostatic pressure are uncertain; in an advanced case may rupture the gut; even in a recent case may fail or may reduce the bulk of the intussusception, but not its apex. Russell ("Intercolonial Med. Jour. of Australasia," March 20, 1902) alludes to the uncertainty of the method. He used hydrostatic pressure in 5 cases. Two died and 2 recovered. In 1 case the method failed and operation was then performed. It is safer and better to operate early, but if the conservative plan is tried and fails, operation should certainly be done at once, because an early operation enables the surgeon easily to effect reduction, and also because early complications are unusual. The incision is made in the midline above the umbilicus. The surgeon endeavors by manipulation to reduce the intussusception by pushing it back, not by pulling it out. If the intussusception is gangrenous, perform intestinal resection and circular enterorrhaphy. The same rule maintains when malignant disease of the gut exists (D'Arcy Power). It is inadvisable to make an artificial anus. *Maunsell's operation* is suited to cases of irreducible intussusception. It is performed as follows: A longitudinal incision is made in the intussuscipiens. The intussusception is gently pulled upon and is caused to protrude from this opening. Two straight needles threaded with horse-hair are passed so as to transfix the base, and $\frac{1}{4}$ inch above the needles the intussusception is cut off. The needles are carried completely through, the sutures are hooked up in the middle and cut, and the two ends are tied on each side. These sutures unite the intussusception to the intussuscipiens. The two surfaces are now carefully approximated by sutures. The sutures are cut. The stump is replaced. The longitudinal incision is closed with Lembert sutures.[1]

Russell (Ibid.) reports 16 cases operated upon: 12 recovered and 4 died. In every one of the 4 fatal cases the diagnosis was not made until the disease had lasted several days. In only 2 of the successful cases the diagnosis was made late. If operation is done in the first twelve hours the mortality, even in infants, will probably be comparatively small. If gangrene exists the mortality is enormous (at least 90 per cent.).

**Senn's Operation for Fecal Fistula.**—Suture the opening transversely with Czerny sutures of silk in order to prevent infection. Cleanse the surface thoroughly. Open the abdomen and separate the edges of the bowel from the parietes. Deliver the portion of bowel which contains the fistula and apply Lembert sutures over the Czerny sutures. Another method is to open the abdomen above the fistula, insert the fingers, cut out the skin and tissues around the fistula in an elliptical course, leaving them attached to the bowel, draw the bowel from the abdomen, pack gauze around, remove the tissues adherent to it, and suture the fistula transversely.

**Enterostomy** is the making of an artificial anus. If performed in the large bowel, it is called *colostomy*. In some cases of intestinal obstruction it is necessary to open the small intestine, and if this is required, the artificial anus should be made as near as possible to the cecum. The higher above the cecum it is made, the more apt is the patient to die of lack of nourishment. A small intestinal anus may be made in the middle line or in the

[1] T. Pickering Pick, "Quarterly Med. Jour.," Jan., 1897.

right iliac region. The bowel is fixed and opened as directed under Colostomy. In acute intestinal obstruction it may be necessary to open the bowel at once. In such a case Paul's tube (Fig. 718) is very useful. It is made of glass, is bent to a right angle, and has a rim near each end. The large tube is used in the colon, the small tube in the small intestine. A small opening is made in the intestine, the tube is introduced, and is tied in place by a silk suture which surrounds all the coats of the bowel, a gush of feces is caught in a basin, a rubber tube is fastened to the glass tube, and fluid feces are collected in a bottle and beneath an antiseptic fluid.[1] In from three or four days to a week the tube becomes loose and can be removed. Stewart's method of enterostomy was outlined on page 992.

**Valvular Cecostomy (Gibson's Operation).**—This operation was devised in 1900 by Charles L. Gibson, of New York. It is used in chronic dysentery. It allows us to flush the large intestine and to apply remedies to it. The incision is made over the caput coli, a small puncture is made in it, a soft catheter (No. 30 Fr.) is introduced well into the bowel, and is fixed there as is the tube in Kader's gastrostomy (see page 1087). After ten or twelve days the tube is not kept in place, but is introduced when needed. The fistula closes spontaneously on the discontinuance of introducing the catheter daily. Appendicostomy has, to a great extent, replaced cecostomy, because, as a rule, it is easier and safer. "The appendix may, however, not be of a suitable size or position (retrocecal) to lend itself properly to the procedure" (Gibson, paper read before the Internat. Surg. Assoc. at Brussels, Sept., 1911).

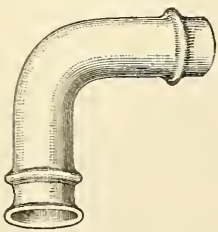


Fig. 718.—Paul's tube.

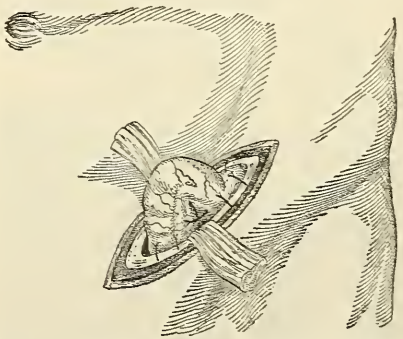


Fig. 719.—Inguinal colostomy (after Zuckerkandl).

**Inguinal Colostomy (Maydl's Operation)** (Fig. 719).—In this operation a vertical or oblique incision 4 inches in length is made over the portion of colon to be incised. In all cases when possible do a left inguinal colostomy. In right inguinal colostomy it is more difficult to deliver the bowel than in a left inguinal colostomy, because of shortness or absence of mesocolon at this point of the colon. Right inguinal colostomy has been performed for chronic amebic dysentery. It puts the colon at rest and permits of free irrigation. It is kept open until the dysentery is well. Appendicostomy and valvular cecostomy have replaced it for dysentery. It has also been employed for the treatment of ulceration of the colon. After the incision on the left side the colon usually bulges into the wound, but if it does not, it may easily be found by following with the finger the parietal peritoneum outward, backward, and inward, the first obstruction it encounters being the mesocolon. Draw the colon out of the wound until its mesenteric attachment is level with the abdominal incision. Push a glass bar through a slit in the mesocolon near the bowel, and wrap the ends of the bar with iodoform gauze to prevent slipping. Instead of the bar, a piece of gauze can be employed (Fig. 719), or a bridge

[1] Paul, in "Liverpool Med.-Chir. Jour.," July, 1892.

of skin can be made under the bowel by suturing the two skin edges. In order to make a spur the two parts of the flexure are stitched together by sutures which penetrate to and catch the submucous coat. Stitch the serous coat of the bowel to the parietal peritoneum (Fig. 719). Whenever possible, wait from

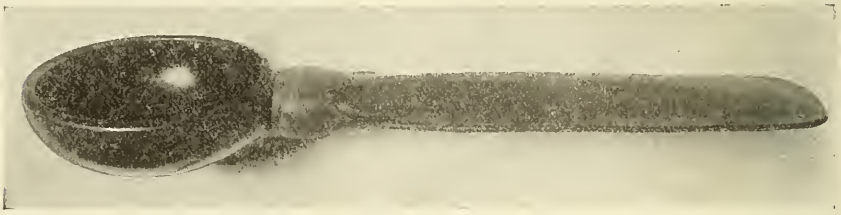


Fig. 720.—Stevenson's bag for inguinal colostomy.

twenty-four to forty-eight hours before opening the gut. The colon is opened by the cautery or by scissors. If the artificial anus is to be permanent, make a transverse incision through the bowel. Cut one-fourth way across the colon when it is first opened, and entirely across at a later period. If the artificial anus is to be temporary, the incision should be longitudinal. Maydl's operation has great advantages: it is quick, certain, reasonably safe, satisfactorily prevents fecal accumulation below the opening, and is rarely followed by absolute fecal incontinence. In many cases the bowels move but two or three times a day. The movements, however, come quickly with but little warning. Sometimes there is no warning. If diarrhea develops, there will be fecal incontinence as long as it lasts. An air-pad covered with gauze may be held in place by a firm belt, or the appliance shown in Figs. 720 and 721 may be worn.

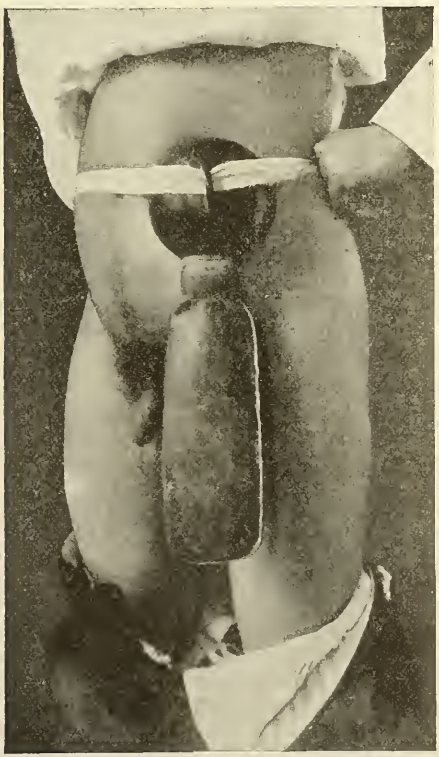


Fig. 721.—Stevenson's bag applied.

**Bodine's Operation** (Figs. 722, 723).—Bodine's method of colostomy permits of a future restoration of the fecal current by an easily performed anastomosis. This surgeon maintains that the spur after colostomy should reach to and remain at the level of the skin, a condition impossible of attainment by hanging the bowel over a rod or piece of gauze, because a spur thus formed is not thick and rigid and is inevitably dragged below the skin level, and when this dragging has taken place, some fecal matter will pass into the bowel below the artificial anus. Bodine opens the abdomen, sutures the parietal peritoneum to the skin, seeks for the lesion, and draws it with 6 inches of healthy bowel out of the incision. He lays the limbs of the loop side by side. He inserts a silk stitch, beginning at the point where exsection is to be made, and

for 6 inches unites the two segments close to their mesenteric borders. The loop is dropped into the abdomen until the beginning of the suture is on a level with the skin, and at this point it is fastened to the abdominal wound with a continuous catgut suture. The protruding lesion is cut off along the dotted line (Fig. 722). The artificial anus is thus established. When it is desired to close the artificial anus, divide the septum with scissors or a Grant clamp (Fig. 723) and close the abdominal wound.[1]

**Lumbar colostomy** is a most unsatisfactory operation. It does not completely intercept the fecal current, and it leaves the patient in a condition of wretched discomfort because fecal incontinence is inevitable. A patient who has had lumbar colostomy performed upon him either obtains little benefit, because the feces pass into the bowel below the opening which was made to intercept them or else they pour out of the opening uncontrolled, making the poor unfortunate a living horror to himself and others. It is rarely performed at the present day.

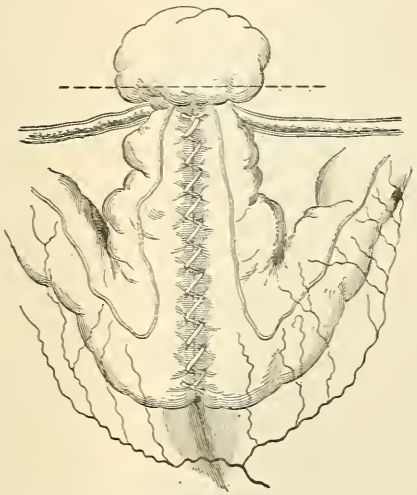


Fig. 722.—Bodine's method of colostomy, showing one side of the loop after it has been sutured, passed back into the cavity and stitched into the abdominal wound. The lesion is left protruding, and the dotted line indicates where the protrusion is to be clipped off.

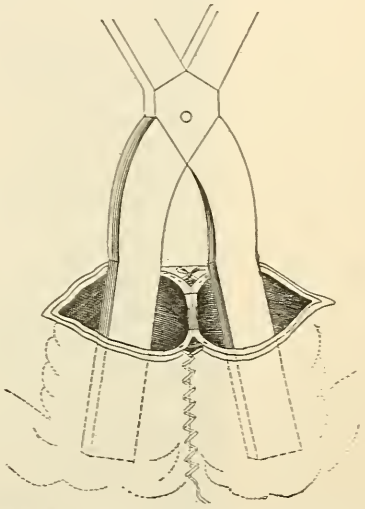


Fig. 723.—Bodine's method of colostomy, showing the septum to be divided in restoring the fecal current; Grant's clamp in position for the division. (In permanent colostomy this septum remains as a rigid and effective spur.)

The **healthy gall-bladder** has a capacity of about 1 oz., and its hue is bluish. If a gall-bladder contains calculi or has contained them, its hue is grayish-white or yellowish (Moynihan).

**Congenital Absence of the Gall-bladder.**—When the gall-bladder is shrunken and buried in adhesions, it is very difficult to find it at operation. Sometimes it is not found and one may jump to the conclusion that it is congenitally absent. This is occasionally, but very seldom, the case. Gray collected 19 instances of congenital absence of the gall-bladder ("Trans. of Chicago Path. Soc.," 1902). When it is absent the subject seems to have gotten along perfectly well without it.

**The Incision for Operations Upon the Gall-bladder and Bile-ducts.**—I have employed several methods, and have frequently used Bevan's incision (Fig. 724, B). The primary portion of the incision is shaped like the italic letter *f*. It is by the side of or through the right rectus muscle,

[1] "New York Polyclinic," Feb. 15, 1897.

and is shown by the double line in Fig. 724, B. The primary incision is used for exploration and cholecystotomy. The primary incision is from 3 to 4 inches long, and the extended portions, shown by heavy lines in Fig. 724, B, are added if required (Arthur Dean Bevan, "Annals of Surgery," July, 1899). This incision gives most satisfactory exposure, its edges can be separated without tension, and it injures but few of the nerves of the abdominal walls.

Kocher's incision (Fig. 724, A) gives a very satisfactory exposure. It cuts the two obliques and the transversalis muscle and divides intercostal nerves, but can be sewed up evenly and is seldom followed by hernia. Mayo-Robson's incision (Fig. 724, D) gives an admirable exposure of the common duct, although it damages the right rectus muscle and the nerve-supply of the inner part of the muscle. None of these incisions are entirely satisfactory. Collins's incision (Figs. 724, C, 725) ("Surg., Gynec., and Obstet.," March, 1909) seems to

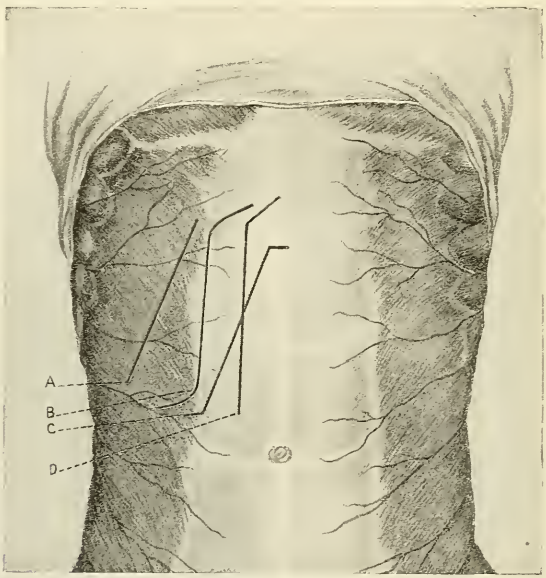



Fig. 724.—Incisions for the surgery of the bile-tracts: A, Kocher's incision; B, Bevan's incision; C, Collins's incision; D, Mayo-Robson's incision (Collins).

largely correct the defects of the previous operations. Collins thus describes his incision:

"The incision for the bile-tracts begins at the inner edge of the right muscle, 1 or 2 inches from the ensiform cartilage, and extends diagonally downward and outward to the outer edge of the right rectus, close to the level of the umbilicus. It cuts through the skin, fat, and anterior wall of the sheath of the rectus (Fig. 725). A short transverse incision about 1 inch in length may be made inward from the upper end of the diagonal incision through the skin, fat, and linea alba; and a similar one through the linea semilunaris at the lower end. In case more room is required the upper transverse incision may be extended further into the anterior and posterior walls of the sheath of the left rectus.

"The rectus muscle is then separated from its sheath. It is easily separated from the posterior portion of its sheath by blunt dissection, but the anterior portion presents some difficulty at the insertion of the linea transversæ, one of which is found about midway between the ensiform cartilage and umbilicus and is crossed by this incision. The attachment of the muscle to

the anterior wall of its sheath is very close at this linea transversa, and requires sharp dissection with knife or scissors.

"When the muscle is thoroughly freed from its sheath except at its outer border, it is easily retracted outward and allows the posterior wall of its sheath and the peritoneum to be incised in the same direction as the skin and anterior wall. The upper end of this diagonal incision through the posterior wall extends into the short, transverse incision across the linea alba. When this last cut is made the incision pulls open and gives ready access to the right upper abdomen." This incision does not damage the intercostal nerves, hence muscular atrophy is avoided. The opening through the different planes of the abdominal wall are not continuous, hence closure will be more

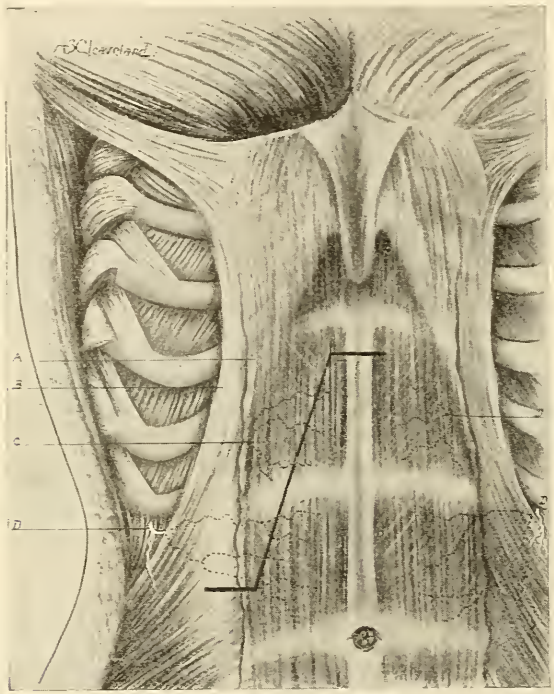



Fig. 725.—A, Anterior wall of sheath of rectus muscle; B, posterior wall of sheath; C, rectus muscle; D, intercostal nerves. The direction of the incision through the skin, fat, and anterior wall of the sheath of the rectus muscle (Collins).

solid. The opening in the posterior portion of the rectus sheath is protected by uninjured muscle.

**Cholecystostomy** or, as many call it, **cholecystotomy,**[1] is the operation of opening and draining the gall-bladder in order to extract gall-stones or secure the removal of infectious material. In the hands of the Mayos operations for stone exhibit a mortality of less than 1 per cent.; Kocher's mortality is 2 per cent. When death follows an operation on the gall-bladder or ducts, in about one-half the cases it is due to duct infection and is preceded by grave nervous symptoms (Mayo). Cholecystostomy is performed in cases of acute cholecystitis, in hydrops, and in empyema of the gall-bladder; in gall-stone cases in which jaundice has lasted for four weeks or more, and in colic of the gall-bladder with fever, the colic having recurred a second or third time

[1] First performed by Bobbs, of Indianapolis, in 1867.

(Carl Beck). The operation completed in one stage is performed as follows: The patient is placed recumbent with a sand-pillow beneath the liver and the incision is made. The peritoneum is opened. If the gall-bladder is distended, it is surrounded with pads and aspirated, and is then opened. Gall-stones are removed by forceps, the scoop, or irrigation. The gall-ducts are examined by the fingers external to them before opening the gall-bladder, and are sounded, if possible. If a stone is wedged in the duct, try to manipulate it back into the gall-bladder. If this fails, introduce an instrument from the gall-bladder and break up the stone; if this fails, open the duct, remove the stone, and close the incision in the duct (A. W. Mayo Robson). The only way to be certain that stones have been removed entirely from the cystic duct is to insert a finger and dilate. Sounds are unreliable. After the removal of all stones and fragments pass a rubber tube which has no side perforations into the gall-bladder, purse up the cut in the gall-bladder around the tube by means of a catgut suture, and suture the gall-bladder to the abdominal aponeurosis. If sutured to the skin, a permanent biliary fistula is apt to follow. It will seldom follow if the gall-bladder is sutured to the aponeurosis. A small piece of gauze is retained under the gall-bladder in case there should be a leak into the peritoneum and in case the peritoneum may have been soiled. If gauze causes no trouble it is retained in place from five to eight days. It can then be removed easily and without breaking encompassing adhesions. The drainage-tube, which drains into a bottle outside of the dressings and below the level of the bed, can usually be dispensed with in from one week to ten days. It should not be dispensed with until the bile becomes sterile.

Some surgeons have advocated immediate suture of the gall-bladder after removing a stone (*ideal cholecystotomy*). I believe this is never advisable when the stones are active for harm, because small calculi may be in the ducts and minute fragments of stone are often left in the bladder, and the drainage will remove them. Drainage also relieves the diseased condition of the gall-ducts and bladder. In Kocher's 31 operations by this method, gall-stones re-formed in 3 cases. Further, the operation with immediate suture is decidedly more dangerous when infection exists. The Mayos only employ it in latent cases of gall-stone disease when the existence of stones is discovered during the performance of an abdominal operation.

It is advised by some that the operation of cholecystostomy be performed in *two stages*. First, the bladder is exposed and sutured to the parietal peritoneum. When adhesion takes place, the gall-bladder can be opened without risk of infecting the general peritoneal surface. Riedel advocates operation in two stages, and so did Christian Fenger in certain cases. The two-stage operation is objectionable because it does not permit of satisfactory exploration of the ducts. The *biliary fistula* which is left by cholecystostomy usually closes spontaneously, but may not. If it does not close and the secretion is pure mucus, it is evident that the cystic duct is absolutely blocked and cholecystectomy should be performed.

If the secretion from a persistent fistula is bile and if the common duct is not obstructed, separate the edges of the gall-bladder opening from the parietal peritoneum, endeavoring to avoid entering the abdominal cavity, and close the fistula with Lembert or Halsted sutures. If the secretion is bile and the common duct is obstructed permanently, perform *cholecystenterostomy*. If the secretion does not contain bile and the cystic duct is blocked, remove the gall-bladder. At the end of 1907 Hans Kehr placed his mortality at 2 per cent. ("Jour. de Chir.," Oct., 1908). The report of St. Mary's Hospital, Rochester (the Mayo clinic), for 1912 shows 426 cholecystostomies, with 3 deaths.

**The McArthur Drip.**—In 1909 Lewis L. McArthur suggested using a biliary fistula as a means for introducing fluid or food into the duodenum. He

proved that if a tube draining bile from the gall-bladder is connected to an irrigator containing salt solution (elevated not more than 20 inches and giving a rate of flow not over 5 or 6 drops a second), there will be a continuous flow of fluid into the duodenum without any discomfort to the patient (McArthur, in "New York Med. Jour.," Jan. 27, 1912). Matas has improved the method by introducing into the duodenum at the time of operation and by way of the common duct a ureteral catheter, through which food, fluid, or medicine can be at any time carried into the duodenum (Rudolph Matas, in "New Orleans Med. and Surg. Jour.," Oct., 1911). The method is used in many cases of obstructive jaundice (after removal of the obstruction). The salt solution relieves thirst, removes toxins, and stimulates the kidneys. It relieves post-operative vomiting, favors intestinal peristalsis, combats flatulent distention, and causes movements of the bowels. Matas points out that liquid food

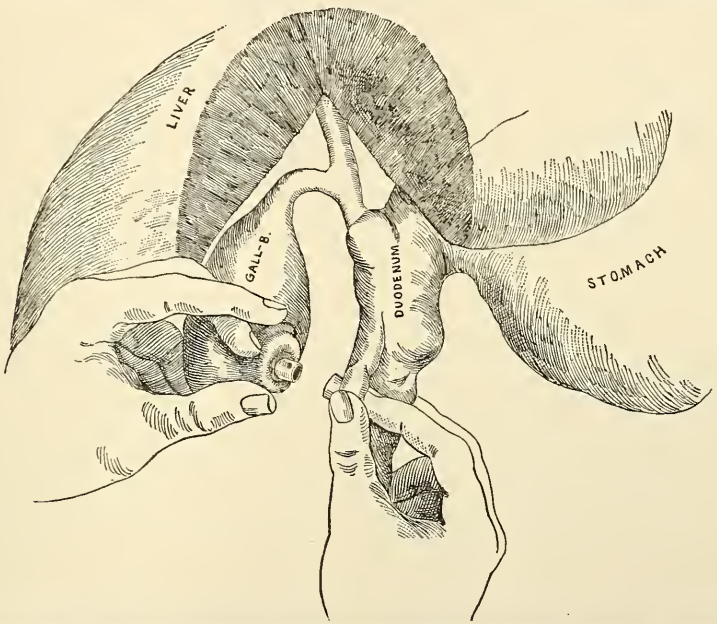



Fig. 726.—Showing method of holding parts while approximating a Murphy button in cholecystenterostomy.

and medicines (strychnin, castor oil, Hunyadi water, etc.) can be given by the biliary route. McArthur believes that the method can be used instead of jejunostomy in certain cases of pyloric obstruction and stomach ulceration.

**Cholecystenterostomy**[1] consists in making an anastomosis between the gall-bladder and intestine, preferably the duodenum, or, if this cannot be done, the jejunum. It is employed in cases of irremovable obstruction of the common duct. It is done chiefly in cases of malignant obstruction. It is not a suitable operation for gall-stones impacted in the common duct, because it does not remove the cause of trouble, infection of the bile-passages may follow, and the fistula is liable to contract. In those rare cases of common duct obstruction from gall-stones, in which the gall-bladder is distended and the patient is desperately ill, it may be done (Robson). In such a case Robson attaches the gall-bladder to the colon because the operation is easier and because he considers it as useful as the attachment to the duo-

[1] The operation was suggested by Nussbaum, but was first performed by Winiwarter in 1882 ("A Manual of Operative Surgery," by Sir Frederick Treves).

denum. Cholecystenterostomy can be done most rapidly and successfully by means of a small Murphy button. Before the gall-bladder is incised it is aspirated. Murphy's operation is shown in Fig. 726, and is similar in performance to intestinal anastomosis. I believe that Brentano is right and that it is best to do posterior cholecystenterostomy, bringing the jejunum through an opening made in the transverse mesocolon.

**Cholecystectomy** is the extirpation of the gall-bladder. It was first performed by Langenbuch in 1882. Sometimes primary extirpation is performed; at other times cholecystectomy is performed as a secondary operation, cholecystostomy for drainage having been first performed. Its performance may be demanded by the existence of phlegmonous inflammation or gangrene, ulceration, "in chronic cholecystitis from gall-stones where the gall-bladder is shrunken and too small to safely drain, and where the common duct is free from obstruction" (A. W. Mayo Robson), in empyema with greatly damaged walls, in fistula associated with irremediable obstruction of the cystic duct, the common duct being free, in cancer, and in some wounds of the gall-bladder. Objections to the operation are that drainage can only be obtained by putting a tube into the hepatic or the common duct, and that, should renewed drainage be subsequently required, the necessary operation will prove difficult and dangerous (Maurice H. Richardson, "Medical News," May 2, 1903).

After opening the abdomen the gall-bladder is found and is drawn into the wound. If it is distended and tense or if it is thought "to contain infectious fluid" (Lilienthal), it is packed about with iodoform gauze and emptied by an aspirating trocar. "When the walls are very friable, it is even wise to incise and empty the viscus, closing the opening by ligature or clamp before proceeding with the extirpation. The gall-bladder is usually quite a tough organ, and in the majority of cases it may be grasped with an ovarian ring-clamp applied near its fundus, which at the same time closes the aspiration puncture" (Lilienthal, "Annals of Surgery," July, 1904). The peritoneum which covers the gall-bladder must be divided just below the liver, the gall-bladder is dissected from the liver until the cystic duct is reached, the cystic artery is tied and divided, and if the liver ducts are healthy, the cystic duct is ligated with silk and divided, the stump is touched with pure carbolic acid and is covered with a layer of peritoneum fastened by sutures of fine silk. In cases free from infection it is not necessary to drain the bile-ducts. In cases with cholangitis external drainage is necessary, and it is obtained by incising the hepatic duct and inserting a drainage-tube, or, better, by leaving the stump of the cystic duct open. The report of St. Mary's Hospital, Rochester, Minnesota (the Mayo clinic), shows 255 cases of cholecystectomy with 5 deaths. Howard Lilienthal (Ibid.) reported 42 cases with 1 death. Hans Kehr's mortality at the end of 1907 was 3.6 per cent. ("Jour. de Chir.," Oct., 1908).

**Removal of the Mucous Membrane of the Gall-bladder.**—Mayo has suggested the removal of the fundus and of all the mucous membrane of the gall-bladder as an occasional substitute for cholecystectomy. By this operation we are enabled to drain the cystic duct and through it the hepatic ducts. A serious objection to the operation is that, as glands pass from the mucous coat to and through the muscular coat, it is impossible absolutely to remove the mucous membrane of the gall-bladder alone (Emil Reis).

**Drainage of the Hepatic Duct.**—This operation is employed for certain hepatic infections. It was first performed by Cabot in 1892. If the cystic duct is dilated throughout, it may be carried out through that. After opening the gall-bladder a tube is passed through the cystic and into the hepatic duct. It is often done after opening the common duct, a tube being carried up into the hepatic duct. The hepatic duct may be exposed and

opened directly, a tube being carried into it for a short distance and stitched to the edges of the incision in the duct by catgut. The tube should be surrounded by iodoform gauze.

**Supraduodenal choledochotomy** is the operation of incising the common bile-duct above the duodenum for the removal of a stone. It is also called *choledocholithotomy*. If drainage is used it is *choledochostomy*. It was first performed by Kümmel in 1889. Courvoisier did his first operation in 1890.

Cases upon which this operation is done are often deeply jaundiced and there is grave danger of infection and perhaps of fatal oozing of blood. In 1 of my cases this happened. The patient was laboring under stones in the common duct, associated with cancer of the head of the pancreas. In every case in which operation is contemplated for obstruction of the bile-ducts take the coagulation time of the blood. Normal coagulation time (taken by Wright's coagulometer) is from three to six minutes. Prolongation to seven or eight minutes calls for pre-operative treatment to hasten coagulability. If jaundice exists, it is customary to endeavor to prevent hemorrhage by employing Robson's plan: Give by the mouth from 30 to 60 gr. of chlorid of calcium three times a day during the twenty-four or forty-eight hours preceding operation, and 60 gr. by enema three times a day for the forty-eight hours following operation. I have followed this course in a number of cases, but am not convinced of its value. Instead of this method we may follow the plan of giving thyroid extract (5 gr. three times a day) for several days preceding operation.

The plan I now pursue I am certain does reduce the coagulation time distinctly. I give an injection of horse serum the day before operation and another the morning of the operation.

When ready to operate, a sand-bag should be placed under the lower ribs. This will bring the liver at least 2 inches nearer to the abdominal wound. The abdominal incision must be longer than that employed for cholecystostomy. The pylorus and stomach are drawn to the left, the colon and omentum are drawn downward, and the liver and ribs are lifted strongly upward. Gauze packs are inserted.

"The operator should now, after having separated adhesions, have a good view of the common duct within the free border of the lesser omentum, and on inserting his left index-finger into the foramen of Winslow, or on grasping the duct between the index-finger and thumb, he can, without difficulty, bring the duct well within reach, the concretion making a distinct projection."[1] A longitudinal incision is made, the stone is removed, and a probe is introduced into the duct to determine whether other stones are present.

Stones in the second and third portions of the duct are often missed and the second portion of the duodenum should always be palpated with the utmost care. If the lowermost stone removed from the common duct is faceted, we should always search most carefully to find a concretion which is lower still (F. Gregory Connell, "Annals of Surgery," April, 1908).

If a calculus is found in the lower part of the common duct, the surgeon tries to push it up so that he may reach it. This can usually be accomplished. Failing to push the stone up into reach, some try and force it into the duodenum. This attempt will sometimes, but seldom, succeed. If it does not succeed, the surgeon must perform a transduodenal, or a retroduodenal operation. Only in cases so shocked that prolonged operation is impossible is it proper to do cholecystostomy or cholecystenterostomy. If either of these palliative operations is performed a radical operation must be done later.

Many surgeons suture the incision in the duct. This procedure is rendered easier by the use of Halsted's hammer, which draws the duct toward the surface and keeps it under control (Fig. 727).

[1] A. W. Mayo-Robson's "Treatise on Diseases of the Gall-bladder and Bile-ducts."

Interrupted sutures of fine catgut are used. The muscular and serous coats may be included in each suture, and over this layer Lembert or Halsted sutures are applied. A drainage-tube is inserted and a piece of iodoform gauze is placed upon the suture line, the other end being brought out of the abdominal wound. This precaution is taken because leakage may occur. If it is found impossible to suture the wound in the duct, the operation then becomes a *choledochostomy* (although this term is usually used only when the incised duct is stitched to the abdominal wall). The surgeon carries a glass tube down to the opening and surrounds it with iodoform gauze, or inserts a rubber drainage-tube into the opening and carries it up toward the hepatic duct, or makes an incision into the right loin after the plan of Rutherford Morison, and carries a tube into the right kidney pouch, which is the most dependent part of the peritoneal cavity when the patient is recumbent. Personally I always drain the duct, when I have opened it for stone, carrying the tube up toward the hepatic duct. The same reasons which cause us to drain the gall-bladder after removing stones should influence us in this case.

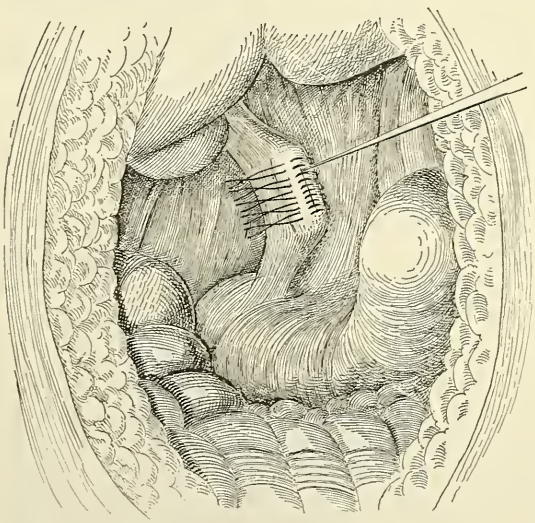


Fig. 727.—Suture of duct over Halsted's hammer.

Robson ("Lancet," April 22, 1902) has performed the operation of choledochotomy 60 times. In 10 cases of stone in the common duct he manipulated the stone back into the gall-bladder and removed it through an incision in that viscus by means of a scoop. The above maneuver is impossible unless the cystic duct is dilated. In 30 cases he crushed the stones between his finger and thumb, but this is only possible when the stones are soft, and it has the objection that it may leave fragments. If a stone is lodged in the common duct and cannot be manipulated back into the gall-bladder, choledochotomy should be performed. Robson's mortality in 60 cases of choledochotomy was 16.6 per cent. Since 1900 his mortality has been 7.1 per cent. Before that it was 23.8 per cent. Kehr's mortality is 4.1 per cent. During 1912 the Mayos performed choledochotomy 96 times, with 5 deaths ("Report of St. Mary's Hospital for 1912").

**Hepaticotomy.**—By this term we mean the opening of the hepatic duct. If the opening is drained the procedure is, in reality, *hepaticostomy*, although this term is seldom used to designate it. Hepaticotomy is performed for stone in the hepatic duct. The operation was first performed by Kocher in 1889. There were 7 cases on record in 1903 (Delagenière, in "Bull. et Mém. de Chir. de Paris," No. 10, 1903).

**Duodenocholedochotomy (McBurney's Operation; the Transduodenal Route).**—This operation is seldom necessary. In the more than 2000 operations performed by the Mayos on the gall-bladder and ducts, it was only required in 4 cases. I have never performed it. In 1891 McBurney proposed the method for the removal of gall-stones impacted near the papilla

("Annals of Surgery," Oct., 1898). McBurney's original suggestion was to open the duodenum, dilate or incise the papilla, remove the stone, and suture the duodenum. The duodenum must be mobilized so that it may be lifted into the wound. If the stone is located in the diverticulum of Vater, it may, in some few cases, be removed by simply stretching the opening of the duct with forceps (Collins's method). If this is not possible, the opening in the papilla may be enlarged by cutting or the duodenal mucous membrane over the stone may be incised (McBurney's plan). When the stone is not impacted at the outlet, but is lodged a little higher up, and when dense adhesions render access by the ordinary supraduodenal route difficult or impossible, the anterior wall of the duodenum may be opened longitudinally, the posterior wall of the duodenum and the common duct incised over the stone, the stone removed, the duodenum and common duct sutured together (Kocher's method, or *internal choledochoduodenostomy*), and the anterior wall of the duodenum closed. (See Charles Otto Thienhaus, in "Annals of Surgery," Dec., 1902.) After finding and removing a stone by the transduodenal route we must make a careful search to see that no stones are left before closing the duodenal incision. Robson opposes the transduodenal route and says he has abandoned it because of the danger of sepsis. Thienhaus (Ibid.) opposes this view of Robson and shows that in 29 operations by the transduodenal route there were but 2 deaths.

Connell ("Annals of Surgery," Jan., 1908) has collected 77 cases in which stones were removed by the transduodenal route. There were 10 deaths. In 2 of these cases duodenal fistula preceded death.

**Retroduodenal Choledochotomy.**—In this operation the second portion of the common duct is incised back of the duodenum without opening the gut. That this may be done the duodenum must first be drawn toward the midline of the body, and this can be done only by "mobilizing" the duodenum, incising the posterior layer of the parietal peritoneum 1 inch to the right of the descending portion of the duodenum. After freeing the gut and retroperitoneal structures the duodenum becomes sufficiently free to lift toward the left with a rotation. We thus expose the posterior aspect of the duodenum, the head of the pancreas, and the common duct.

The duct is opened, the stone removed, the duct sutured, and a drain inserted.

This operation has been successfully performed by a number of surgeons, but there is a great objection to it. In nearly all cases the common bile-duct passes *through* the pancreas rather than back of it. Büngner found this to be the case in 55 out of 58 dissections ("Gray's Anatomy," Seventeenth American edition, p. 1352). Hence, incision of the common duct in this situation means in nearly all cases incision of the pancreas and all the grave dangers of leaking of pancreatic fluid. The transduodenal operation is a much better procedure.

**Total Splenectomy.**—This operation is performed for wounds and rupture of the spleen, tumors, cysts, floating spleen, and non-leukemic splenic hypertrophy. Twisting of the pedicle of an ectopic or wandering spleen calls imperatively for operation. It should not be performed for hypertrophy in leukemia.

Vedova (quoted in "Practical Medicine Series," 1913, vol. ii) collected 134 cases of splenectomy for traumatic rupture of the spleen. There were 40 deaths. He adds these to Berger's 60 cases, with 25 deaths. This makes a total of 194 cases, with 65 deaths (33.5 per cent.).

In view of the bone-marrow changes in splenomyelogenous leukemia, we cannot hope to cure a patient by removing the spleen. In a leukemic patient the operation has a very high mortality from shock and hemorrhage. Geo. Ben Johnston ("Annals of Surgery," Jan., 1908) has collected 49 splenectomies in leukemia with only 6 operative recoveries (a mortality of 87.7 per cent.).

One of the 6 cases lived eight months, 1 lived four years, and it is claimed that 1 was cured.

Splenectomy has been performed for malarial hypertrophy (ague-cake). The operation has been advocated on the theory that by removing the spleen we get rid of the lurking place of the malarial parasites, but they also lurk in the bone-marrow and in the capillaries of the liver. The operation should not be performed for malarial spleen unless the organ is movable, unless it greatly interferes with the patient's comfort or occupation, or unless we fear rupture, and then, if it is done, it is for the movability, the discomfort, or the danger of rupture, and not for the malaria. It is to be noted that the operation does not cure the malaria. Johnston ("Annals of Surgery," Jan., 1908) collected 58 splenectomies performed for malarial hypertrophy since 1900. There were 50 recoveries and 8 deaths. To these he adds 3 successful ones of his own, making 61 cases, with 8 deaths (a mortality of 13.1 per cent.).

A number of operations have been done for splenic anemia or its terminal stage, which is known as Banti's disease. Those who believe that the splenic enlargement and anemia result from some underlying condition common to both do not operate. The theory of the operation is that removal of the spleen stops the production of some toxic material which causes anemia and cirrhosis of the liver, in other words, that the splenic disease causes the anemia. There seems ample evidence that splenectomy, if done early, may save the patient. It is useless and highly dangerous to do it after the development of the second stage of Banti's disease. Cushing's case was alive and well eight years after the operation.

Johnston (Ibid.) has collected 61 splenectomies for splenic anemia or Banti's disease. There were 49 recoveries and 12 deaths, a mortality of 19.5 per cent. I have performed splenectomy twice for Banti's disease, with one recovery and one death.

Johnston (Ibid.) notes 12 splenectomies for sarcoma of the spleen, with 9 recoveries. One lived eight and one-half years and died of heart disease; 3 are known to have died from recurrent sarcoma.

It is stated that there are on record 4 splenectomies for cancer. Moynihan doubts the diagnostic accuracy of the three earlier reports. Mary A. Smith records a case of colloid cancer occurring in a woman who had been operated on ten years before for ovarian cyst associated with pseudomyxoma of the peritoneum. The pathologist reported that the growth in the spleen was a metastasis of colloid carcinoma. This patient died seven months after the splenectomy from peritoneal and omental cancer ("Annals of Surg.," Jan., 1908).

In Johnston's table of 708 splenectomies for various causes (Loc. cit.) the mortality is 27.4 per cent. In the cases operated upon from 1900 to 1907 inclusive the mortality is 18.5 per cent.

In order to remove the spleen most operators make an incision from the anterosuperior spine of the ilium to the ribs. I prefer to make an incision below the left costal margin like Kocher's incision on the right side to reach the gall-bladder. This incision can be extended to any necessary degree, and posterior drainage of the pancreas region can emerge from its outer end. Open the peritoneum and divide adhesions between ligatures. If the spleen is adherent to the pancreas, it may be necessary to remove a fragment of the last-named organ. It is a very undesirable thing to have to do, and I lost a case from pancreatic leakage after having done it. Ligate the suspensory ligament and divide it. Bring the spleen well out of the wound. Surround it with gauze pads. Transfix the pedicle with stout silk. Tie it firmly, leaving the ends of the ligature long for a time, and cut through the pedicle beyond the ligature. Ligate the vessels separately with catgut. Cut off the long ends of the silk ligature and drop the pedicle back, unless apprehensive of bleeding, when it may

be fastened to the surface. The wound is closed without drainage, unless the pancreas has been injured, in which case posterior drainage is employed. Traction upon and ligation of the vessels in the pedicle may cause profound shock by injuring the splenic plexus, which is in close relation with the solar plexus (Jordan, in "Lancet," Jan. 22, 1899).

**Changes After Splenectomy.**—About two weeks after the removal of a normal spleen certain definite changes happen in adults, but not in children. These changes last for several weeks, and are manifested by enlargement of the lymph-glands, pain in and tenderness of bones, blood changes, loss of weight, weakness, thirst, polyuria, abdominal pain, elevation of temperature, and rapid pulse.[1] Tizzoni says that these changes are not obvious in children, because in them compensatory organs act at once, whereas in adults compensatory organs act slowly and with painful effort. Such symptoms are noticed when the spleen is removed because of a wound or a rupture, but rarely after removal of a diseased spleen. It is likely that compensating organs become active when the spleen is diseased, and consequently are in full operation when such a spleen is removed. After partial splenectomy these changes are not noted (Jordan). Changes can be prevented after splenectomy by the administration of tablets of extract of spleen, and red bone-marrow (Ballance), and iron (especially in foods) is of value.

The blood changes after splenectomy consist of diminution in hemoglobin and red blood-cells. The coloring-matter and cells do not become normal for two or three months.

**Splenopexy.**—This is the operation of anchoring a movable spleen. It should only be used when the spleen is not enlarged and is not diseased. Rydygier in 1895 published the first case, although both Tuffier and Kouwer operated before this date. Sutures should not be passed through the spleen: the structure is so soft that stitches are bound to loosen and the insertion will cause bleeding. A promising method is to create adhesions by the use of iodoform gauze, as is done for movable kidney, and as was done by Kouwer. Some advocate making a pocket outside of the peritoneum and bringing the spleen into this pocket, thus placing it extraperitoneal.

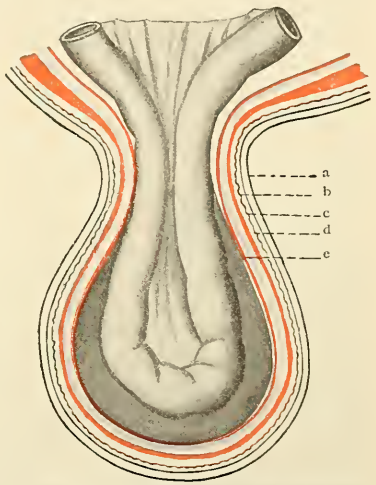



Fig. 728.—A diagrammatic representation of the coverings of a hernia: *a*, The skin; *b*, the superficial fascia; *c*, the muscular layer—*e. g.*, the cremaster muscle in an inguinal hernia; *d*, the transversalis fascia; *c*, *d*, have also been called the fascia propria herniæ; *e*, the peritoneum—*i. e.*, the sac of the hernia (Sultan).

**Abdominal Hernia or Rupture.**—A hernia is a protrusion of peritoneum liable to contain, containing at times, or permanently containing any viscus or part of a viscus from the abdominal cavity. MacCormac says the term implies that the protruded viscus is covered with integument; hence a protrusion of viscera through a wound does not constitute a hernia. A hernia has three parts—the sac, the sac-contents, and the sac-coverings (Fig. 728). The *sac* is formed of peritoneum. A *congenital sac* is due to developmental defect, and may be in the inguinal region, the femoral region, the umbilical region, the lumbar region, or in the epigastric region. In the epigastric region it is a result of a congenital slit in the transversalis fascia. It used to be stated that femoral hernia was never congenital, but

[1] Ballance, in "Practitioner," April, 1898; H. Martyn Jordan, in "Lancet," Jan. 22, 1898.

Russell and Coley both say that a femoral hernia may have a congenital sac. In 100 necropsies Murray found 20 potential femoral sacs (sacs into which a hernia had not entered). An *acquired sac* is due to intra-abdominal pressure bulging the peritoneal covering of an abdominal ring and converting it into a pouch. The sac comprises a *body*, a *neck*, and a *mouth*. A sac once formed is almost certain to persist, because it adheres by its outer surface to surrounding parts, and hence the sac of a hernia is usually irreducible even when the contents are reducible. The *neck of the sac* is due to the constriction through which the sac passes; it becomes furrowed and folded, and the adhesion of these folds causes thickening and rigidity. Hernia of the bladder or of the cecum may have no sac or but a partial sac. A ventral hernia following an abdominal operation may be without a sac. The *contents of the sac* depend chiefly on the situation, a portion of the ileum being the usual contents. The colon, the stomach, the great omentum, the bladder, and other structures may enter the hernial sac. An *enterocele* contains only intestine; an *epiplocele* contains only omentum; an *entero-epiplocele* contains both omentum and intestine; a *cystocele* contains a portion of the bladder. The *coverings of the sac*, which vary with its situation, will be set forth during the consideration of special forms of hernia. In old hernia the layers are never distinct, fat and muscle waste, tissues adhere, and the skin stretches and atrophies. The sac of an old hernia occasionally becomes tuberculous, and the disease may remain local in the hernial sac or spread to the general peritoneum. Renault tells us that *tuberculosis of a hernia* is made manifest by increase in size, pain on pressure, and loss of body weight.

**Causes of Hernia.**—Hernia is a common trouble. According to Berger, in 1000 people 4.4 per cent. suffer from hernia. It occurs at all periods of life, and hereditary predisposition sometimes seems to exist. The male sex is three times as liable to hernia as the female sex. That increase of intra-abdominal tension is a common cause in children has been amply demonstrated. (See Hernia in Childhood, page 1158.) Excessive length of the mesentery has been assigned as a cause. In some instances a mass of fat forms (*fat hernia*) and advances before the hernia, and seems to bear a causative relation to it. Lucas-Championnière explains this as follows: When a person begins to take on fat, it is deposited not only under the skin, but also in the omentum, mesentery, and subperitoneal tissues. The semifluid fat is easily influenced by pressure. The deposit of fat within the abdomen lessens the size of that cavity, intra-abdominal pressure is increased, and subperitoneal fat protrudes at any weak spot in the wall. The protruding mass of fat adheres to and makes traction upon the peritoneum, and this membrane is drawn upon to form a sac, and the sac is surrounded by fat. This method of formation is frequently noticed in umbilical herniæ, and occasionally in inguinal herniæ. Any laborious occupation predisposes to rupture. Any condition which weakens the abdominal wall predisposes to rupture (muscular relaxation from ill-health, relaxation of abdominal walls following the termination of pregnancy, the removal of a large tumor or tapping for ascites, and wounds or abscesses of the abdominal wall). The commonly assigned cause is repeated muscular effort which increases intra-abdominal tension (straining at stool, coughing, lifting weights, jumping, the sexual act, and straining during micturition). In 25 per cent. of cases the cause is supposed to have been lifting or carrying a weight (Coley). I am satisfied that in some cases at least the external abdominal ring enlarges before it has been stretched by a descending hernia. Such a condition predisposes to hernia by weakening muscular support of the abdomen. A hernia may appear gradually or suddenly. Berger and Coley state that nearly 70 per cent. of herniæ in adult males appear gradually. The sac of an acquired hernia exists for a longer or shorter time before the hernia

enters it. The sac of a congenital hernia is present at birth. The sac of an acquired hernia forms gradually. A sac may exist for years and yet remain empty. When bowel or omentum enters it from some strain or effort, the parts were long prepared to receive the extruded mass. This extrusion may occur gradually or it may occur suddenly. If it occurs suddenly, the sufferer believes that his hernia was formed then and there, but, as a matter of fact, the extrusion of bowel or omentum and its entrance into the sac were but the last of a long series of antecedent and preparatory changes. Finally, a hernia appears, and often does so during effort. In rare cases traumatism may cause a hernia immediately, no sac existing before the accident. It does so in the inguinal region by stretching or tearing the internal ring, the inguinal canal at once enlarging. Such a condition is a true *traumatic hernia*, traumatism being the sole cause and not simply the exciting cause.

The old and erroneous idea was that a hernia was always formed by tearing of the peritoneum; hence the term *rupture*. This mode of formation is extremely unusual, but occasionally does occur. Coley saw such a case. An ordinary non-traumatic hernia, when the bowel suddenly and for the first time enters the sac, is the seat of some pain, but the pain is not disabling and the lump disappears on recumbency. In many cases the bowel or omentum gradually finds a way into the sac, and in such cases pain is usually trivial and may even be absent. In true traumatic hernia there are violent pain, collapse, vomiting, inability to walk and stand, and the mass does not return to the belly on recumbency, but must be reduced by taxis or operation. True traumatic herniæ may occur anywhere in the abdomen, but are most common in the inguinal region, where they are direct herniæ. (The relation born by accidents to the development of hernia is discussed by Paul Berger, in "Rev. de Chir.," April and May, 1906, and by Wm. B. Coley, in "Internat. Jour. of Surg.," Feb., 1908). All congenital herniæ are due to structural defects. Herniæ are divided clinically into *reducible*, *irreducible*, *incarcerated*, *inflamed*, and *strangulated*.

**Reducible Hernia.**—In this form of hernia the contents of the sac can be reduced into the abdominal cavity. At a known hernial opening the patient has a smooth enlargement (narrower above than below), which began to grow above and extended downward. A distinct neck can often be felt. In enterocele, straining, lifting, or standing enlarges the mass; the protrusion becomes smaller and may disappear on lying down; cough causes impulse or succussion, the protrusion is elastic, and may be tympanitic on percussion, and on reduction the mass suddenly disappears and there is a gurgling sound. In epiplocele the mass is often irregular and compressible, and feels boggy rather than elastic; muscular effort does not have much influence in enlarging it; impulse on coughing is slight; percussion gives a dull note, and reduction is accomplished gradually and produces no gurgling sound. In entero-epiplocele some parts of the mass are smooth, elastic, and perhaps tympanitic; others are dull on percussion, irregular, and flabby, but the diagnosis of this especial form from the other forms is often uncertain. The victims of reducible hernia complain of some pain on exertion, of dyspepsia, and often of constipation.

When a hernia is beginning to form there is often *premonotory uneasiness;* the patient complains of muscular pain in the lower abdomen, and this condition may exist for weeks or months before it is recognized that a hernia is present. An inguinal hernia can be recognized before it protrudes from the external ring. The tip of the finger is inserted in the ring and the patient is asked to cough. If a hernia has entered the canal, succussion will be detected on coughing. In a healthy man the external ring should admit the tip of the little finger, but not the end of the index-finger. If the end of the index-finger can be made to enter the ring that aperture is dilated, and even if there is no hernia in the canal, in future a hernia will probably descend. In a man, if the

surgeon desires to examine the ring, he inverts the skin of the scrotum over the finger and carries the finger to or in the ring. When the hernia first appears there may be pain, faintness, and some sick stomach, but often there is no pain or any discomfort.

**Treatment of Reducible Hernia.**—*Palliative Treatment.*—Prevent constipation and forbid sudden strains and violent exercise. If operation is refused or inadvisable, order a truss. The continued employment of a truss in young persons may bring about a cure. The day truss should be applied before rising in the morning and be removed after lying down at night, when a light truss should be substituted. A special truss is applied before bathing. In very fat people there is always trouble in adjusting a truss. A femoral hernia is more difficult to keep reduced than an inguinal hernia. In a hernia in which the gut is replaceable, but a portion of omentum is irreducible, it is difficult to maintain reduction of the gut with a truss, and an operation should be performed. In an oblique inguinal hernia the pad of the truss fits over the internal abdominal ring; in a direct inguinal hernia, over the external abdominal ring; in a femoral hernia, over the femoral ring at the level of Gimbernat's ligament. MacCormac's method of measuring for a truss is as follows: In either inguinal or femoral hernia start the tape from the *lower part* of the hernial opening, carry it up to the anterior superior iliac spine of the same side, then take it around the body, 1 inch below the crest of the ilium, to the other anterior superior iliac spine, and then to the upper part of the hernial opening.[1] A well-fitting truss will keep the hernia up even when the patient sits in a position to relax the abdominal walls and coughs and strains. A truss is always uncomfortable at first, but a person usually becomes accustomed to it. It should be kept scrupulously clean, and borated talc powder should be dusted upon the skin under the pad at least once a day. A truss which does not keep the hernia up or which causes pain does harm. Too strong a spring tends to enlarge the hernial orifice, and thus aggravates the case. Even after an apparent cure with a truss the instrument must be worn for a long time.

*Radical treatment of reducible and of non-strangulated hernia* seeks to obtain cure by plugging the mouth of the sac or by obliterating the canal of descent. Radical operations should be performed when a strangulated hernia is operated upon, in ordinary cases of reducible hernia, particularly if a truss is very painful or does not keep the bowel up, in most cases of irreducible hernia, and in any case of hernia in which there are occasional attacks of obstruction. It was formerly believed that a cure would fail if the subject was under three years of age, but Coley and others have proved that it is a very successful operation in childhood. It is rarely recommended under the age of four, because in two-thirds of the cases a truss will cure very young subjects. It is strongly advised in children after the age of four when a truss has failed, when there is irreducible omentum, or when there is a reducible hydrocele which prevents the truss from holding (Wm. B. Coley, in "Annals of Surgery," June, 1903). The radical operation is almost without danger in properly selected cases, and is one of the most successful of surgical procedures. We are justified in doing the operation upon an individual under fifty years of age and free from complications, purely to relieve him or her from the annoyance of wearing a truss. If, however, a patient is sixty years of age or over and a truss keeps the hernia up satisfactorily, the operation should not be performed unless it is demanded by some complication. Organic diseases of the heart, lungs, and kidneys are contra-indications. Enormous herniæ (Figs. 729 and 730) are unfavorable for operation. Restoration is difficult or impossible, the forcible handling produces much shock, and recurrence is to be expected.

[1] Treves's "Manual of Surgery," "Hernia."

Restoration is difficult or impossible because the abdominal cavity has contracted and holds with difficulty or cannot hold the huge hernia. As J. L. Petit said, the hernia has *forfeited the right of domicile* (Fig. 729). In an operation for an enormous hernia a great quantity of omentum will require removal, and it may be necessary to resect a considerable piece of intestine. If we decide to operate upon an enormous hernia, treat the patient some time before with the object of making him lose flesh. The absorption of mesenteric fat lessens intra-abdominal pressure. That operation may succeed in such cases is shown by Figs. 730 and 731. In any operation for the radical cure of inguinal hernia always remember that the *bladder* may be part of the hernia, and be on the lookout for it. Eggenberger's table of 6778 hernial operations shows 75 bladder herniæ (1 per cent.). As a rule, it is covered with cellular fat, which differs in color and consistence from omental fat and from other fat which may be found about a hernia. The presence of a quantity of extraperitoneal fat outside of the sac suggests the adjacency of the bladder and warns us not to tie off the sac very high up. It was the author's misfortune on two occasions to open a bladder in operating for inguinal hernia. In each case the bladder was sutured and both patients recovered. It has been estimated that the mortality after this accident, even when the bladder is sutured, is from 6 to 16 per cent. Among other possible accidents which may occur during hernia operations are: injury of an iliac vessel, of a femoral vessel, or of an epigastric vessel.

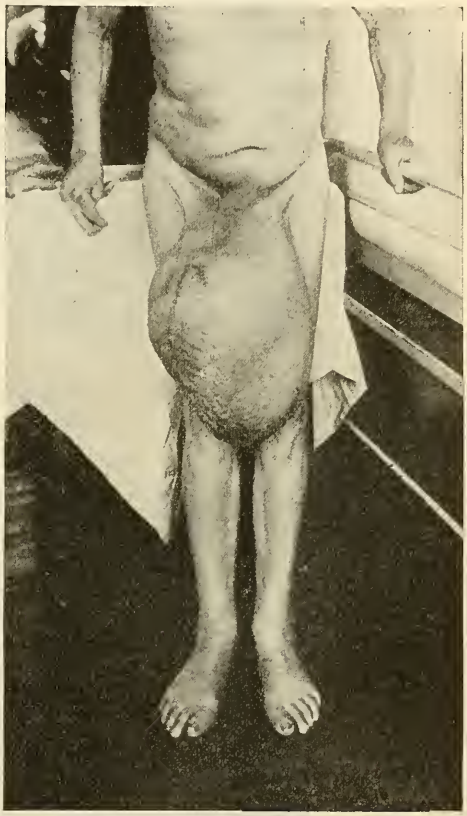


Fig. 729.—Hernia which has "forfeited the right of domicile."

The success of an operation for the radical cure of a hernia depends upon the attainment of primary union. Primary union is favored by thorough cleanliness; by wearing gloves while operating; by cutting the parts with a sharp knife instead of tearing them with a dissector; by removing some fat and any superfluous tissue fragments; by tying the stitches firmly, but not tightly (a tight stitch causes necrosis and creates a point of least resistance); by careful closure; by dressing with pressure; and by keeping the patient recumbent for from fifteen days to three weeks.

A truss is not to be used after operation. Ten years ago Wm. B. Coley ("Annals of Surgery," June, 1903) had operated upon 1075 cases of inguinal and femoral hernia. In his report he did not consider operations performed within the preceding six months, and so presented a study of 1003 cases. Of these, 937 cases were inguinal, 66 cases were femoral. In the 1003 cases, 647 were traced and were found well from one to eleven years after operation; 705

were well from six months to eleven years; 460 were well from two to eleven years. If the patient is well one year after operation, he will probably remain well. This is proved by Coley's study of relapses, an investigation which shows that

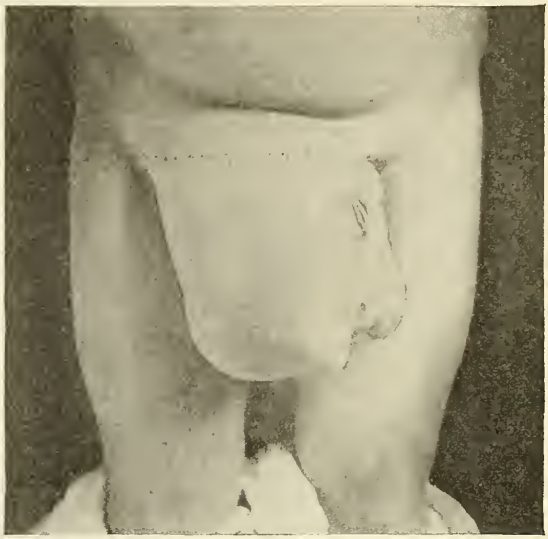


Fig. 730.—Oblique inguinal hernia of large size (duration, sixteen years).

65 per cent. of relapses occur within six months of operation and 80 per cent. within the first year. Only 13⅔ per cent. occur from one to two years, and only

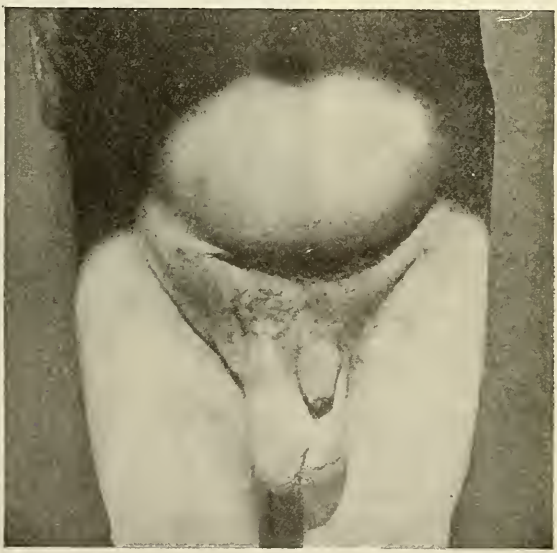


Fig. 731.—The case shown in Fig. 730 six months after operation.

6⅔ per cent. after two years. Coley had 2 deaths in 1075 cases (less than ⅕ of 1 per cent.). After Bassini's operation there are about 1 per cent. of relapses. Coley reports that from Dec., 1891, to Jan., 1909, there were per-

formed by Drs. Bull, Walker, and himself, in the Hospital for Ruptured and Crippled, 2384 operations for the radical cure of hernia. Of these, 2218 were inguinal (only 445 in females). In the 1773 male cases the typical or modified Bassini operation was done with 12 relapses, or .68 per cent. ("Progressive Medicine," June, 1909).

*Lannelongue's Method.*—Lannelongue has for certain cases returned to the old injection plan, using a 10 per cent. solution of chlorid of zinc instead of white oak bark. The hernia is first reduced and is held up by an assistant, who closes the internal ring with a finger and also holds the cord aside. Several injections of 10 min. each are thrown in the region of the internal pillar, the region of the external pillar, and into the canal behind and outside of the cord. The surgeon must be careful that no zinc solution escapes into the subcutaneous tissue. The effect of the chlorid of zinc is to cause the formation of quantities of fibrous tissue. It is scarcely to be expected that a cure so produced will be permanent in an adult, though it may be in a child.

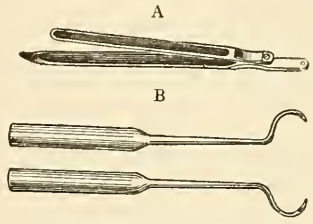



Fig. 732.—A, Hinged hernia director; B, hernia needles.

*Macewen's Operation for Inguinal Hernia.*—A hernia director (Fig. 732, A) and special hernia needles (Fig. 732, B) are required for this operation. The

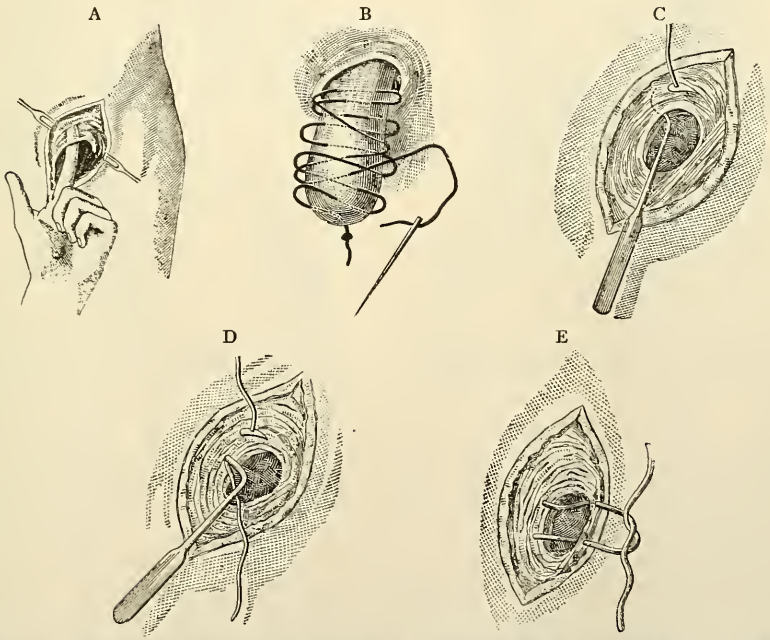



Fig. 733.—Macewen's operation for radical cure of inguinal hernia: A, Stripping of the sac: B, purse-string suture; C, fastening the purse-string suture; D, passing, and E, tying, the sutures for the internal ring.

patient lies recumbent, the thigh being abducted and partly flexed and resting on a pillow beneath the knee. The bowel is reduced, and an incision 3 inches long is made in the direction of the inguinal canal, the center of the incision corresponding to the external ring. The sac is freed from its attachments below and is lifted up. The surgeon introduces a finger into the inguinal

canal and separates the sac from the cord and from the walls of the canal, and then carries the finger through the internal ring and separates the peritoneum for 1 inch about the periphery of this aperture (Fig. 733, A). A chromicized catgut stitch is fastened to the lowest portion of the sac, and is passed through the sac several times, so that pulling on the stitch will purse the sac (Fig. 733, B). The free end of this stitch is carried through the internal ring into the belly, and is pushed out through the abdominal muscles 1 inch above the internal ring, the skin being pushed aside so as to escape perforation by the needle. The thread is tightened so as to fold up the sac and pull it into the belly. This plugs the ring. The thread is handed to an assistant to keep tight until the sutures are introduced into the ring, when the sac is permanently anchored by taking several stitches in the external oblique muscle. A strong catgut suture is passed with a Macewen needle through the conjoined tendon from below upward, the ends of this suture being carried through Poupart's ligament and the outer border of the internal ring from within outward. This suture is tightened and closes the internal ring. The external ring is sutured and the skin is stitched (Fig. 733, C, D, and E).

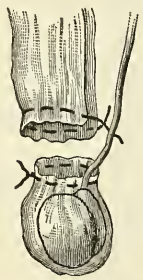


Fig. 734.—Macewen's operation for the radical cure of congenital hernia.

In congenital hernia the sac is divided in its middle, and the lower part is closed by stitches of chromicized catgut, forming a tunica vaginalis. The upper part of the sac is slit posteriorly to permit the escape of the cord, and is closed by stitches of chromicized catgut (Fig. 734). The operation is finished as in the acquired form. After Macewen's operation the patient should stay in bed for at least three weeks, and must not work for eight or nine weeks. Workmen after this operation should always wear for a time a pad and a spica bandage. Children require no pad. Never apply a truss, as strong pressure will produce atrophy of the curative scar.

*Bassini's Operation for Oblique Inguinal Hernia.*—(See E. Wyllys Andrews, in "Med. Record," Oct. 28, 1899, who describes from personal observation how Bassini does his operation. I have drawn upon his description in the following section.) Bassini's operation displaces the spermatic cord from the old canal and places it in a new canal, and this new canal is oblique. Curved and rounded needles are employed to insert the stitches. The suture material is kangaroo-tendon or chromicized catgut. Silk or silver wire is apt to make trouble—it may be long after the operation. The patient is placed supine with the thighs extended. An incision is made parallel to Poupart's ligament and extending from the external ring to a point external to the internal ring. The incision is about $1\frac{1}{2}$ inches above the ligament and is from 5 to 7 inches in length. By this incision the aponeurosis of the external oblique and the pillars of the external ring are exposed. All bleeding is arrested, the aponeurosis is incised in the direction of its fibers and from above downward, and the inguinal canal is opened. The aponeurosis of the external oblique is dissected up with a blunt instrument until Poupart's ligament is exposed. We speak of this ligament as the shelf. A mass containing the sac of the hernia, the cord, the cremaster muscle, and considerable fat is lifted up. Bassini employs blunt dissection. Coley advocates the use of the knife. Masses of fat and usually the cremaster muscle are removed. The sac is isolated first at its neck and the neck is stripped from the inner aspect of the internal ring for the distance of $\frac{4}{5}$ inch. The object of this stripping is to permit the removal of the sac at a high level. High removal obviates the leaving of a funnel-shaped depression of peri-

toneum. Such a depression would predispose to relapse. The sac is opened at the fundus, the interior is investigated, and if the contents are reducible, they are restored to the abdominal cavity and the neck of the sac is clamped high up. If adherent masses of omentum are found, the adhesions are separated, bleeding is arrested, and the omentum is restored to the abdomen unless it is in a hard and thick mass, when it is tied off and removed. Bassini ties off the neck of the sac above the clamp with a strong ligature of silkworm-gut. If the sac is large and thick, he also threads both ends of a ligature upon a needle, passes the strand through the stump, and ties around over the first loop. (See E. Wyllys Andrews, "Med. Record," Oct. 28, 1899.) Coley and many other operators prefer to tie off the sac with a catgut suture rather than with silkworm-gut or silk. It is my usual custom to employ fine black silk, catching it to prevent slipping by running a stitch through the wall of the neck of the sac. After ligating the neck of the sac the sac is cut across and removed. The cord is now lifted out of the way (Fig. 735, A), the inner surface of Poupart's ligament is exposed by retraction, and the deep sutures are passed (Fig. 735, A). Bassini uses silk which has been boiled in glycerin. Most

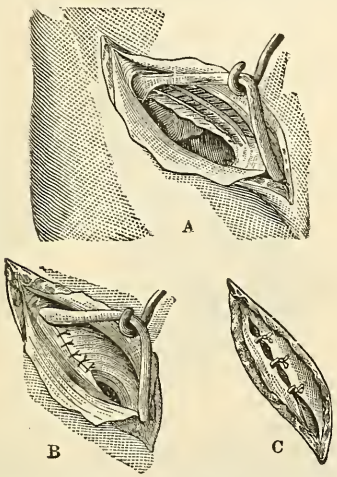



Fig. 735.—A–C, Bassini's operation for the cure of inguinal hernia (Esmarch and Kowalzig).

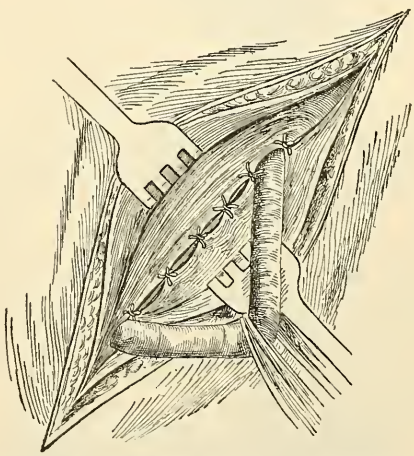


Fig. 736.—Bassini's operation (deep sutures), showing extra suture above the cord, as advised by Coley.

American operators use kangaroo-tendon or chromicized catgut. Bassini inserts first the sutures nearest to the pubes. The first suture—and sometimes also the second—includes part of the rectus sheath and rectus muscle. Each stitch includes the internal oblique and transversalis muscle in the upper edge and the shelf of Poupart's ligament below the lower margin, and from four to six stitches are passed behind the cord (Fig. 735, B). The last stitch narrows the internal ring so that it fits tightly around the cord (E. Wyllys Andrews, Ibid.). Coley's rule for passing this suture is to insert it so "that it just touches the lower border of the cord when the latter is brought vertically to the plane of the abdomen" ("Annals of Surgery," June, 1903). He always places a suture above the cord, and believes it tends to prevent relapse (Fig. 736). The sutures are tied from above downward. The cord is laid upon this new floor and the aponeurosis of the external oblique is sutured over it (Fig. 735, C). I close the aponeurosis by a continuous suture of chromic catgut and the skin with interrupted sutures of silkworm-gut or fine silk. Drainage is not used. The wound is covered with a roll of iodoform gauze and some pieces of sterile gauze, and compression is made by strips of adhesive plaster, and a piece

of adhesive plaster run from one thigh to the other acts as a shelf for the testicles to rest upon. The adhesive plaster is overlaid with dry gauze, and this is covered with absorbent cotton and the dressing is retained in place by a firm spica of the groin (*Coley's dressing*). The wound is dressed on the seventh or eighth day, and the patient is kept in bed for two weeks and is allowed to get about in two and one-half to three weeks, wearing a bandage until four weeks after operation.

In Bassini's operation some surgeons treat the sac as in Macewen's operation, carrying out the rest of the procedure as directed above. In a pure Bassini operation the funnel-shaped depression in the peritoneum at the point of emergence of the cord may remain and predispose to hernia, but the use of Macewen's plan for treating the sac obviates this.

*Halsted's Old Operation* (as described by J. C. Bloodgood, in "Johns Hopkins Hosp. Report," vol. vii).—The skin incision is not parallel to Poupart's ligament, but at an angle of 25 degrees to it (Fig. 737). Poupart's ligament is

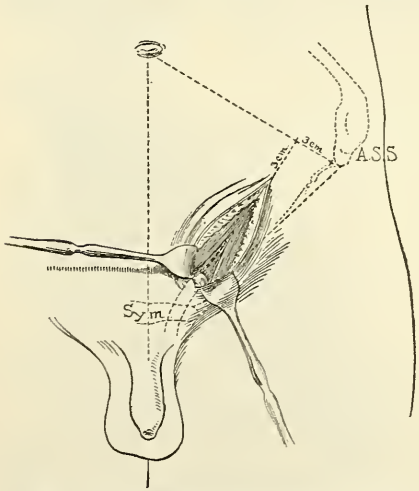



Fig. 737.—The skin incision, retractors in the lower angle of the wound dislocating the opening in the skin and subcutaneous fat downward, exposing the aponeurosis of the external oblique and external ring. The dotted line within the wound represents the direction of the division of aponeurosis of external oblique (Bloodgood).

Fig. 738.—The aponeurosis of external oblique has been divided and retracted, uncovering the internal oblique muscle and inguinal canal. The lines on the muscle represent the direction and extent of the division. The dotted line in the inguinal canal is the direction and extent of the division of the coverings of sac (Bloodgood).

well exposed to within 2 cm. of the pubic spine. The aponeurosis of the external oblique muscle is divided. Free the lower border of the internal oblique muscle and divide the edge of the muscle at a right angle to its fibers (Fig. 738), and as far as possible from the linea semilunaris. The coverings of the sac near the neck are picked up with mouse-toothed forceps and are divided. The division of the fasciæ is continued from the neck of the sac downward toward the pubes. The sac is then lifted from the inguinal canal and it brings with it "the larger bundle of veins and the vas deferens" (Fig. 739). The sac is separated from the veins and the vas with a knife or scissors, and the separation is carried to and beyond the neck of the sac. In "*certain cases* the larger bundle of veins is separated from the vas deferens, ligated, and excised" (Fig. 740). Whether the veins are excised or not, the sac is opened, its contents reduced, the opening into the peritoneal cavity closed with a continuous silk suture, and the excess of sac excised. During the entire operation the vas and its vessels "should be handled very little, and should not be torn from their bed

in the inguinal canal." Every point of bleeding should be ligated. At this stage the vas is gently picked up and a blunt-pointed hook is used to tear the mesocord. The freed vas is lifted into the upper angle of the divided internal oblique muscle, and is held there until the sutures are inserted. The deep

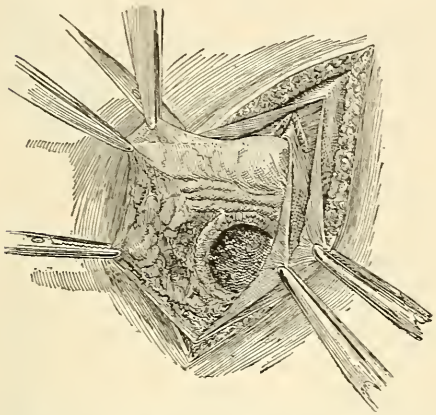


Fig. 739.—The internal oblique muscle and the coverings of the sac have been divided, the sac with the veins and vas deferens are drawn out of the wound preparatory to the excision of the sac and the ligation and excision of the veins (Bloodgood).

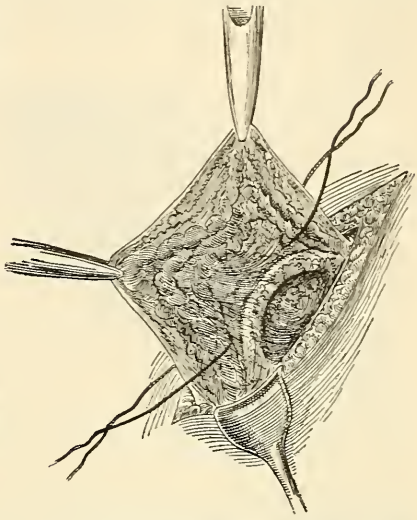


Fig. 740.—The method of excision of veins in operations for hernia and variocele. The vas deferens and its "immediate" vessels and the mesocord have not been disturbed (Bloodgood).

sutures of silver wire are next inserted. Usually five are needed. The upper one is passed first. These sutures are shown in Fig. 741. The cord emerges

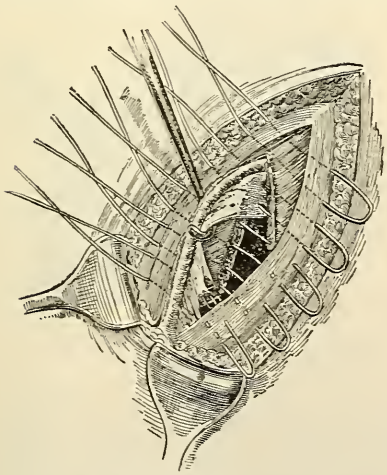


Fig. 741.—The insertion of the deep silver wire sutures, one above and four below the cord. The veins have been ligated and excised. The mesocord has been torn gently in its center only (Bloodgood).

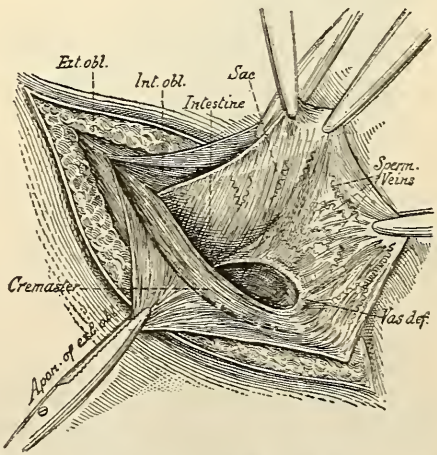



Fig. 742.—Exposure of the sac, the vas, and the spermatic veins (Halsted).

from the cut in the internal oblique muscle between the first and second sutures. Sutures No. 1 and No. 2 pierce the mesocord, but care is taken to see that they

do not injure the vas or its vessels. Each suture is drawn upon and twisted about six times. The cut and twisted ends are caught by forceps and turned in. The skin wound is closed with a subcuticular stitch of silver wire. It is covered with silver-foil and dry gauze, and often a plaster-of-Paris bandage and splints are used, "the splints extending from just above the knee to near the costal margins."

*The Modified Halsted Operation.*—The operation at present performed by Professor Halsted and his assistants has been evolved from the former operation so long associated with his name, and has been greatly modified by him and by Dr. Bloodgood. In this operation the skin and the aponeurosis of the external oblique are incised exactly as in performing Bassini's operation, and flaps of aponeurosis are raised. Next, the cremaster muscle and the cremaster fascia are incised in a line slightly above the center of the spermatic cord. The internal oblique muscle is then brought into distinct view at the side of the inguinal canal and the hernia is carefully inspected (Fig. 742). If the veins are found to be large they should be excised; but the surgeon does not lift the vas from its bed, and even avoids touching it, if he possibly can, for fear that thrombosis may occur in its veins. The veins are tied above, well up in the abdomen; and below, well above the testicle, and excised between the ligatures. The sac is then ligated or sutured with a purse-string suture. One end of the thread that ties or sutures the sac is carried, by means of a long, curved needle, in an outward direction under the internal oblique muscle, through which it is then pulled. The other end of the thread is also pulled through the muscle $\frac{1}{2}$ inch from the first end, and these two ends are tied together. It will be observed that this treatment of the neck of the sac is somewhat similar to the method practised by Kocher.

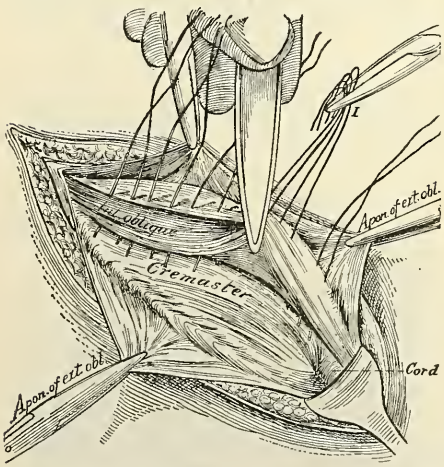



Fig. 743.—Suture of the cremaster to the internal oblique (Halsted).

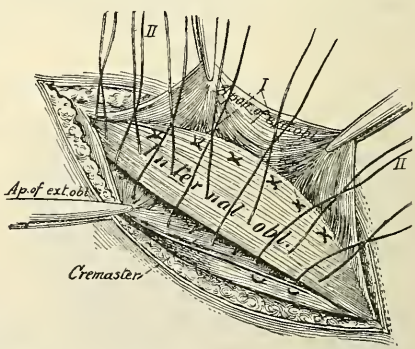



Fig. 744.—Suture of the lower edge of the internal oblique to Poupart's ligament (Halsted).

The next step is to carry the inferior flap, composed of cremaster muscle and fascia, under the internal oblique muscle, and suture it there (Fig. 743). We next suture the internal oblique muscle and the conjoined tendon to Poupart's ligament, the lower edge of the internal oblique being tucked under the edge of the ligament (Fig. 744). In order to accomplish this, it may be necessary to release the muscle by incising the anterior rectal sheath. The incision in the external oblique is now closed with sutures that overlap the margins (Figs. 745 and 746), and the skin wound is also closed.

*Halsted's Operation Plus Bloodgood's Method of Transplanting the Rectus Muscle.*—(See Jos. C. Bloodgood, in "Johns Hopkins Hosp. Reports," vol.

vii.) When the conjoined tendon is very thin or obliterated, the ordinary operation is not enough. Insufficiency of the conjoined tendon is known to exist when a finger does not meet any obstruction after passing through the

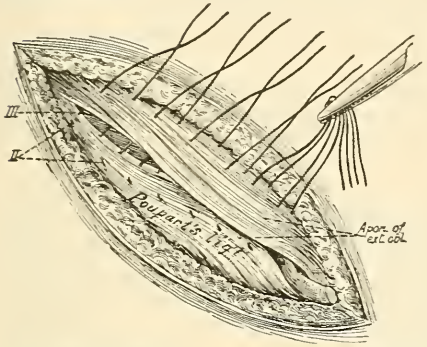



Fig. 745.—Suture of the aponeurosis of the external oblique (Halsted).

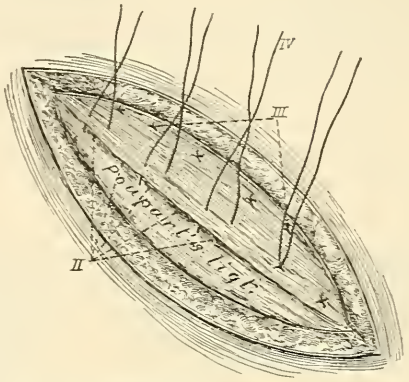



Fig. 746.—Suture of the margin of aponeurosis to Poupart's ligament (Halsted).

external abdominal ring, but can be introduced for some distance into the abdominal cavity (Bloodgood). To meet this condition of affairs, Bloodgood devised "a plastic operation on the rectus muscle, bringing this muscle down

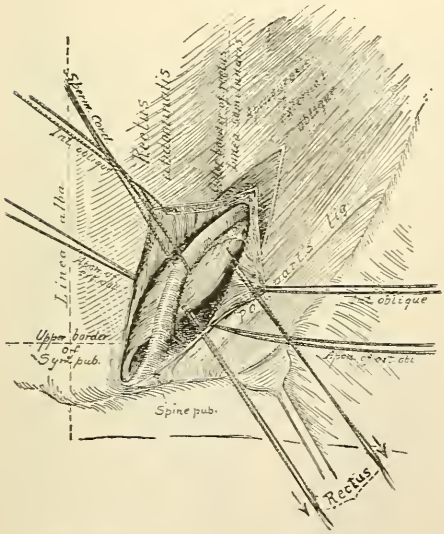



Fig. 747.—The method of transplanting the rectus muscle. The sac has been excised and the peritoneal cavity closed; internal oblique muscle has been divided, the rectus exposed and transplanted; at this stage the wound is ready for the deep sutures. This illustration shows how perfectly the transplanted rectus muscle lines the lower half of the wound (Bloodgood).

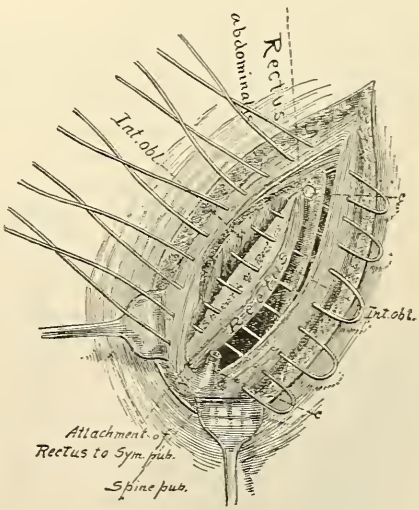



Fig. 748.—The transplanted rectus included by the deep sutures. In this illustration the cord has been excised in order to demonstrate the operation more clearly (Bloodgood).

and suturing it with the other available tissue to Poupart's ligament and to the aponeurosis of the external oblique from the arch of the pubis up to the position of the transplanted cord" (Bloodgood), in previously mentioned re-

port). The first steps of the operation are identical with those previously described, but before the insertion of the deep stitches the rectus sheath is exposed and divided in the direction of the muscle-fibers, from the pubic insertion upward for 5 cm. The muscle bulges from the cut and is caught with silk sutures (Fig. 747). Deep sutures are now introduced as in Halsted's operation, except that they include the rectus and its sheath (Fig. 748). The operation is completed as is Halsted's. I have performed this operation a number of times with entire satisfaction.

*Kocher's Operation.*—Kocher exposes the aponeurosis of the external oblique, makes a small incision through the aponeurosis above and external to the internal ring, and draws the sac through this incision and sutures it in place.

*Fowler's operation* is as follows: An incision is made parallel with Poupart's ligament from the spine of the pubis to the level of the internal ring, and a flap is turned up. The inguinal canal is opened and the sac and cord are isolated. The sac is opened, its contents reduced, it is cut off, and its edges grasped with forceps. The deep epigastric artery and vein are sought for, each is tied in two places and divided between the ligatures. The index-finger is introduced into the belly, and on this as a guide the floor of the canal is divided (transversalis fascia, subserous tissue, and peritoneum). The cord is placed in the peritoneal cavity. The edges of the opening are sutured so that broad serous surfaces are approximated, through-and-through sutures being passed from side to side. The cord is brought out at the inner end of the incision, the lower angle of the cut being at such a level that the cord curves upward and forward as it leaves the abdomen. The inguinal canal, the gap in the aponeurosis, and the skin wound are closed.[1]

*Ferguson's Operation.*—In studying a number of recurrences after operation A. H. Ferguson observed that a hernial protrusion is apt to return at the upper and outer portion of the scar, above the cord and near Poupart's ligament. When he operated upon relapsed cases, he discovered a slit of the aponeurosis of the external abdominal wall, through which the sac and some fat protruded. In order to determine the cause of the failure of these operations, he thought it proper to make a semilunar incision and raise a flap of skin, fascia, and aponeurosis of the external oblique. On doing this, he was surprised to find an angle between the lower border of the internal oblique muscle and the inner aspect of Poupart's ligament absolutely unprotected by the internal oblique or the transversalis muscle. In some cases this angle extended upward and outward to the anterior superior iliac spine. He, therefore, concluded positively that the cause of a rupture returning in this angle after an operation for radical cure is deficient origin of the internal oblique muscle and of the transversalis muscle at Poupart's ligament. He is now persuaded that in all cases of hernia there is a deficient origin of these muscles, and he has demonstrated the same thing in a series of dissections in the inguinal region. Ferguson describes his operation as follows ("Jour. Am. Med. Assoc.," July 1, 1899): He begins his incision over Poupart's ligament, $1\frac{1}{2}$ inches below the anterior superior iliac spine, carries it inward and downward in a semilunar curve, and terminates it over the conjoined tendon, near the pubic bone. The incision goes down to the aponeurosis of the external oblique, and the flap, with its fat and fascia, is turned downward and outward (Figs. 749 and 750). The next step is to incise the external abdominal ring to the intercolumnar fascia and separate the longitudinal fibers of the external oblique over the inguinal canal to beyond the internal ring, at a point nearly opposite the anterior superior spine of the ilium. Any transverse fibers that may be encountered are severed. The separated aponeurosis of the external oblique muscle is then retracted. One has then

[1] "Annals of Surgery," Nov., 1897.

brought into view the contents of the inguinal canal, the hernial sac and its adhesions, the spermatic cord, the ilio-inguinal nerve, the internal abdominal ring, the subserous fat, the cremaster muscle, the conjoined tendon, the internal oblique and its deficient origin at Poupart's ligament, the transversalis fascia, and the internal surface of Poupart's ligament. The sac is now dissected from

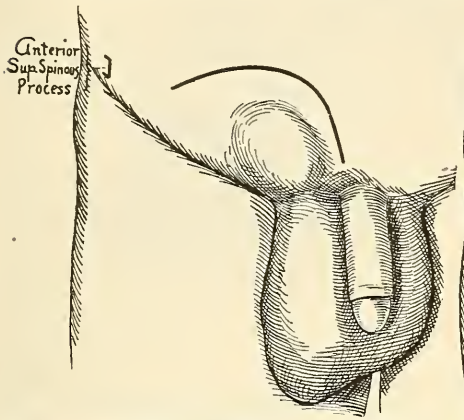



Fig. 749.—Ferguson's operation: The semilunar skin incision ("Jour. Am. Med. Assoc.").

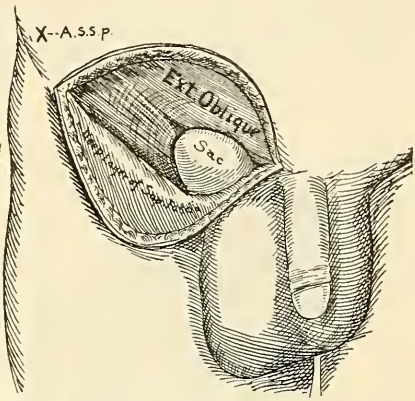



Fig. 750.—Ferguson's operation: Flap turned back exposing the aponeurosis and the sac of the hernia ("Jour. Am. Med. Assoc.").

the cord and the internal ring. It is opened and its contents are inspected and properly dealt with. It is tied high up and cut off, and the stump is dropped into the abdomen (Fig. 751). If the sac is congenital it is divided into two parts: the distal portion is used to make a tunic for the testicle and the proximal

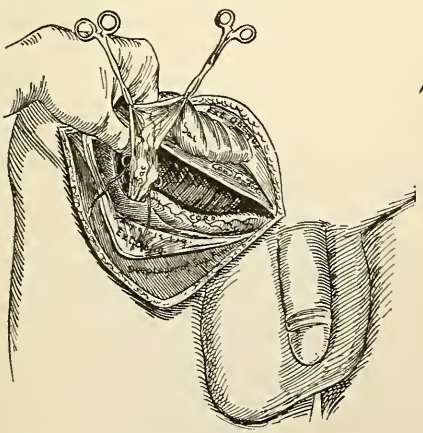


Fig. 751.—Ferguson's operation: Dealing with the sac and its contents ("Jour. Am. Med. Assoc.").

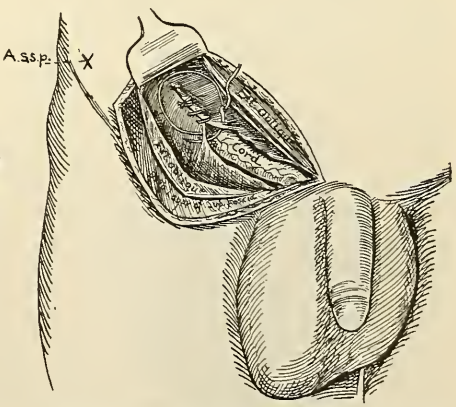



Fig. 752.—Ferguson's operation: Suture of the slack in the transversalis fascia ("Jour. Am. Med. Assoc.").

portion is treated as above directed. The cord is not disturbed, and it is beyond doubt that Ferguson is right in saying that the testicle frequently comes to harm after operations that disturb the cord. The veins in the cord should not be touched unless a varicocele also exists. Any excessive quantity of subserous adipose tissue should be removed. The next step in the operation is

to restore the structures to their normal position; and one should remember that in the transversalis fascia is the internal ring. In hernia the internal ring is large and the transversalis fascia bulges outward; one must, therefore, take up the slack in this fascia and make a well-fitting ring for the cord by means of a catgut suture, either interrupted or continuous (Fig. 752). After this has been accomplished the internal oblique and transversalis muscle are sutured to the internal aspect of Poupart's ligament, after the lower borders of the muscles have been freshened and Poupart's ligament has been scarified. The sutures must be carried two-thirds of the way down Poupart's ligament, which is about the normal origin of this muscle in the female (Fig. 753). The next step is to suture the edges of the divided aponeurosis of the external oblique; this restores the external abdominal ring. The skin-flap is then carefully sutured.

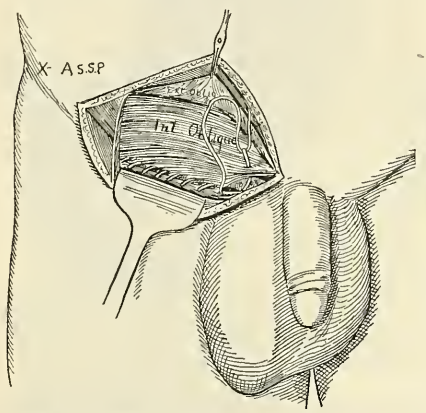



Fig. 753.—Ferguson's operation: Suture of the internal oblique and of the transversalis muscle to the internal aspect of Poupart's ligament ("Jour. Am. Med. Assoc.").

*Radical Cure of Direct Inguinal Hernia.*—If the hernia goes through the conjoined tendon or pushes that structure before it, the operation should consist in transplanting the rectus muscle as practised by Bloodgood (see page 1145) and suturing the arched fibers of the internal oblique and conjoined tendon to Poupart's ligament and beneath the cord.

If the hernia passes around the outer edge of the conjoined tendon an overlapping operation, like the Mayo operation for umbilical hernia, should be performed (G. G. Davis, in "Annals of Surgery," Jan., 1906).

*Radical Cure of Umbilical Hernia.*—The results of operations for umbilical herniæ have not been satisfactory. Recurrences are frequent. This is probably due to the fact that most of the subjects are fat, and that the muscles are thin and flabby. The usual operation may be thus described: Make a longitudinally elliptical incision through the skin around the mass. Endeavor to separate the sac from the superficial tissue. If this cannot be done, open the sac and separate it from the contents. Even if the sac can be stripped from the skin, always open it and separate the contents. Return any bowel which may be present, and do not forget that there may be a small portion of bowel completely encased in omentum. Tie into segments and cut off the superfluous omentum and return the stump into the belly. Excise the umbilicus (*omphalectomy*). Suture the peritoneum with a continuous catgut suture. Close the musculofascial wall with two layers of interrupted sutures of kangaroo-tendon or chromic catgut, or one layer of silver wire mattress sutures. Close the skin by interrupted sutures of silkworm-gut or a subcuticular stitch.

*Mayo's Operation.*—This is a vast improvement on the older operation. It gives a firm cicatrix free from disastrous traction. Mayo believes that the defect in the old operation is that the recti muscles are naturally separated at the level of the umbilicus, and in bringing the recti together we have virtually performed muscle transplantation, and these thin muscles are of no great value in preventing relapse, and in a large hernia it is not even possible to cover the gap by muscle. Mayo now operates as follows: Transverse elliptical incisions are made around the umbilicus and hernia and the base of the protrusion is exposed (Fig. 754). The surface of the aponeurosis is cleared for $1\frac{1}{2}$ inches around the neck of the sac. The fibrous and peritoneal coverings of the hernia

are divided by a circular incision around the neck of the sac. Intestine is freed from adhesions and placed within the abdomen. Omentum is ligated and removed with the sac. The margins of the ring are grasped and overlapped in order to indicate in which way it can be most easily done; thus is the direction of the closure indicated. An incision is made through the fibrous and peritoneal coverings of the ring 1 inch or more transversely on each side, and

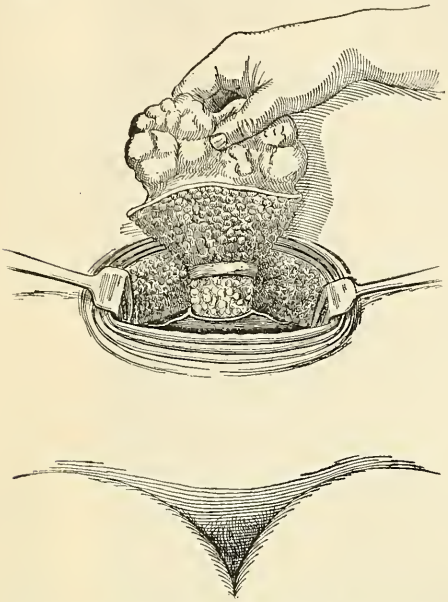


Fig. 754.—Mayo's operation for the radical cure of umbilical hernia: Exposure of hernia and lateral incision.

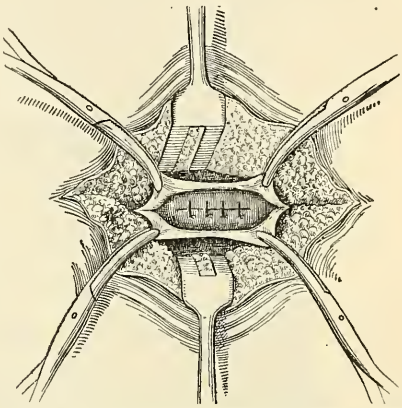


Fig. 755.—Mayo's operation for the radical cure of umbilical hernia: Peritoneum sutured.

the peritoneum is stripped from the under surface of the upper flap. Several mattress sutures of silver wire are introduced 1 inch above the edge of the upper flap and are carried through the margin of the lower flap; sufficient traction is made to permit of the closing of the peritoneum with a continuous catgut

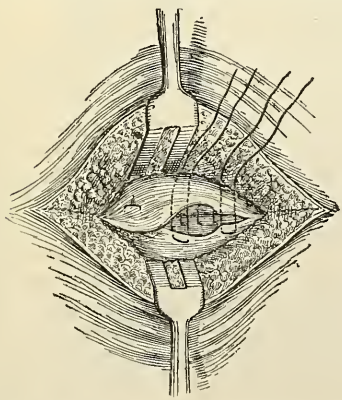


Fig. 756.—Mayo's operation for the radical cure of umbilical hernia: Aponeurosis sutured.

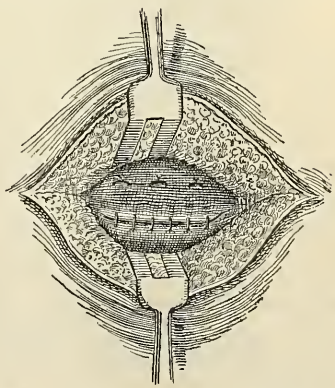


Fig. 757.—Mayo's operation for the radical cure of umbilical hernia: Aponeurosis sutured second time with gut sutures.

suture (Fig. 755). When this has been accomplished, the silver wire sutures are drawn so as to slide the lower flap into the pocket between the peritoneum and the under surface of the upper flap (Fig. 756). The free margin of the

upper flap is fixed by catgut sutures to the aponeurosis (Fig. 757), and the superficial incision is closed as usual. Wm. J. Mayo ("Jour. Am. Med. Assoc.," June 1, 1907) reported upon 88 operations for umbilical hernia by this method between 1894 and 1905; 75 were traced; 1 had a partial relapse; 1 was supposed to have a relapse, but operation disclosed a second opening above and outside of the closed umbilical opening.

*Radical Cure of Femoral Hernia.*—Cheyne ligates the neck of the sac, stitches the stump to the abdominal wall, dissects out a flap from the pectineus muscle, stitches this flap to Poupart's ligament and to the abdominal wall, and thus fills up the crural canal. Bassini makes an incision parallel to Poupart's ligament, ties the neck of the sac, cuts off the sac below the ligature, and returns the stump into the belly. He attaches by deep sutures Poupart's ligament to the pectineal aponeurosis as high up as the pectineal eminence, the cord or round ligament being drawn out of the way. Superficial sutures are passed between the pubic portion and the iliac portion of the fascia lata.

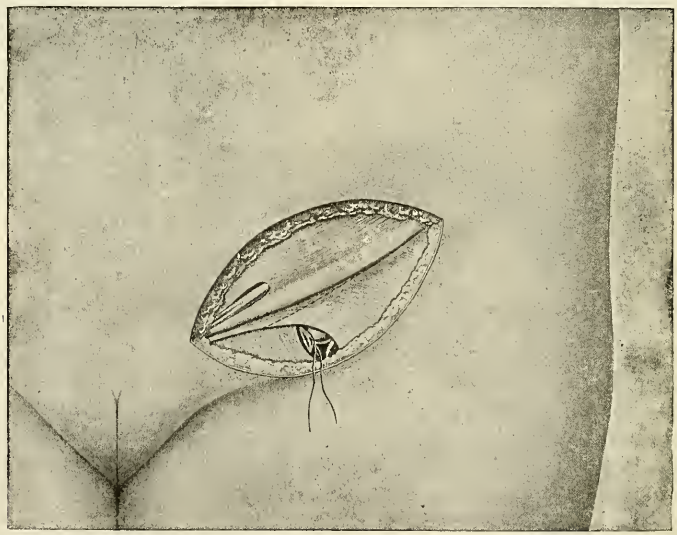


Fig. 758.—Fabricius's operation for the radical cure of femoral hernia: Neck of sac shown. Sac cut away. Dotted line shows line of separation of Poupart's ligament and fascia lata (Fowler).

The *operation of Fabricius* is very satisfactory. It is performed as follows: An incision is begun over the pubic spine and is carried outward for 5 inches parallel with Poupart's ligament. The sac is exposed, isolated, and opened, its contents are reduced, its neck is ligated, the sac is cut off, and the stump is dropped back (Fig. 758). An incision is now made below Poupart's ligament so as to separate this structure and the fascia lata, and the flap of fascia is turned down (Fig. 759). The crural sheath and the vessels are retracted outward. The surgeon is careful not to injure the obturator artery and vein. The origin of the pectineus muscle is sutured to Poupart's ligament. The lower stitches include the periosteum of the horizontal ramus of the pubes as well as the beginning of the muscle (Fig. 760). Care must be taken in passing certain of them to avoid injuring the deep epigastric vessels. When these stitches are tied, the femoral canal is obliterated. The flap of fascia lata is sutured to the aponeurosis of the external oblique, and the skin is sutured.

*Operative Treatment of Adherent (Sliding) Hernia of the Ascending and Descending Colon.*—My personal experience consists of 8 cases of right and 2

cases of left inguinal hernia. The sac is deficient posteriorly and externally (see page 1161). In order to restore the bowel into the abdomen many opera-

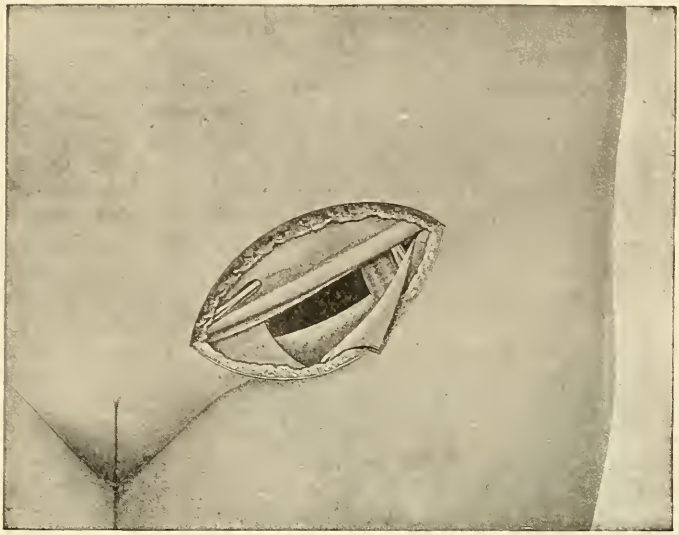


Fig. 759.—Fabricius's operation for femoral hernia: Fascia lata turned back, exposing crural sheath and origin of pectineus muscle (Fowler).

tors have sought to force up the adherent intestine to the external ring, and others have stripped the gut from the subperitoneal tissues in order to per-

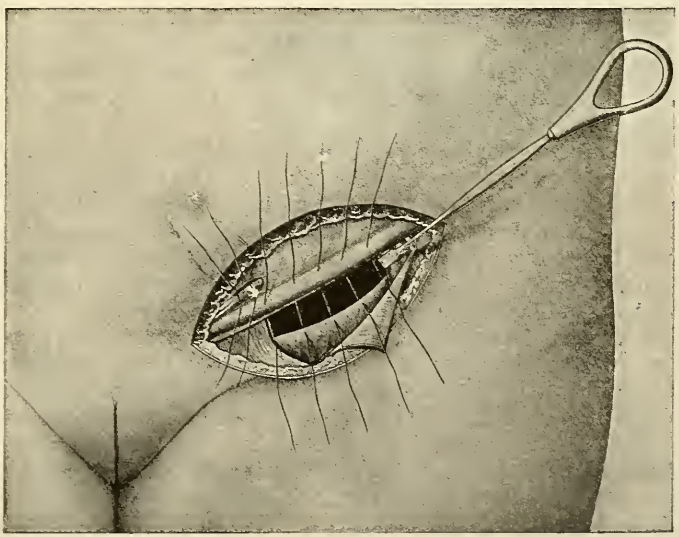


Fig. 760.—Fabricius's operation for femoral hernia: Crural sheath and vessels retracted and kangaroo-tendon sutures applied to Poupart's ligament and origin of pectineus, ready for tying. Two sutures are placed in position to approximate the pillars of the external ring (Fowler).

mit of reduction. The first plan should never be followed. If it should be employed, sutures will fail to hold the bowel up. The second plan is risky and may be followed by gangrene of the bowel. In my cases I followed

Weir's plan ("Med. Record," Feb. 24, 1900), and, after dissecting up the peritoneum on each side to a little above the internal ring, freed the bowel from its bed and covered the new surface with the peritoneal flaps (Fig. 761). The bowel was then restored and a radical cure was made.

**Irreducible Hernia.**—The swelling in irreducible rupture presents the usual evidences of hernia, imparts an impulse on coughing, but cannot be replaced in the abdomen. Sometimes a portion is reducible and a portion is irreducible. A hernia may become irreducible because of the size of the mass, because of adhesions, or because of excessive growth of omental fat. An irreducible hernia is liable to be bruised and to cause much distress and pain, and is always a menace to life because of the danger of obstruction and strangulation. It was formerly the custom to support a small irreducible hernia by a hollow, padded truss, but now operation should be advised. A large hernia of this variety, if operation is refused, must be carried in a bag truss. The patient must not take very active exercise, must keep the bowels regular, and must live upon a plain diet. Most cases of irreducible hernia should be treated by operation.

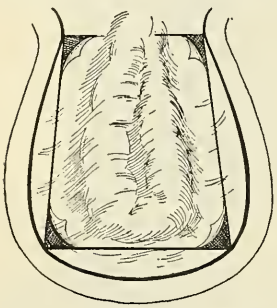


Fig. 761.—Outline of peritoneal lining of sac utilized as a flap to cover posterior surface after it has been freed by dissection (Weir).

**Incarcerated or Obstructed Hernia.**—Obstruction takes place by the damming up of feces or of undigested food, the fecal current being arrested, but the blood-current in the wall of the bowel not being cut off. Incarceration is commonest in irreducible hernia, especially umbilical hernia, and during the existence of constipation. The hernia enlarges and becomes tender, painful, and dull on percussion; pressure may diminish it somewhat in size. It remains irreducible, but still presents impulse on coughing. The abdomen is somewhat distended and painful; there are nausea, constipation, and not unusually slight vomiting. Constitutional disturbance is trivial and constipation is not absolute, gas at least usually passing. Vomiting is not fecal.

The *treatment* is rest in bed in a position to relax the belly, an ice-bag over the hernia for a very few hours, and a little opium for pain. Do not give a particle of food for twenty-four hours; when the active symptoms subside give an enema, and, after this acts, a dose of castor oil. Do not employ taxis, as bruising the bowel may produce strangulation. If improvement does not rapidly occur, operate. Prompt operation saves the patient from the danger of strangulation and cures the hernia.

**Inflamed Hernia.**—Inflammation of a hernia is local peritonitis due to injury of an irreducible hernia. The mass becomes tender and painful, and perhaps heat is noted. In enterocele much fluid forms; in epiplocele the mass becomes hard. The hernia cannot be reduced; there is constipation, often vomiting, usually elevated temperature, but the mass still shows impulse on coughing. Vomiting is not fecal. Some gas is usually passed through the bowels. Constitutional symptoms are slight.

The *treatment* usually recommended is rest in bed with abdominal relaxation, an ice-bag to the tumor for a few hours, a small amount of opium by the mouth if pain is severe, an enema, and, after this acts, a saline. In an inflamed hernia there is great danger of strangulation, and operation should be performed in preference to relying upon the conservative plan.

**Strangulated hernia** is a condition in which, if the hernia contains bowel, not only is the fecal circulation arrested and gas prevented from passing, but the circulation of blood in the bowel wall is also arrested. The bowel is

irreducible and obstructed, and the blood ceases to circulate. If the hernia contains omentum, the omental vessels are tightly constricted. In both bowel and omentum gangrene soon occurs, but sooner in bowel than in omentum. Strangulation is commonest in old inguinal ruptures in active, middle-aged men, and is more frequent in enteroceles than in epiploceles. It is most common when the hernial orifice is small and is seldom seen in large ruptures.[1] Strangulation is rare in childhood. Strangulation is much more dangerous if bowel is present in the sac than if only omentum is present. If in a subject of hernia the abdominal pressure is suddenly increased, as by a violent cough or a muscular effort, the hernial orifice is dilated for a moment, more intestine or omentum may enter the sac, and if it does, it may be caught and constricted by the now constricted hernial orifice and strangulation begins. Strangulation so caused is called *elastic strangulation*. A sudden increase of intra-abdominal pressure may force a quantity of fecal matter into the herniated intestine. The sudden entry of a quantity of fluid and gas into the herniated coil causes *fecal strangulation*, the mechanism of which is obscure. By *retrograde strangulation* we mean a condition in which the end of a loop of bowel or a piece of omentum in a hernia re-enters the abdomen and then becomes strangulated, the balance of the hernia not being strangulated. Strangulation may be due to active peristalsis or to congestion, and it may arise from inflammation or from incarceration. The constriction may be at the neck of the sac, in the outside tissues, or even in the sac itself. In an hour-glass hernia the constriction may be in the body of the sac. In inguinal hernia a tight external ring is a common cause of strangulation and is the commonest cause in children. As Coley shows, the neck of the sac is very seldom the cause in children. Adhesions within the sac may cause strangulation. Spasmodic contraction of the tissues about the neck of the sack is an exploded hypothesis. The obstructed veins dilate and the blood in them ceases to move, the bowel becomes deep bluish and finally black, effusions of blood occur beneath the peritoneum, and the intestinal wall becomes edematous. Fluid transudes into the sac, and the fluid, at first clear, assumes a bloody hue, and finally becomes dry and foul. The peritoneum ceases to glisten, becomes dry and rough, and coated here and there with lymph. Strangulated omentum undergoes edema and hemorrhagic infarction and thrombosis occurs. When strangulation once begins the hernia swells, a furrow forms on the bowel at the seat of constriction, the bowel and omentum below the constriction become deeply congested and edematous, and, finally, the hernia passes into a state of moist gangrene (Fig. 763). The gangrene may be in spots or the entire mass may be gangrenous. The mucous membrane may be gangrenous when the serous coat looks fairly

Fig. 762.—Strangulated umbilical hernia containing nearly all the intestines and part of the stomach. Strangulation under bands within the sac.

[1] Strangulation developed in the large herniæ shown in Figs. 730 and 762.

sound. When gangrene is once established, the bowel is in danger of rupturing. At the point of constriction there may be a line of ulceration or of gangrene even when the balance of the gut looks fairly safe. A strangulated femoral hernia becomes gangrenous more rapidly than a strangulated inguinal hernia.

*Symptoms.*—This condition is sometimes preceded by diarrhea and uneasiness or pain about the hernial orifice. When strangulation begins the victim is seized with pain in and about the hernia and with violent colicky pain about the umbilicus, and the paroxysms of colic become more and more frequent, until finally the pain may become continuous. The hernia is found to be irreducible; larger than usual, tender, painful, dull on percussion, without impulse on coughing, and the skin above it may be reddened. Eructations of gas are frequent, and generally uncontrollable vomiting and prostration come on. Vomiting, as a rule, is an early symptom, and one which increases in severity. Occasionally it only follows the swallowing of liquids. Not unusually there is retching rather than vomiting. In rare cases vomiting does not begin for twenty-four to forty-eight hours. Vomiting is earlier and more violent when bowel is present in the sac than when the hernia is purely omental.

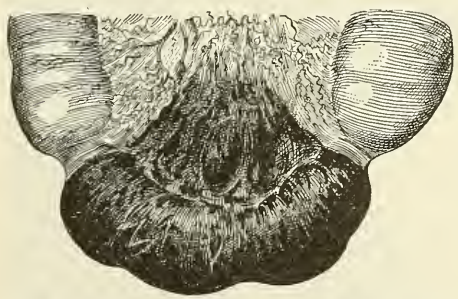


Fig. 763.—A strangulated coil of intestine after the strangulation existed for a considerable period of time. The color has become almost black and the peritoneal surface is dull and covered with flakes of fibrin. The constriction rings are deeply sunken, their walls markedly thinned, relaxed, and dirty gray in color. Both constriction rings are gangrenous and hemorrhages are observed in the mesentery (Sultan).

During the course of a strangulation vomiting may cease for a day or more, and it not unusually ceases toward the end, when prostration is profound. The early vomiting is due to reflex causes; the later vomiting is due to waves of peristalsis which produce regurgitation (Macready's "Treatise on Ruptures"). The vomiting is first of the alimentary contents of the stomach, next of mucus and bilious matter, and finally of the contents of the small bowel (*fecal* or *stercoraceous vomiting*). Stercoraceous vomiting rarely arises until strangulation has lasted forty-eight hours, and may not appear until much later. "It is seldom met with in inguinal, more often in femoral, and more often still in obturator, hernia" (Macready, Ibid.). Prostration is a marked symptom of a strangulated hernia, and it increases hour by hour and goes on to collapse. Early in the case there may be some elevation of temperature, but later it becomes normal or subnormal. The pulse is small, irregular, rapid, and very weak; the extremities cold; the face becomes Hippocratic. Constipation is absolute, no gas even being passed, though in the very beginning there may be some diarrheal passages from below the constriction. The urine is scanty and high colored, and contains only a small amount of the chlorids; the tongue becomes dry and brown; the thirst is torturing, and the patient often has an imperative desire to go to stool. Pains in the abdomen and in the hernia become more and more violent, and collapse rapidly increases. When gangrene begins the symptoms apparently lessen in violence: there is a *delusive calm.* Vomiting usually ceases, though regurgitation may take its place; hiccup begins; the pain abates or disappears; the pulse becomes very frequent, feeble, and intermittent; collapse deepens, and delirium is usual. It is a safe clinical rule that in strangulated hernia sudden cessation of pain without the relief of constriction, the disappearance of the lump, or the use of opiates means that gangrene has begun. In some cases of strangulation there are muscular

cramps in the legs (Berger). In children convulsions are not unusual. In a pure omental hernia strangulation produces similar but less decided symptoms. It may be that only a portion of the circumference of the bowel is caught and constricted in a hernial orifice (see Fig. 770, A). Such a condition is encountered occasionally in the femoral ring, and is called *partial enterocele* or *Richter's hernia*. The name *Littré's hernia* is often wrongly given to this condition. What Littré described was a hernia of Meckel's diverticulum (see Fig. 770, B). In a strangulated Richter's hernia constipation is rarely absolute and often no protrusion is discovered.

*Treatment.*—In treating strangulated hernia place the patient upon his back, bend the knees over a pillow, and rigidly interdict the administration of food. An attempt may usually be made to effect reduction by gentle manipulation or *taxis*. In applying taxis to a femoral or inguinal hernia, flex and adduct the thigh of the affected side. In applying taxis to an umbilical hernia both thighs should be flexed upon the abdomen. Always lower the shoulders and head and raise the pelvis, and accomplish this by lifting the foot of the bed and placing pillows under the pelvis. In some cases raise the entire body and lower the head. Grasp the neck of the sac with the fingers and thumb of one hand, and employ the other hand to squeeze the hernia and urge it toward the belly. In direct inguinal hernia the pressure should be backward and a little upward; in umbilical hernia it should be backward; in oblique inguinal hernia it should be upward, outward, and backward; in femoral hernia it should be downward until the hernia enters the saphenous opening, and then "backward toward the pubic spine" (MacCormac, in Treves's "Manual of Surgery"). If the bowel should be reduced, it will pass from the hand with a sudden slip and enter the belly with an audible gurgle; omentum, when reduced, slowly glides back without gurgling. Taxis is never to be continued long, and it is not even to be attempted in cases of great acuteness, in cases in which strangulation has lasted for several days, in cases known to have been previously irreducible, in cases associated with stercoraceous vomiting, or in inflamed or gangrenous herniæ.

If taxis fails, obtain the patient's permission to operate. Anesthetize; in some cases try taxis again upon the unconscious patient and while ether is being dropped upon the hernia to cause cold; if reduction fails, at once perform herniotomy. Taxis possesses certain dangers: It may rupture the bowel; it may rupture the neck of the sac and force the bowel through the rent into the tissues of the abdominal wall; it may strip the peritoneum from around the hernial orifice and force the bowel between the detached peritoneum and the abdominal wall; it may reduce a hernia into the belly when the bowel is still strangulated by adhesions; it may reduce the hernia *en masse* or *en bloc*. By the term "reduction *en masse*" we mean that the sac has been separated and dislocated and with the constricted bowel within it has been forced through the internal ring. By "reduction *en bissac*" is meant the forcing of a congenital hernia into a congenital pouch or diverticulum. Reduction *en masse* is a rare accident. Corner and Howitt ("Annals of Surgery," vol. xlvii) collected 137 cases of reduction *en masse* of strangulated hernia. Of these, 110 were males, 113 were inguinal, 22 femoral, and 2 obturator herniæ. No ventral or umbilical cases are recorded. The accident is a very dangerous one. According to Corner and Howitt (Ibid.), the mortality after inguinal reductions *en masse* is 48 per cent., and after femoral reductions, 72 per cent. Strange to say, reduction *en masse* can occur spontaneously. The subject most liable to reduction *en masse* is an elderly person with an old hernia. In acute cases the small bowel is the viscus which was reduced. In subacute and chronic reductions *en masse* the omentum, large bowel, or bladder was reduced (Corner and Howitt, Ibid.). Subacute and chronic cases may

happen in non-strangulated hernia. In any of the above accidents obstruction may persist after apparent reduction by taxis. Persisting obstruction means strangulation or peritonitis and calls for instant laparotomy—in most instances through the hernial aperture. If taxis is successful, put the patient to bed, apply a pad and bandage, allow no food until vomiting ceases, merely permit him to take a little hot water during the first twenty-four hours, and keep him on a liquid diet for several days. At the end of the first week give solid food. Do not disturb the bowels for a few days, but if they have not acted when four or five days have elapsed since the operation, give a saline cathartic followed in a few hours by a purgative enema. There is usually a spontaneous movement within twenty-four hours after reduction by taxis.

**Herniotomy.**—If there has been stercoraceous vomiting the stomach must be washed out before giving the anesthetic, and during the administration of the anesthetic the head should be turned upon its side. In most cases a general anesthetic can be given, but in desperate cases it is not justifiable to give ether or chloroform, and a local anesthetic must be used (infiltration anesthesia). Wrap the patient up in blankets. In most cases try gentle taxis for a brief time after the patient has been anesthetized and while ether is being dropped upon the hernia to cause cold. Never try taxis if stercoraceous vomiting has occurred. If taxis fails, at once sterilize the parts and operate. Always lay out a hernia knife (Fig. 764), a director (see Fig. 732, A), and Murphy buttons. During the *operation* the patient lies

Fig. 764.—Cooper's curved herniotome.

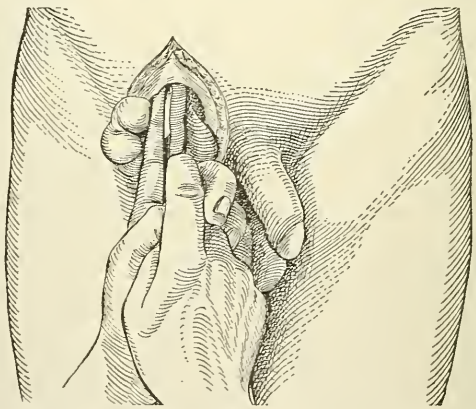


Fig. 765.—The division of the constriction from within outward (Sultan).

upon his back with the shoulders raised, the surgeon standing to the patient's right side. In *oblique inguinal hernia* it has been the custom since the days of Scultetus to raise a fold of skin at a right angle to the axis of the external ring and transfix it, the wound which results being extended until it becomes 3 inches in length. This incision possesses no special merit. It is better to cut from without inward, and to make the same incision as for the performance of a radical cure in a non-strangulated case. The superficial tissues are divided, the aponeurosis is opened as in Bassini's operation, and the sac is reached. In most cases the constriction is relieved as soon as the external ring is nicked, and in many cases fibrous adhesions will be found in that region, gluing the sac to the ring. The sac must be identified; and it is known by the fat which usually covers it, by the fluid within the sac, by the arborescent

arrangement of its vessels, and by the fact that it can be pinched up between the finger and thumb and the layers rolled over each other. Should the sac be opened? It may not be actually necessary in every recent case, but if there is any doubt as to the condition of the bowel or if a radical cure is to be attempted, open the sac and be certain as to the condition of its contents. As there is always some doubt as to the condition of the contents, and as a radical cure is to be made, make it a rule to always open the sac. The sac is opened and the contents examined for fecal odor (which is not unusual) and for gangrenous smell; the thickness of the bowel is estimated and the color and luster are determined. If a constriction exists at the neck, it is nicked with a hernia knife. If the hernia is oblique inguinal and is caught at the internal ring, nick the constriction upward and outward or directly upward, as shown in Fig. 765. In direct inguinal hernia and in an oblique inguinal hernia caught at the external ring the cut is made upward and inward. Always pull the bowel down and examine the seat of constriction to see what damage has been inflicted at that point. If the serous coat glistens; if the proper color comes back to the bowel wall after irrigation with very hot water; and if there are no spots of gangrene, restore the bowel to the abdomen and do a radical cure. If the bowel is in a doubtful condition, fasten it to the incision, apply a dressing, and watch the development of events. If the bowel is gangrenous, our action depends upon the condition of the patient. If the patient is in good condition, resect the gangrenous portion and perform end-to-end approximation. If the patient's condition is bad, draw the gangrenous portion well out, anchor it to prevent leakage and retraction, make an artificial anus, and at a later period perform anastomosis. An artificial anus can be made by the method of Bodine (see page 1122). Unfortunately in these cases the artificial anus must usually be made in the small intestine. In many cases in which there is some uncertainty as to the need for an artificial anus prepare the bowel for the opening, but do not open at once, because the bowel may recover in a day or two, when it can be restored to the belly, or it may slough and form an artificial anus. In such doubtful cases fasten the bowel to the belly wall with sutures, dust it with iodoform, dress it with hot antiseptic fomentations, and await future developments. Gangrenous omentum requires ligation and resection. If the bowel is fit to reduce, push it just inside the ring, irrigate the parts, suture, and perform a radical cure. In *femoral hernia* we can make the incision 1 inch internal to and parallel with the femoral vessels and crossing the tumor and ligament (Barker); but it is better to make the incision of Fabricius for radical cure. Divide the constriction by cutting upward and a little inward. If the gut is found gangrenous or in a doubtful condition, follow Blake's advice ("Surg., Gynec., and Obstet.," May, 1906), make an incision at the edge of the rectus, draw the affected portion of gut into the abdomen and out of the incision, and either resect, fix it under or out of the incision, or wait for return of circulation, as may be indicated in the case. To draw the gut out of the femoral ring makes so much traction that return of circulation may be prevented, and any intestinal operation is difficult in this region without splitting Poupart's ligament. In *umbilical hernia* make a slightly curved incision a little to one side of the middle of the tumor, open the sac, separate adhesions, and divide the constriction by cutting upward or downward, and sometimes also laterally, or, better, operate as for radical cure (see page 1148).

After an operation for strangulated hernia put the patient to bed; bend the knees over a pillow; give no food by the mouth for thirty-six hours (MacCormac), only allowing hot water, and every sixth hour give an enema of salt solution containing brandy. Abdominal pain and tenderness call for the administration of saline cathartics and enemata containing turpentine or oil of rue. The enema rutæ is a favorite preparation in St. George's Hospital,

London. It is made as follows: Take 16 oz. of an infusion of camomile, warm it, and pour it upon 3 dr. of confection of senna (Sheild). If there is no abdominal pain and no tenderness the bowels need not be disturbed for a few days; but if at the end of four or five days they have not acted, give a saline cathartic and a few hours later a purgative enema. At the end of about three weeks get the patient up. If a radical cure has not been attempted, apply a pad and a spica bandage to the groin, and later a truss. A truss should not be worn if a radical cure has been made.

*Mortality.*—Cases of strangulated hernia irreducible by taxis will practically all die without operation. The mortality following operation is large; it is not due to operation, but is due to the condition, and is due particularly to delay in operating or to forcible antecedent taxis. Sultan, from a total of 1429 herniotomies, estimates the mortality at 20.7 per cent. Estimating the mortality according to the time of strangulation, Henggeler reaches the following conclusions: The mortality of cases operated upon "the first day after the strangulation is 8.09 per cent.; during the second day, 22.2 per cent.; during the third day, 45.5 per cent.; during the fourth day, 60 per cent." ("Atlas and Epitome of Abdomianl Hernias," by Dr. George Sultan. Translated and edited by Wm. B. Coley, M. D.). The mortality in children is smaller than in adults. In Coley's 12 cases of children under two years of age there was not a death.

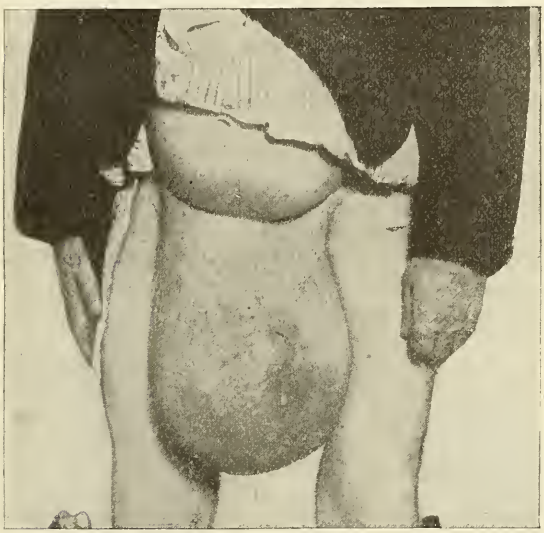


Fig. 766.—Double inguinal rupture.

**Hernia in Childhood.**—Hernia is extremely common in children, but it is an interesting fact that if one conducts a careful investigation of hernia in adults, it will be found that but 5 or 6 per cent. of them have suffered from the hernia in childhood. This fact seems to demonstrate positively that the majority of cases of hernia in childhood are recovered from. A. J. Ochsner ("Jour. Am. Med. Assoc.," Dec. 22, 1900), in commenting upon the frequency of hernia in childhood, alludes to Malgaigne's statistics. Malgaigne estimated that during the first year of life 1 child in every 21 has hernia, and that this proportion is maintained until the age of six. Then it diminishes rapidly until the age of thirteen, at which age there is 1 hernia in every 77 children. It is, therefore, obvious that 75 per cent. of all herniæ in children of six years will heal spontaneously before the age of thirteen. Ochsner states that 95 per cent. of herniæ in children will be cured without operation. He points out that between the ages of thirteen and twenty hernia is fairly common among boys, but very rare among girls. The reason for the tendency to cure is somewhat uncertain. The view advocated by Thomas C. Martin is that, as the pelvis broadens, the parietal peritoneum enlarges. It does this at the expense of the mesentery, which is shortened, and the internal abdominal ring is displaced. In a very instructive analysis of this condition Ochsner shows that

in 25 per cent. of cases of hernia in childhood hereditary weakness exists; that the condition is commoner among the poorer classes than among the rich; that in many cases there is an undescended testicle; and that the chief cause is an excess of intra-abdominal pressure. This excess of intra-abdominal pressure may result from flatulent distention of the stomach and intestines, the product of bad feeding; constipation and straining; straining on urinating, due to the existence of phimosis; vomiting, or cough. He thinks that, as a rule, indigestion causes flatulence and pain; that the child cries; that this increases the pressure; that the mother then feeds it in order to keep it quiet, and that this makes it worse. Strangulation is rare in childhood.

*Treatment.*—Strangulated herniæ, irreducible herniæ, herniæ with very large rings, cases in which trusses fail, and cases associated with reducible hydrocele require operation (Ochsner). Most cases are curable without operation, the ring being guarded by a truss of rubber or a pad of lamb's wool. Ochsner believes that many cases can be cured by keeping the child recumbent, with the foot of the bed raised, from four to six weeks. If phimosis exists, it should be operated upon, and any other causative condition should be treated (cough, vomiting, constipation, flatulent indigestion, etc.). An umbilical hernia can usually be cured by the use of a cork. The cork should be 1 inch in diameter and $1\frac{1}{4}$ inches in length, and shaped like a cone. The smaller end is pushed into the ring and the cork is held in place by adhesive plaster. In two weeks a smaller cork must be used, and in six or eight weeks it can usually be dispensed with. Radical cure operations are seldom done before the age of four (see page 1136).

**Varieties of Hernia.**—*Direct inguinal herniæ* comprise less than 2 per cent. of cases of inguinal herniæ. In direct hernia the contents pass out through Hesselbach's triangle internal to the deep epigastric artery. They enter the inguinal canal low down, and pass outside the conjoined tendon or force the conjoined tendon before them or split through the tendon. They do not enter the scrotum. The neck of the sac is internal to the deep epigastric artery. The protrusion is globular in shape, unless it emerges around the edge of the tendon, in which case it is pear shaped. The coverings of this hernia, when it passes external to the conjoined tendon, are the same as those of an indirect inguinal hernia, except that the transversalis fascia instead of the infundibuliform process of the transversalis fascia is one of the layers. When a direct hernia pushes before it the conjoined tendon, its coverings are skin, superficial fascia, intercolumnar fascia, conjoined tendon, transversalis fascia, subserous tissue, and peritoneum.

In *indirect inguinal hernia* the contents pass through the internal abdominal ring external to Hesselbach's triangle and external to the deep epigastric artery. They pass down the inguinal canal and emerge from the external ring; and may or may not enter the scrotum or labium (*scrotal* or *labial hernia*). The protrusion is pear shaped. The neck of the sac is external to the deep epigastric artery. Its coverings are skin, superficial fascia, intercolumnar fascia, cremaster muscle, infundibuliform fascia, subserous tissue, and peritoneum.

*Congenital inguinal hernia* is a portion of bowel within an unclosed vaginal process. The bowel in congenital hernia has one layer of peritoneum in front of it. The testicle is posterior and below (Fig. 767, *b*). Always remember that bowel may not enter the sac of a congenital hernia for several months or longer after birth. Congenital hernia conceals or buries the testicle; acquired hernia does not. If a vaginal process, open above and closed below, contains a hernia, the condition is called *hernia into the funicular process* (Fig. 767, *c*).

If the funicular process is closed at the abdominal end, but not below, a hernia in a special sac may descend back of the vaginal tunic. This condition is known as *infantile hernia*. In infantile hernia there are three layers

of peritoneum in front of the bowel—the two layers of the vaginal tunic and the one layer of sac. The testicle is in front (Fig. 767, *d*).

If the tunica vaginalis is closed above and not below, and a hernia pushes down the vaginal process and causes it to double on itself, the condition is known as *encysted infantile hernia* (Fig. 767, *e*).

In *femoral hernia* the contents descend along the femoral canal to the inner side of the vein, and the neck of the sac is at the femoral ring. The neck of a femoral rupture is always external to the pubic spine; the neck of an inguinal rupture is always internal to the pubic spine. Femoral hernia contains omentum, but seldom intestine, except in strangulated cases. It used to be said that femoral hernia is never congenital. Russell and Coley show that it may be (see page 1133). The coverings of a femoral hernia are skin, superficial fascia, cribriform fascia, crural sheath, septum crurale, subserous tissue, and peritoneum.

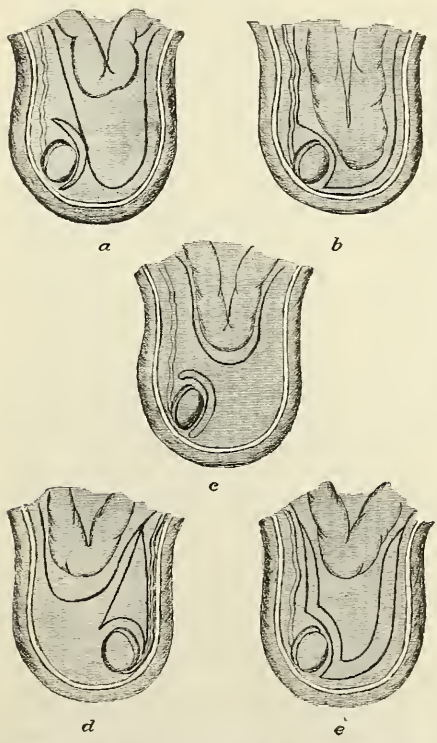



Fig. 767.—*a*, Scrotal hernia; *b*, congenital hernia; *c*, funicular hernia; *d*, infantile hernia; *e*, encysted hernia.

Occasionally a femoral hernia may pass in front of the vessels (*prevascular femoral hernia*). Moschchowitz operated upon such a case ("Annals of Surgery," June, 1912). A hernia may be external to the femoral artery (*Hesselbach's hernia*); may pass through an opening in Gimbernat's ligament (*Laugier's hernia*); may come down alongside of the femoral vein, but instead of emerging from the saphenous opening spread into the pectineus muscle (*Cloquet's hernia*).

*Umbilical hernia* may be congenital (the ventral plates having closed incompletely), infantile (the cicatrix of the umbilicus having stretched), or acquired.

*Ventral hernia* is a protrusion through any part of the anterior abdominal wall except at the umbilical or inguinal regions. A ventral hernia may be median (*hernia of the linea alba*) or lateral. The treatment is radical operation.

*Epigastric hernia* is a form of ventral hernia. In this condition there is a protrusion of the peritoneum in the space bounded by the ensiform cartilage, the ribs, and the umbilicus. The sac of peritoneum may be empty, may contain omentum, or omentum and bowel. The stomach very rarely passes into the sac. The protrusion is usually, but not invariably, through the linea alba. The condition may be due to a congenital gap in the transversalis fascia or to the growth of a fat hernia. This condition may cause abdominal pain, epigastric pain, nausea and vomiting after eating or effort, and, according to Farrar Cobb ("Annals of Surgery," Jan., 1912), chronic diarrhea.

*Cecal hernia* is very uncommon in women. It may be either congenital, infantile, or acquired. If a vaginal process is open the cecum may readily enter it, it may be drawn in by the plica vascularis as the testicle descends

(Wrisberg), it may be drawn in by a descending testicle the posterior peritoneum over which has formed adhesions to the cecum. Most acquired cecal herniæ are preceded and caused by hernia of the small gut, but they may occur alone. In the simple form the cecum is completely covered by a coat of peritoneum and lies within the sac. Usually there is a complete surrounding sac, but sometimes the sac only partially covers the cecum lying in front and to the inner side. In these cases the cecum is on the posterior and external aspect of the wall of the sac. The appendix may be in the sac, outside of the sac, or part may be within and part without. If the sac is incomplete, it means that we have one of the 18 per cent. of cases in which the cecum is not completely covered with peritoneum. A cecal hernia may be and usually is right inguinal, but may be right femoral, left inguinal, or left femoral. It is most common in advanced life and is frequently irreducible.

*Hernia of the appendix* may occur alone, and Merigot de Treigney collected 22 cases of it ("Thèse de Paris," 1887). In 17, the hernia was inguinal; in 5 it was femoral. I operated upon a case of appendicitis in which the inflamed appendix was the sole occupant of an incomplete right inguinal hernia sac. In some cases the appendix accompanies the cecum into a hernia.

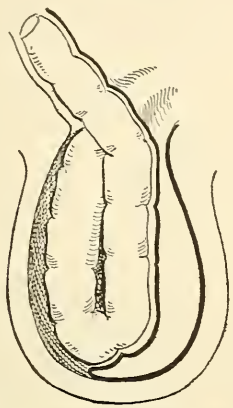


Fig. 768.—The large intestine behind the peritoneum (Weir).

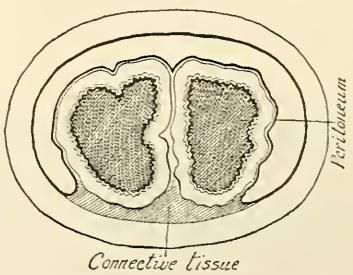



Fig. 769.—The retroperitoneal large intestine in a cross-section of the hernia with its incomplete sac (Weir).

*Adherent hernia of the large intestine (sliding hernia)* is a condition first described by Scarpa, in which the sac of a right-sided hernia contains ascending colon; of a left-sided hernia, descending colon. The sac is complete on the anterior, but apparently absent on the posterolateral aspect, where the bowel seems as though fused to it. This condition has often been called "hernia with incomplete sac." A commonly accepted theory is that because of looseness of the peritoneum of the iliac region a portion of the large bowel slides into the hernia. In such a case the posterolateral aspect of the sac is absent (Figs. 768 and 769). In a right-sided condition the descending bowel carries with it into the scrotum a fold of loosened peritoneum, just as in the descent of the testis (Weir, in "Med. Record," Feb. 24, 1900). Sliding hernia of the ascending colon is wrongly called sliding hernia of the cecum. In most cases of sliding hernia of the left side the descending colon is dragged into a preëxisting hernia sac containing small bowel, omentum, or both. The large bowel is covered with peritoneum except posteriorly, where the mesocolon is attached. This form is nearly always irreducible and occurs particularly in elderly men. In another group of cases the large bowel makes a direct inguinal hernia and the sac is limited to the anterior surface of the protruded gut.

In this edition I have adopted the name "adherent hernia" instead of "sliding hernia." L. J. Ransohoff ("Annals of Surgery," August, 1912) seems to

demonstrate that the condition is not due to sliding of the peritoneum, and hence is not a sliding hernia; that an incomplete sac is a secondary process, the sac having been complete in its incipiency, but obliterated posteriorly "by secondary adhesions of the embryonic type." The immobile loop of intestine in the sac was originally mobile; "the hernia is primary, the adhesions secondary" (Ransohoff, "Annals of Surgery," Aug., 1912).

In *properitoneal hernia* the sac is between the peritoneum and transversalis fascia. The form of hernia is sometimes produced by making taxis on an inguinal hernia, when the internal ring is small or is blocked by an undescended testicle. In properitoneal inguinal hernia, which is the most common form, there are two sacs, one in the scrotum, the other parallel with Poupart's ligament, and as one sac is emptied, the other distends (Breiter, of Zürich).

In *interstitial* or *interparietal inguinal hernia* the hernia sac is between the transversalis muscle and fascia, or between the external and internal oblique muscles, or in the midst of the fibers of the internal oblique muscle, or between the external oblique muscle and the transversalis fascia, the internal oblique and transversalis muscles being pushed aside (Sultan's "Atlas of Abdominal Hernias").

In *superficial inguinal hernia* the sac is between the aponeurosis of the external oblique muscle and the superficial fascia. This variety of hernia is always congenital and the testicle is invariably misplaced.

*Obturator hernia* passes through the obturator membrane or the obturator canal, and is felt below the horizontal ramus of the pubes, internal to the femoral vessels. The obturator nerve is pressed upon and pain arises in its trajectory.

*Lumbar hernia* is a very rare condition. It may be congenital, traumatic, or spontaneous, and may follow a lumbar abscess. Braacz collected 68 cases. It occurs through the triangle of Petit, or just below the twelfth rib through the superior lumbar triangle, or through a congenital defect in the aponeurosis of the latissimus dorsi muscle, or through a defect near the triangle of Petit (Dowd, in "Annals of Surgery," Feb., 1907). A lumbar hernia may be present at the edge of or through the quadratus lumborum muscles.

The *triangle of Petit* is bounded in front by the external oblique, behind by the latissimus dorsi, below by the iliac crest, and its floor is formed by the internal oblique. The *superior lumbar* triangle (of Grynfelt and Lessbaft) is bounded anteriorly by the external oblique, posteriorly by the iliocostal muscle, above by the serratus posticus inferior and the end of the twelfth rib, and below by the internal oblique. The latissimus dorsi overlies it.

*Sciatic* or *gluteal hernia* passes through the great sacrosciatic foramen, above or below the pyriformis muscle, or through the lesser sacrosciatic foramen.

*Pudendal hernia* protrudes into the lower part of the labium, the bowel having descended between the ischial ramus and the vagina.

*Perineal hernia* presents in the perineum, between the rectum and the prostate gland or between the rectum and the vagina.

*Internal, retroperitoneal*, or *intra-abdominal herniæ* include hernia into the *foramen of Winslow*, hernia into the *retroduodenal fossæ*, the *retrocecal fossæ*, and the *intersigmoid fossa* (see page 981).

*Vaginal hernia* is associated with uterine prolapse or ensues upon destruction of the vaginal wall.

**Richter's hernia** (**partial enterocele** or **hernia of the intestinal wall**, Fig. 770, A) was described by Richter in 1778. He called it "the small rupture." It occurs only in adults and is most common in women. It is the catching of a portion of the circumference of the bowel, usually a portion of the lower part of the ileum. It is usually femoral, but may be inguinal, and even epigastric or

obturator. It arises usually in an old, reducible hernia (Royal Hamilton Fowler, "Am. Jour. Surg.," Jan., 1912). Some cases are due to adhesions. It may be due to truss pressure on an incompletely reduced hernia (Fowler, Ibid.). Strangulation of a partial enterocele may not completely close the lumen of the gut. There may not be stercoraceous vomiting or absolute constipation, and the protrusion is barely perceptible or cannot be palpated.

**Littré's hernia** is hernia of Meckel's diverticulum (Fig. 770, B). It was described by Littré in 1700. This diverticulum is the persistent vitelline duct and comes off from the ileum from 12 to 36 inches above the ileocecal valve. It arises from the convex side of the gut and rarely has a mesentery (see pages 982 and 988).

**Rokitansky's diverticular herniæ** are due to separation of the muscular fibers of the bowel, permitting the sacculation of mucous membrane and peritoneum. These false diverticula may be no larger than peas or may be larger than walnuts, and there may be scores of them in one patient. They may produce no symptoms or may lead to peritonitis, abscess about the bowel, perforation, or to symptoms of intestinal obstruction.

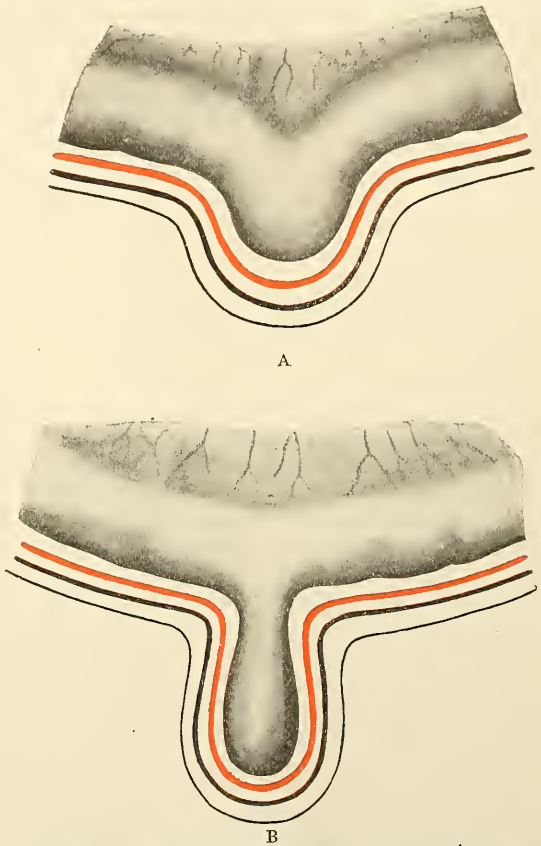



Fig. 770.—A, Diagrammatic representation of Richter's hernia of intestinal wall. B, Diagrammatic representation of Littré's hernia, which is a hernia of Meckel's diverticulum (Sultan).

**Hernia of the Bladder.**—This is a protrusion of a portion of the bladder wall through a hernial opening. Eggenberger adds 110 cases to Brunner's 182 cases, a total of 292 cases ("Deutsch. Zeitschr. f. Chir.," Oct., 1908). Most cases are instances of false hernia, there being no sac of peritoneum. The protrusion may or may not be covered with peritoneum, and in most cases it is not so covered, but lies by the side of a hernial sac and not inside of it. Brunner's table shows only 5 cases of true bladder hernia, that is, of intraperitoneal hernia. It is most frequently met with in the inguinal region. Brunner describes three forms: (1) Entirely without a peritoneal covering (extraperitoneal); (2) partly covered with peritoneum (paraperitoneal—the commonest form); (3) completely covered with peritoneum (intraperitoneal). The bladder may constitute the hernia, or there may be an ordinary hernia and also a cystocele. In an inguinal hernia the bladder will be internal and somewhat behind the other constituent parts of the protrusion. Hernia of the bladder is much more common in men than in women.

A hernia of the bladder may become strangulated. In some cases a diagnosis of hernia of the bladder can be made by the fact that the protrusion lessens in size when the patient micturates, and increases in size as urine gathers or when the bladder is injected with fluid. The treatment should be operative. When the bladder is exposed it is replaced if possible without resection of a portion.

**Diaphragmatic Hernia.**—The majority of cases are congenital and in 90 per cent. of them there is no sac. The hernia may pass through a natural opening or through a gap due to congenital defect. The hernia is most common on the left side, and the stomach is the organ which is most often found in the hernia, but the colon, the omentum, the small intestine, the spleen, liver, duodenum, cecum, pancreas, or kidney may be found (Cranwell, "Rev. de Chir.," 1908, No. 1). Violent traumatism may be the cause. When the stomach passes suddenly through the left side of the diaphragm there will be dyspnea, cyanosis, displacement of the heart to the right, pain in the upper abdomen, thirst, and in most cases rapid death. When the stomach or intestine has entered the left side of the thorax, there is a tympanitic note on percussing over that area of the thorax, the heart is displaced to the right, and the side of the chest is unduly prominent. The upper border of the tympanitic area does not move with respiration. There are no breath sounds audible over the tympanitic area, but gurgling is heard. In 250 cases of traumatic diaphragmatic hernia collected by Leichtenstern the diagnosis was made before death in but 5 cases. The *x*-rays are of value in diagnosis, especially if the stomach is in the hernia. Strangulation of a diaphragmatic hernia produces severe pain in the upper abdomen, violent vomiting, constipation, boat-shaped abdomen, great thirst, rapid wasting, and the excretion of a very small amount of urea (Mackenzie and Battle, "Lancet," Dec. 7, 1901). Diaphragmatic hernia may be confused with *eventration of the diaphragm*, an unnaturally high position of the left half of the diaphragm, with ascent of the viscera of the abdomen, especially the stomach. It was first described by Petit in 1790. Sailer and Rhein reported a case and collected 12 others ("Am. Jour. Med. Sciences," April, 1905). The physical signs of eventration are practically identical with those of diaphragmatic hernia, except that in the former the upper border of the tympany moves on respiration.

*Treatment.*—Open the belly for exploration. If a hernia is found, return it to the abdomen; open the chest and suture the diaphragm from above (transpleural suturing). Mackenzie and Battle, Mikulicz, Humbert, and others have operated for this condition.

**Hernia of the Ovary.**—The ovary, because of failure of descent, may remain in the lumbar region. It may pass into the inguinal canal or labium majus (inguinal hernia); to the gluteal region (gluteal hernia); to the region of the obturator foramen (obturator hernia); or to the front of the abdomen (ventral hernia). In congenital inguinal hernia there may be ovary alone, or ovary, tube, omentum, and even part of a bicornate uterus (Garrigues). It is impossible to restore a congenital hernia. Acquired hernia may follow a fall and sometimes it can be restored. A femoral or crural ovarian hernia, a condition in which the ovary passes to the front of the thigh below Poupart's ligament, is never congenital. In some cases a herniated ovary can be returned within the abdomen. Any herniated ovary may inflame.

*Treatment.*—If the ovary can be restored, a truss will probably retain it, but even in such a case operative cure is better. If it cannot be restored or if it is painful or undesirable to wear a truss, operation must be done. Expose the ovary, return it to the belly if healthy, and do a radical cure of the hernia. In some conditions of disease remove the ovary.

**Hernia of the Uterus.**—This condition is a surgical curiosity, but a few

cases have been reported (John Howard Jopson's case in "Annals of Surgery," July, 1904). The hernia may be umbilical, ventral, inguinal, or femoral. Hernia of the unimpregnated womb may be congenital or acquired; impregnation may occur when the uterus is herniated, or an impregnated uterus may pass into a preëxisting hernia sac. If a herniated uterus becomes impregnated or if an impregnated uterus becomes herniated, pregnancy may go on to term. Multiple pregnancies predispose to uterine hernia. Ovarian hernia may precede uterine hernia, or hernia of omentum adherent to the uterus may pull that organ into the sac. In many cases congenital anomalies have been found to exist (bicornate uterus, rudimentary uterus, shortness of the round ligament, imperforate vagina, etc.). A hernia of the uterus enlarges and becomes painful during menstruation, and a vaginal examination shows that the uterus is absent from its normal position and that the direction of the cervix and vagina are abnormal (Jopson, Ibid.). A uterine sound cannot be passed at all or can be passed with great difficulty. The hernia is hard and probably pyriform. If impregnation occurs, there are the ordinary signs of pregnancy and progressive enlargement of the hernia.

*Treatment.*—Expose the mass by incision. If conditions justify such a course, return the uterus and adnexa, if they are present (one or both ovaries and tubes may be present), to the abdomen and do a radical cure. If the uterus is infected, remove it. Jopson in his case removed the uterus and right ovary and fastened the uterine stump into the wound.

## XXIX. DISEASES AND INJURIES OF THE RECTUM AND ANUS

**Teratoids and Dermoids Associated with the Sacrococcygeal Region.**—In the sacrococcygeal region there are many opportunities for developmental error. In this region the caudal end of the primitive streak must undergo evolution and involution, the neurenteric canal must form and disappear, the anus must be formed, the posterior fissure must close, the sacrum and coccyx must develop, and various other processes must go on correctly and uniformly if complete development is to be obtained. As Coplin puts it: "Perfect evolution of the tissues embraced in this part of the body is beset by many narrow escapes" ("Publications from the Laboratories of Jefferson Med. College Hospital," 1906, vol. iii). Fissures and clefts may fail to close, fragments from one blastodermic layer may be lodged in another layer, groups of cells may be sequestered, closing clefts may include tissue elements, and parts that should normally atrophy may do so late or not at all.

In order to understand teratomata and dermoids arising in the rectal region we must recall some facts of development.

Early in embryonic life the central canal of the spinal cord and the alimentary canal are continuous around the caudal end of the notochord by a communicating path called the neurenteric canal.

When the anal pit undergoes invagination it meets the gut considerably in front of the region where the neurenteric canal joins the gut. The portion of intestine between the anus and the opening of the neurenteric canal is called the *postanal* gut. The postanal gut disappears during normal development. If it persists it causes tumor formation.

The congenital tumors of the sacrococcygeal region are: (1) Tumors of the postanal gut; (2) dermoids back of the rectum; (3) rectal dermoids.

According to Bland-Sutton, tumors of the postanal gut "are composed of closed vesicles lined with glandular epithelium and contain glue-like fluid" ("Tumors, Innocent and Malignant," by Sir J. Bland-Sutton). These tumors grow to a large size.

Dermoids back of the rectum often contain teeth, may attain large size, are apt to mount up behind the peritoneum, and may rupture and form a fistula. I helped Prof. Keen operate on such a case. The skin over the mass showed a growth of hair, there was a gap or cleft in the sacrum, and through this there was a clear passage from the rectum to the skin. This sinus closely resembled the trachea in structure.

Dermoids occasionally grow from the mucous membrane of the rectum and are apt to contain an abundant growth of hair.

**Examination of the Anus and Rectum.**—There are four positions in which we may place the patient for rectal examination, the one to be selected depending upon the probable local difficulty. These positions are the left lateral prone, the knee-chest, the exaggerated lithotomy, and the squatting position. The knee-chest position is desirable if the sigmoidoscope is to be used. A commonly employed position is the *left lateral prone position of Sims*, in which the patient lies on the left side, the chest on the table, the left arm behind the back, the knees drawn up, and the pelvis elevated on a hard pillow.

Very stout people should be placed in the *knee-chest position* or *exaggerated lithotomy position*, as the rectum cannot be seen when they lie on the side.

A *squatting position*, when the patient is placed as though on a commode, is best adapted to cases of prolapse and hemorrhoids. It is also used to bring within the range of the finger strictures or new growths that in the other attitudes would be beyond digital reach.

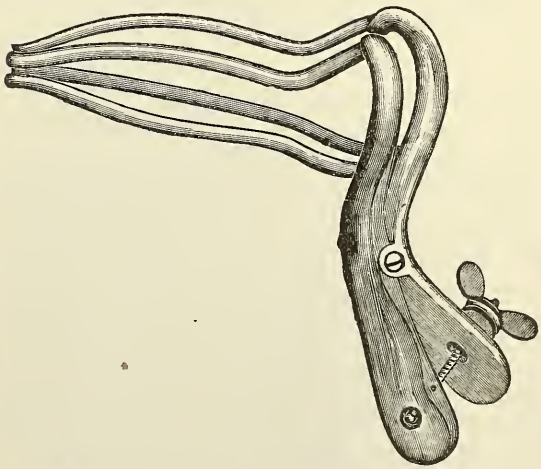


Fig. 771.—Mathews's self-retaining rectal speculum.

Sometimes the surgeon is able to pass a sigmoidoscope upon a patient in the exaggerated lithotomy position when it has been found impossible to carry the instrument high enough with the patient in any other position.

While making a rectal examination the patient should have no constriction about the abdomen, such as corsets, bandages, or tight clothing.

It is important that the first examination be made when no cathartics or enemata have been administered, so that the condition of the excretions and secretions may be noted. By such first examination pus, blood, mucus, or inspissated fecal matter if present may be seen, and from their varying characteristics, quantities, and locations inferences as to causation may be drawn. After these observations have been made an enema should be given and the anus and rectum be well cleansed. When the parts have been cleaned and the bowel washed out the anus is carefully inspected, the anal folds being opened during the process. By inspection the surgeon can notice the external opening of a fistula, external piles, protruding internal piles, mixed piles, pruritus, discharge from the rectum, eczema, fissure, tumor, ulcer, condylomata, abscess, whether or not the anus is retracted and funnel-shaped or protruding, or if there be parasites on the anal hairs.

Next the thumbs should be placed on either side of the anus and gently separated; this maneuver, aided by a bearing down effort on the part of the

patient, will often cause piles to protrude, exhibit fissures and polypi, and reveal the condition of the mucocutaneous border.

Next, a digital examination of the rectum is made. The nail of the index-finger is filled with soap and the finger is oiled or, better, is covered with a rubber finger-tip which is oiled. The digit is gently inserted through the sphincter, the patient being asked to strain lightly while it is passing. The finger is inserted with a gentle boring motion and is pointed toward the um-

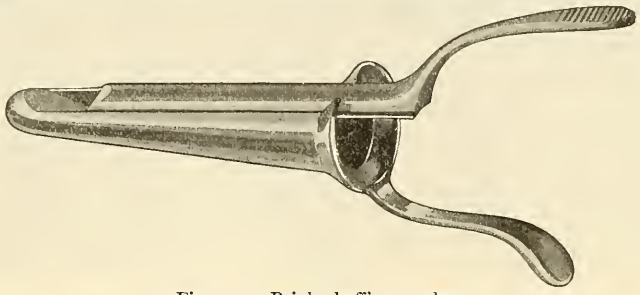


Fig. 772.—Brinkerhoff's speculum.

bilicus until the sphincter is passed. A digital examination enables the surgeon to detect an ulcer, a polypus, a tumor, a stricture, and to determine certain points regarding the condition of the prostate in the male and the uterus in the female. Non-indurated piles cannot be detected by the finger. A speculum will be needed to discover them if they are not protruding.

Next, in some cases, the rectum must be examined by means of a speculum. It is not often necessary to give ether. Mathews's speculum (Fig. 771) is very serviceable. Sims's duck-bill speculum is a valuable instrument for certain cases. The speculum is warmed, oiled, and slowly introduced. It is

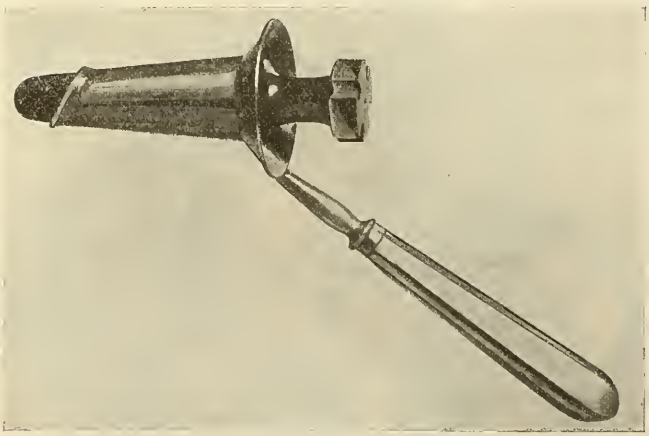


Fig. 773.—Martin's speculum.

first directed toward the umbilicus, and when it passes the sphincter its direction is gradually altered until it is toward the promontory of the sacrum. Illumination is obtained by direct sunlight or by a forehead mirror and an electric light. This examination will extend, confirm, or disprove the findings of the digital examination; ulcers, hemorrhoids, and malignant growths can be carefully examined, and the condition of the rectal mucous membrane can be thoroughly investigated.

Marion Sims in 1845 demonstrated the ballooning of the vagina by atmospheric pressure, and in 1870 Van Buren applied this method to the rectum. Kelly in 1895 put forth his straight tubes and described in detail the methods

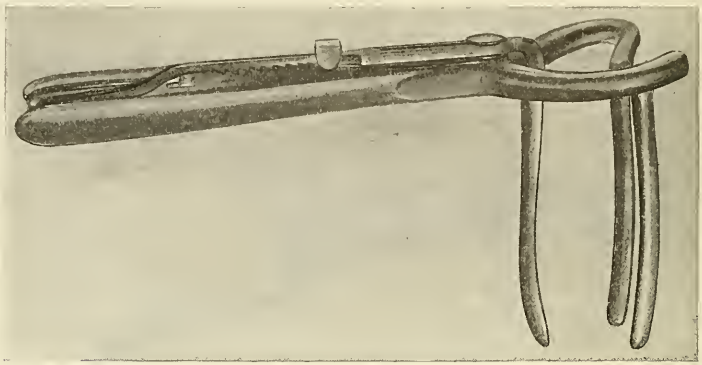


Fig. 774.—Cook's operating speculum.

and advantages of examination by them, and the great diagnostic value of ballooning the rectum. Kelly's method of examination is shown in Fig. 775. The tubes are shown in Fig. 776. It is not necessary to give ether. The

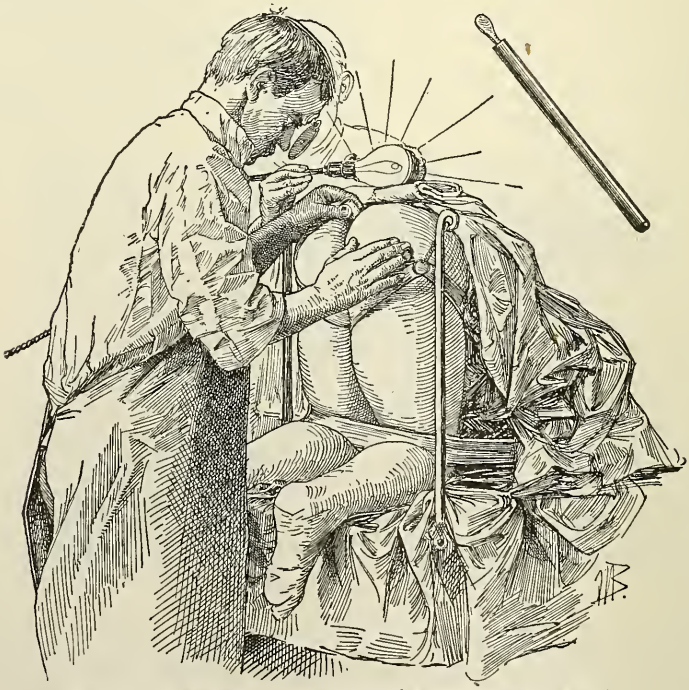


Fig. 775.—Examination of the rectum by reflected light (Kelly).

patient is placed in the knee-chest position. A tube containing an obturator is well greased with vaselin. "The buttocks are drawn apart, and the blunt end of the obturator is laid on the anus, which is also coated with

vaselin. The direction of the instrument should be first downward and forward, and, when the sphincter is well passed, up under the sacral promon-

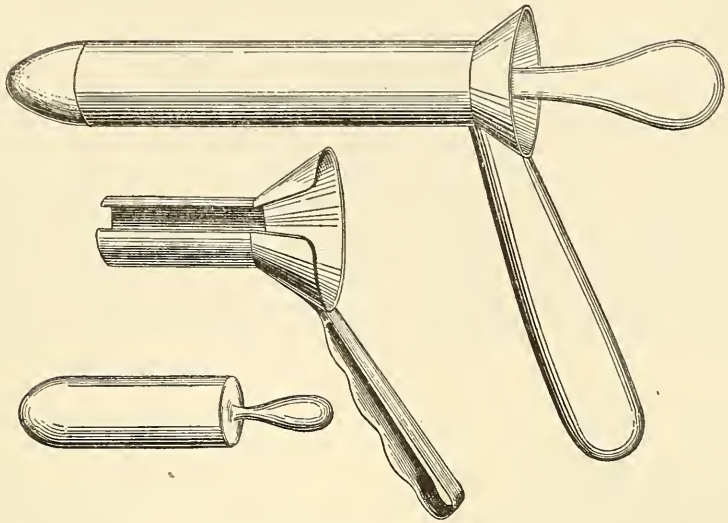


Fig. 776.—Kelly's rectal specula.

tory. The moment the speculum clears the sphincter ani and the obturator is withdrawn, air rushes in audibly and distends the bowel." When the entering instrument is pressed gently against the sphincter a sharp muscular contraction ensues. If the instrument is gently and slightly withdrawn, relaxation occurs, and the moment of relaxation may be seized to make an entry. An entry so made is rapid and unresisted. The bowel being distended with air, the mucous membrane is plainly seen as the tube is slowly withdrawn and the electric light is reflected into the speculum by a forehead mirror. The normal mucous membrane is dull red, like the nasal mucosa, and the blood-vessels are plainly distinguishable. The Kelly tube must be used with great care, as harm may be done by it, and the longest tube should be used only in exceptional cases.

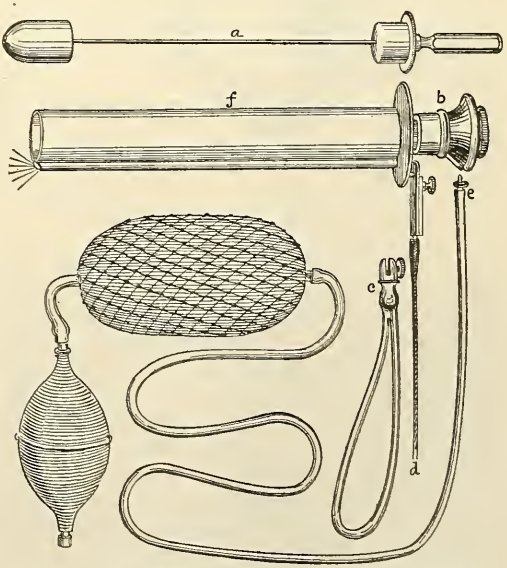



Fig. 777.—Tuttle's pneumatic proctoscope: *a*, Obturator; *b*, plug with glass window closing end of tube; *c*, handle; *d*, cords connecting instrument with battery; *e*, inflating apparatus; *f*, main tube of proctoscope.

I use with the greatest satisfaction James P. Tuttle's pneumatic proctoscope (Fig. 777). Dr. Tuttle describes it as follows ("Diseases of the

Anus, Rectum, and Colon"): "This instrument is composed of a large cylinder (*f*), into one part of the circumference of which is fitted a small metallic tube closed by a flint-glass bulb at its distal end. The electric lamp (*d*) is fitted upon a long metallic stem, and carried through the small cylinder to the end of the instrument, as shown in the illustration. The proctoscope is introduced through the anus with the obturator (*a*) in position. As soon as the internal sphincter is passed this obturator is withdrawn and the bayonet-fitting plug (*b*), which contains either a plain glass window or a lens focused to the length of the instrument to be used, is inserted in the proximal end of the instrument. This plug is ground to fit air-tight and thus closes the instrument perfectly. The plug being inserted in the tube, a very slight pressure upon the hand-bulb will cause inflation of the rectal ampulla to such an extent that the whole rectum can be observed and the instrument can be carried up to the promontory of the sacrum without coming in contact with the rectal wall. Further dilatation will show the direction of the canal leading into the sigmoid, and, by a little care in manipulating the instrument and keeping

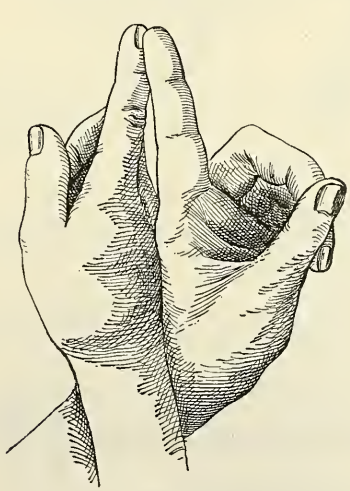


Fig. 778.

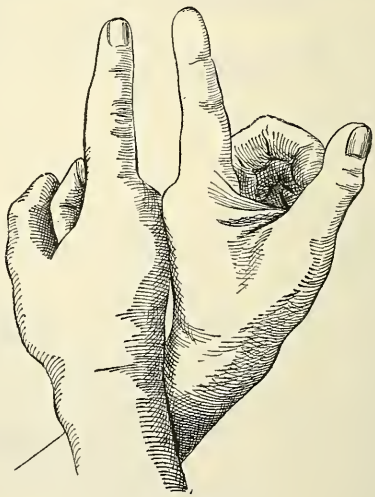


Fig. 779.

Figs. 778, 779.—A new and simple method of proctoscopy (Thomas C. Martin).

the gut well dilated in advance, it can be carried up into this portion of the intestine without the least traumatism of the parts. If any fecal matter obscures the light by being massed or smeared over the glass bulb, the plug can be removed, and a pledget of cotton, introduced with a long dressing forceps, will wipe this off, so that the plug can be reintroduced and the examination continued with very slight delay or inconvenience. The adjustable handle (*c*) fits on the rim of the instrument and thus converts it into a Kelly tube. This instrument is operated with an ordinary dry battery of four cells. It is better, however, to have a battery with six cells, as it will not require being recharged so frequently." All the air must be allowed to escape before the instrument is withdrawn, otherwise colic may develop.

If an anesthetized patient is placed in the knee-chest position, the sphincter can be stretched by the fingers, and the rectum will distend with air and can be easily examined. The fingers are introduced as suggested by Martin (Fig. 778) and the rectum becomes visible when they are separated (Fig. 779).

**Passage of Rectal Tubes.**—Some have asserted that soft tubes can be passed from the anus through the sigmoid. I do not believe this can be done in a normal rectum, it matters not in what position the patient is placed, and whether or not fluid flows from the tube during its introduction. A soft tube always coils up after it has ascended 6 or 7 inches. In a number of laparotomies, desiring to locate the rectum, I have had an assistant pass a rubber tube. It would never ascend above the rectal dome unless I aided it with a hand in the belly, when it could be gotten through the sigmoid. Fortunately it is not necessary to give high enemas, as fluid introduced into the rectum is carried back to the sigmoid. If we wish fluid retained, attempts to pass the tube high up will cause irritation, and irritation will lead to expulsion.

**Foreign Bodies in the Rectum.**—It is not at all unusual for hard, undigested articles taken with the food to lodge in the rectum. They can usually be removed through a speculum by means of forceps. In some cases ether must be given and the sphincter stretched; in others, the sphincter must be divided. Sometimes large bodies are voluntarily inserted and the individual is unable to remove them. Lewis H. Adler ("Am. Med.," July 20, 1901) removed the valve of a steam radiator pipe from the rectum. The small end was $1\frac{1}{2}$ inches in diameter; the large end was $2\frac{1}{2}$ inches in diameter. The patient had been in the habit of introducing it frequently and removing it with a hook of galvanized iron wire. A. Marmaduke Sheild ("Lancet," Oct. 12, 1901) reports the case of a man sixty years of age who forced a gallipot into the rectum. The pot was $2\frac{1}{2}$ inches in diameter and $2\frac{3}{4}$ inches in height. The patient broke it trying to get it out. Sheild incised the sphincter from behind and removed the article by means of obstetric forceps.

If the foreign body is soft, as an apple or a potato, a hole should be bored through to allow the air to pass and thus relieve the suction. It can then be lifted out. If the body is of wood, screw in a gimlet; if of metal, cut with strong forceps; if of glass, remove as Sheild did the gallipot. If the article is rough or has sharp edges, pack gauze all around it as a first step to protect the rectal walls. In a case in which a boar's tail had been introduced, large end first, the holding back of the bristles was overcome by sliding over it a large-sized rubber catheter.

Foreign bodies may escape into the sigmoid and necessitate a laparotomy. They may also cause periproctic inflammation.

A remarkable series of cases of foreign bodies in the rectum may be found in "Anomalies and Curiosities of Medicine," by George M. Gould and Walter L. Pyle.

**Wounds and Injuries of the Rectum.**—These accidents may result from fractures of the pelvis, most often from fractures of the sacrum, from the improper use of enema syringe nozzles or tips, from gunshot-wounds, stab-wounds, foreign bodies introduced by the patient, and, rarely, from digital examinations. The important points to consider in these wounds are whether or not there is penetration of the entire rectal wall, involvement of the perirectal tissues, or peritoneum. Slight wounds involving only the mucous membrane are frequently very grave from the fact that the hemorrhage is constant into the rectum, filling back into the sigmoid, often followed by collapse before or when the blood is voided. The investigations in these cases should always be thorough and by both digital and visual examination, for exact diagnosis is of the utmost importance.

If the peritoneum has been lacerated, laparotomy should be performed at once, the wound in the bowel repaired, the peritoneal cavity cleansed and drained, and the Murphy treatment for general peritonitis instituted (see page 1025). If the laceration involves the perirectal tissues but not the peritoneum, cut down on the side of the bowel that is injured and make free drainage. In the event of very extensive injuries to the perirectal tissues the

question of inguinal colostomy should be considered. If the wound involves only the mucous or submucous coats, dilate the sphincter, arrest hemorrhage by suture or ligature, and follow by irrigation.

**Inflammation of the Rectum and Sigmoid (Proctitis and Sigmoiditis).**—These conditions may be acute or chronic, simple or specific. The simple forms are acute catarrhal, atrophic catarrhal, and hypertrophic catarrhal. The specific forms are gonorrheal catarrhal, diphtheritic catarrhal, erysipelatous catarrhal, dysenteric catarrhal, and syphilitic catarrhal (Tuttle, "Diseases of the Anus, Rectum, and Colon").

**Acute Inflammation.**—Acute catarrhal inflammation may be caused by that class of incidents which are apt to be followed by ordinary catarrh of the respiratory passages and, in addition, errors of diet or sudden change of temperature, as sitting on a cold seat when overheated. The onset is sudden, with chill, general malaise, pain and discomfort locally, and slight fever. There is a sense of fulness and weight, with often a burning sensation referred to the rectum, and at times tenesmus with frequent desire to go to stool. Pain radiates to adjacent parts and there is bladder irritability. Patients usually prefer to be in the recumbent posture.

There will be at first a thin fecal discharge followed by mucus tinged with blood. Ulceration soon supervenes. The parts are hot, dry, and swollen in the first stages and digital examination is very painful. Later the parts are slimy and the mucous membrane is covered with tenacious mucus and pus.

*Treatment.*—First remove irritating intestinal contents and reduce engorgement by lavage and saline cathartics, followed, if early in the case, by irrigations of cold water; if later, by hot water. A useful medicated irrigation is a 5 to 10 per cent. solution of the aqueous extract of krameria.

**Chronic Inflammation.**—In chronic proctitis and sigmoiditis the symptoms are similar to those present in the acute forms of the inflammation, but less severe in character. In addition there is increased secretion of mucus, flatulence, and marked general intestinal disturbance.

The mucous membrane is soft, doughy, and thickened, which condition palpably reduces the caliber of the gut. Through the speculum the membrane is seen to be edematous, pale, and covered with secretion. It bulges into the aperture of the speculum and does not bleed easily.

*Treatment* is hygienic and dietetic, with antiseptic and astringent irrigations.

**Atrophic Inflammation.**—The atrophic variety of inflammation is characterized by long-continued constipation, dry stools covered with blood and mucus. There is such pain on expulsion of feces as to simulate in many cases fissure of the anus. The mucous membrane of the anus ruptures easily when stretched during examination. It is bright red and shiny, does not fill the aperture of the speculum, and bleeds readily. Ulceration is common and hemorrhoids are a frequent complication.

*Treatment* is local and general. Locally, use a dilute solution of nitrate of silver (1:2000), hydrastis (2 per cent. solution), or ichthyol, applied after the rectum is emptied. General treatment is hygienic and dietetic.

**Peri-anal and Perirectal Inflammations.**—These are of two classes —circumscribed inflammations or abscesses and diffuse inflammations that always develop pus. The circumscribed variety may be either superficial or deep. The superficial variety again may be tegmentary, subtegmentary, or *ischiorectal.* The deep variety may be of two forms—retrorectal or superior pelvirectal. It is important to note that right-sided superior pelvirectal abscess may be confounded with appendicitis.

The diffuse perirectal inflammations are variously classified, but the particular names refer only to the degree of the inflammatory process, which may vary from ordinary suppuration to gangrene.

These inflammations travel in the line of least resistance, which is upward, and more often burst into the bowel than externally. They may follow chilling of the region or external traumatisms, may be caused by perforation of the rectum by hard fecal masses, or by the direct passage of bacteria into the fossa through a fissure, an ulcer, or an ulcerated pile. They may be either acute or tuberculous. In many cases the process is at first tuberculous, and secondary infection with pyogenic bacteria takes place.

The **symptoms** are nearly identical with those of abscess elsewhere, the swelling, however, being brawny, and it being difficult or impossible to detect fluctuation. Pain in the groins is often complained of, and there may be enlarged glands in these regions. Abscesses commonly result in fistula, and a patient should be warned of this tendency before operation is performed. Superior pelvirectal abscesses generally follow inflammation of some pelvic organ, viz.: tubes, urethra, prostate, and the symptoms are mainly those of such an inflammatory condition. The usual tendency of the pus is to burrow upward. The presence of pus is indicated by the symptoms of suppurative toxemia, which sometimes simulate typhoid fever.

The **treatment** is instant incision. The patient, after having been anesthetized, is placed in the lithotomy position. The cut should be parallel to the fibers of the external sphincter and well outside of the muscle and longer than the inflamed area. When this incision is made, the finger should be introduced to break down the necrotic septa of cellular tissue. Then an incision should be made from the middle of the first cut radiating outward and opening any pockets external to the original focus. Then the free edges of the cuts are pared away, sacrificing all involved tissue. The wound is irrigated, the sphincter dilated, and the cavity packed with iodoform gauze. If a fistula is found opening into the rectum, it is not to be operated on until after the wound is nearly healed, only a small fistulous tract remaining.

In cases of superior pelvirectal abscess the incision should be made at right angles to the fibers of the levator ani muscle to facilitate drainage. In these cases tubal drainage is used.

**Imperforate Anus.**—There are two forms of this condition. In one form the rectum empties into the bladder, vagina, or urethra. In the other form there is no rectal opening either upon the surface of the body or in the genito-urinary organs. The diagnosis is usually at once apparent, except in cases in which the anus looks normal, when the diagnosis will often not be made until symptoms of obstruction arise.

**Treatment.**—If the rectum bulges when the child cries, open into it with a knife and keep the opening patent by inserting a plug of iodoform gauze. In cases in which the rectum is more deeply seated, a catheter is introduced into the bladder, an incision is made from the anus to the coccyx, the rectum is sought for, and when found is sewed to the anus and is incised. Keen, the author, and others have performed Kraske's sacral resection, pulling down the rectum to the anal margin, sewing it there, and incising the occluded anus. If the rectum cannot be found or cannot be pulled down, an artificial anus must be made.

**Fistula in ano** is the track of an unhealed abscess. An abscess in the anal region is apt to refuse to heal because of the constant movement of the parts (produced by respiration, coughing, the passage of gas, defecation, etc.). The passage of feces along the tract will keep a fistula open. If a tuberculous ulcer perforates a tuberculous sinus forms, and a tuberculous sinus is also apt to follow a cold abscess of the ischiorectal fossa. Fistula is often associated with phthisis pulmonalis, and is not unusually linked with piles, cancer, or stricture.

There are three varieties of fistula—the blind external (Fig. 780, A), the blind internal (Fig. 780, B), and the complete (Fig. 780, C). The *external*

opening is usually near the anus, but may be far away, and there may be only one pathway or there may be several sinuses and openings. In a healthy individual the external orifice is small and a mass of granulations sprouts from it. In a tuberculous fistula the external orifice is large and irregular, with thin and undermined edges, shows no granulations, extrudes small quantities of sanious pus, and the skin about it is purple and congested. In a fistula following an anal abscess the *internal* opening is just above the anus, between the two sphincters. In fistula following an ischiorectal abscess the internal opening is usually near the anus, but may in rare cases be above the internal sphincter. A sinus may run up under the mucous membrane from the internal opening. In a *horseshoe fistula* the internal opening is usually upon the posterior wall of the bowel, "and from this a tract leads into the ischiorectal fossa, not on one side only, but upon both. Therefore we have one opening into the bowel and one through the skin on either side."[1] In some cases of horseshoe fistula there is no internal opening; in other cases there are two openings. In an old fistula the track becomes fibrous and cannot collapse. Two or more fistulæ may exist in the same patient. In dealing with a fistula always determine if the condition is stationary or progressive.

The **symptoms** of a complete fistula are the passage of feces and gas through the opening and the flow of a discharge which stains the clothing. In a complete fistula a probe can be carried from the external opening into the bowel. After a time incontinence of feces is apt to come on, repeated attacks of inflammation thickening the rectum and destroying its sensibility. From time to

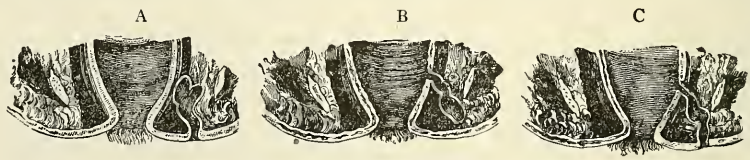



Fig. 780.—Fistula in ano: A, Blind external; B, blind internal; C, complete (Esmarch and Kowalzig).

time the opening will block and new abscesses form. In examining a fistula use Brodie's probe, as its flat handle enables one to locate the direction the bent instrument has taken, and its slender shaft will find its way through a very small channel.

**Treatment.**—In treating a fistula cleanse the parts, as cleanly work, though it will not prevent pus, will limit suppuration. The external parts are washed with soap and water. The rectum, which must be empty, is irrigated with warm saline solution. Corrosive sublimate should not be used in the rectum because it is irritant, causes a flow of serum, and hence lessens tissue resistance, and is rendered inert as an antiseptic by being converted into sulphid of mercury. Anesthetize the patient with ether unless the fistula is tuberculous, in which case use local anesthesia, spinal anesthesia, or nitrous oxid. Ether is avoided in such cases by many surgeons for fear of the existence of a pulmonary focus. A tuberculous focus in the lung may disseminate after inhalation anesthesia. Place the patient in the lithotomy position. If operating upon a complete fistula the usual method is as follows: Pass a grooved director into the external opening, carry it through the sinus, make it enter the bowel, bring its point out externally, and lift the tissue between the sinus and the surface. If the director ascends above the internal opening, the opening must be made into the bowel from the summit of the sinus. If there is no internal opening, make one. Incise the bridge of tissue which is held up on the director (Fig. 781). Cut

[1] "Diseases of the Rectum, Anus, and Sigmoid Flexure," by Joseph M. Mathews.

the sphincter at a right angle to its fibers, and do not cut it more than once at one operation. If a fistula is non-tuberculous, cut with a knife. If it is tuberculous, divide the tissues with a Paquelin cautery in order to lessen the danger of dissemination of the infection. Push the finger to the depth of the wound, to determine that the sinus does not ascend above the internal opening. Search with a small probe for branching sinuses, and if any are found, slit them open. Examine carefully to see if there is a sinus beneath the mucous membrane of the bowel, and if such a sinus is found, slit it up. Curet all sinuses, and if they are very fibrous, clip away the fibrous wall by scissors. Cut away diseased skin, irrigate the wound with salt solution, pack firmly with iodoform gauze to prevent oozing, and dress with gauze and a T-bandage. The packing is removed in twenty-four hours unless it is soiled earlier, in which case it is promptly removed. After twenty-four hours the wound is irrigated and packed lightly with gauze to its full depth. This dressing should be repeated every day and any bridging of the tissues should be broken down by a probe. If the wound becomes sluggish, it is stimulated with nitrate of silver and sodium iodid is given in small doses three times daily.

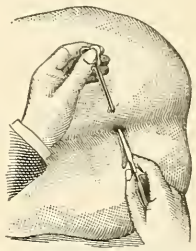


Fig. 781. — Operation for fistula in ano (Esmarch and Kowalzig).

The bowels should be moved after forty-eight hours by enema. The diet should be light and fluid for the first few days after operation, and the bowels should not be restrained by drugs. Get a tuberculous patient out of bed as soon as possible. If there are two fistulæ, cut one through, and when one wound has healed, cut the other. Some straight sinuses can be extirpated and the parts sutured, primary union occasionally resulting.

If a blind external fistula does not heal, every sinus must be incised, and thickened walls must be cut away or scraped away.

In a blind internal fistula an external incision is made to convert the case into a complete fistula, which is then treated as directed above.

In horsehsoe fistula more than one operation may be necessary in order to avoid cutting the sphincter muscle twice in one operation, a proceeding which would probably lead to fecal incontinence. One side alone is operated on at one séance. Sinuses are opened and scraped, the sphincter is divided, the angles and edges of skin are trimmed away, and the wound is packed. When the wound is healed, or nearly healed, the other side should be operated upon.

Any operation will fail if it is not done thoroughly. Operative failure is common after fistula operations. The operation described above usually gives excellent results in simple fistula. It gives reasonably good results even when the internal opening is between the sphincters, although in that case it may leave distinct impairment of fecal control. If the internal opening is in and above the internal sphincter, the operation just outlined leaves as a legacy definite loss of sphincter control and great consequent discomfort (Mackenzie, "Treatment of Fistula in Ano"). For such cases, and particularly for horseshoe fistulæ, Mackenzie (Ibid.) does an operation which does not mutilate the sphincter. He dilates the sphincter; finds and dilates the internal orifice of the fistula; lifts the mucous membrane around it; pares it in the direction of the long axis of the bowel; trims the muscle in the direction of the circumference of the sphincter; sutures the muscle with catgut, the stitches passing at right angles to the fibers; sutures the mucous membrane with chromic gut; makes a flap on the side involved, the flap including the fistulous tract and all of its branches (in a horseshoe fistula he makes a flap on each side); removes all scar from the rectal wall and enfolds the wall by catgut over the stitches within; removes all

of the fistulous tracts from the flap, sutures the fat with buried sutures of catgut, inserts a small drain, and closes the skin.

If fecal incontinence results from an operation for fistula, remove the scar tissue and endeavor to suture the separated muscular fibers. A. W. Mayo Robson ("The Practitioner," Feb., 1903) performs the following operation for incontinence: A crescentic incision, from ½ to ¾ inch in depth and taking in about one-half of the circumference of the bowel, is made at the anterior border of the anus. The middle borders of the incision are then pulled apart until the ends of the cut approximate in the middle line, when they are stitched with deep catgut sutures and the skin is sewed with silkworm gut, the immediate result being an incision apparently radiating from the anus. Should an operation be undertaken for fistula if phthisis exists? Many of the old masters said *no*. Mathews sums up the modern view: In incipient phthisis operate; in rapidly progressive fistula operate whether cough exists or not; if much cough exists do not operate unless the fistula is rapidly progressive; in the last stages of phthisis do not operate.

**Pruritus of the anus** is a symptom and not a disease. It may be due to piles, fissure, seat-worms, eczema, nerve disturbance, kidney disease, gout, jaundice, constipation, inebriety, the opium-habit, torpid liver, dyspepsia, alcohol, tea-drinking, vesical calculus, tobacco-smoking, urethral stricture, uterine disease, diabetes, ovarian trouble, and mental disorder. In some cases it seems to be a pure neurosis and no special causative factor can be recognized. It is vastly more frequent in males than in females, and is especially common in fat men who sweat profusely. It is seldom seen before the age of thirty, except in children suffering from thread-worms. The itching comes on gradually and usually intermittently, but grows progressively worse and worse until it becomes torturing. In many cases it is at first noticed only when warm in bed; in other cases it exists day and night. A violent exacerbation may be excited by worry, anxiety, overwork, dietary indiscretion, a sudden change of temperature, and many other things. The itching finally becomes an unbearable agony, sleep, except in snatches, is impossible, the appetite disappears, the strength fails, and the sufferer may become a nervous wreck. In some cases of pruritus the anal folds are edematous, there are abrasions here and there from scratching, the area is white and moist and gives origin to a fine secretion; in other cases the mucous membrane is dry and fissured.

**Treatment.**—In every case first of all make a careful examination to find a probable or a possible cause, local, reflex, or constitutional, and endeavor to remove this supposed cause. Then undertake treatment for the pruritus. It is very important to prevent constipation. Kelsey directs that the parts be cleansed twice a day, and after each cleansing the following ointment be applied: Menthol, 1 dr.; cerat. simp., 2 oz.; oil of sweet almonds, 1 fl.oz.; acid. carbolic., 1 dr.; pulvis zinc. oxid., 2 oz. Mathews commends the following mixture: Chloral, 1 dr.; gum-camphor, ½ dr.; glycerin and water, each, 1 oz.[1] In this disease a "scarf skin" forms, which must be made to peel off by the application of iodin, pure carbolic acid, corrosive sublimate (4 gr. to 1 oz. of cosmolin), or calomel (2 dr. to 1 oz. of cosmolin). In obstinate cases paint the parts, night and morning, with a mixture of 60 gr. of alum, 30 gr. of calomel, and 300 gr. of glycerin; or smear with an ointment compossed of ½ part of oleate of cocain, 3 parts of lanolin, 2 parts of vaselin, and 2 parts of olive oil (Morain). In very severe cases, in which the skin is dry and cracked, apply a 5 per cent. solution of eucain to the abraded portions and paint the entire surface with a concentrated solution of silver nitrate. It may be necessary to repeat this treatment several times at inter-

[1] "Diseases of the Rectum."

vals of four or five days. Adler advised us to apply to the parts the day after the silver has been used unguentum hydrargyri nitratis in full strength, only discontinuing on the day a fresh application of silver is made, and the next day resuming the applications of ointment. If during treatment the skin becomes sore, use calomel ointment until soreness disappears. Violent attacks of itching are met by applying hot water and black wash or calomel ointment. This plan of treatment must be pursued for some months (Lewis H. Adler, Jr., "New York and Phila. Med. Jour.," July 29, 1905). I have used this plan with some satisfaction. In severe and protracted cases we may employ the *x*-rays twice a week (J. R. Pennington). I have seen their application productive of great benefit. In some cases we employ the Paquelin cautery, in others we resect the mucous membrane, as in Whitehead's operation for hemorrhoids. Ball divides the sensory nerves going to the implicated skin and has obtained excellent results.

**Fissure of the anus** is an irritable ulcer at the anal orifice producing spasm of the sphincter. Pain exists because twigs of nerves are exposed upon the floor of the ulcer. Fissure is caused by constipation or traumatism. It is usually posterior.

The **symptom** is violent, burning pain, sometimes beginning during defecation, but usually at the end of the act, and lasting for some hours. Constipation exists, and often pruritus. Examination discloses a fissure, usually at the posterior margin, running up the bowel $\frac{1}{4}$ to $\frac{1}{2}$ inch. Piles often exist with fissure.

**Treatment.**—The *palliative treatment* is to prevent constipation, to wash out the rectum with cold water, and apply an ointment made by evaporating 2 oz. of the juice of conium down to 2 dr., and adding it to 1 oz. of lanolin and 12 gr. of persulphate of iron. Pure ichthyol frequently promotes healing unless the edges are thick or the base indurated, when operation must be done. Many cases are so sensitive and painful to the touch that medication and examination are almost impossible. These may be made bearable by insufflations of orthoform.

*Operative Treatment.*—Anesthetize the patient. Thoroughly cleanse the parts. Some surgeons advocate operation without stretching the sphincter. It has always been my custom to stretch the sphincter for fissure. Stretching gives us room in which to work, and by thus paralyzing the muscular fibers the raw surface is put at rest and paroxysms of pain cease to occur. In order to stretch the sphincter the patient is anesthetized, the surgeon's thumbs are inserted into the rectum, and the parts are stretched slowly until the thumbs touch the ischia. After stretching the sphincter the fissure is incised through its base $\frac{1}{4}$ inch deeper than the deepest part of the ulcer, extending $\frac{1}{4}$ inch above and below the diseased tissues, so that the healthy muscular fibers at either end are divided. In cases in which the ulcer is at either the anterior or posterior commissure a V-shaped incision is made, the apex of which should begin $\frac{1}{4}$ inch above the highest point of the ulcer and the diverging line running close to the sides of the fissure, but in healthy tissue.

A search is made by a probe to be sure no pockets exist. Any pocket must be opened and scraped. The floor should be curetted and touched with the solid stick of nitrate of silver. If there are redundant edges, exuberant granulations, a sentinel pile, or a polypus, they should be curetted or excised. The wound is then packed lightly with gauze and the patient kept in bed for twenty-four hours.

**Hemorrhoids, or Piles.**—There are three varieties of varicose tumors of the rectum, namely: *external*, which take origin without the external sphincter; *internal*, which take origin within the external sphincter; and *mixed* hemorrhoids, which are a combination of the two.

External hemorrhoids are covered with skin. Internal hemorrhoids are covered with mucous membrane. The term "external hemorrhoids" is not strictly accurate, as hemorrhage does not occur in external piles, and all external piles are not related to the external hemorrhoidal veins. An external pile may involve the veins or the skin.

**External hemorrhoids** are classified as thrombotic, varicose, inflammatory, and connective-tissue external hemorrhoids (Tuttle).

**Thrombotic External Hemorrhoids.**—These are external hemorrhoidal veins filled with clot. When an external hemorrhoidal vein inflames the parts become itchy, painful, and swollen, and defecation increases the pain. The blood clots in the inflamed vein and sometimes the vessel ruptures.

*Symptoms and Treatment.*—External piles of this variety are usually, but not always, multiple. Small oval tumors appear beneath the skin or the junction of the skin and mucous membrane. They appear suddenly. The parts itch and pain, defecation increases the pain, and each pile increases rapidly in size. When the vein ruptures, a livid, soft enlargement rapidly forms. External piles of this variety may be absorbed, may become organized into a scar, or may suppurate. These piles do not bleed. In treating external hemorrhoids some surgeons merely use remedies to combat the inflammation. An old plan of treatment is to incise the blood-tumor, turn out the clot, and pack with a bit of iodoform gauze. Mathews freezes the part or injects cocain, catches up the blood-tumor with a volsellum, excises the tumor and the tabs of inflamed skin, dusts the part with iodoform, and dresses it with antiseptic gauze. The bowels should not be allowed to move for two days. Never inject external piles with carbolic acid; it causes great inflammation, violent pain, sloughing, and is not free from danger. If the patient declines operation, order rest, a non-stimulating diet, avoidance of tobacco (Mathews), the use of saline purgatives, injections into the rectum of cold water several times a day, sponging of the anus frequently with hot water, and the application of hot poultices. As the acute symptoms begin to disappear use extract of hamamelis locally; when they have nearly subsided, apply zinc ointment.

**Varicose External Hemorrhoids.**—They are varicose external hemorrhoidal veins and are visible at the anal margin when the patient strains. They rarely produce pain or discomfort, and it is seldom that operation is necessary. The bowels should be moved daily, but not with violent purgatives, and after each movement cold should be applied to the anus, while the patient is recumbent. Tuttle advocates the use at night of an ointment containing 2 dr. of suprarenal extract and 6 dr. of lanolin; this is spread on cotton-wool, which is applied to the anus and held in place by a T-bandage.

**Inflammatory Piles.**—By this term we mean edematous inflammation of the anal folds. The inflammation may be due to a traumatism, the presence of an ulcer or fissure, etc. There are burning, itching, and swelling of the anus, which are all greatly increased by defecation. One or more pear-shaped swellings can be seen at the anal margin.

In some cases medical treatment produces cure. This treatment consists, during the first twenty-four hours, in the use of cold and of rest in bed. After this period heat should be employed. Tuttle applies gauze soaked in a 25 per cent. solution of boroglycerid and places a hot-water bag over this. He also recommends the following ointment, to be applied two or three times a day:

℞. Morphinæ sulph. ..... gr. v–x;
Ichthyol ..... ʒiv;
Ung. belladonnæ } ..... āā ʒj.
Ung. stramonii }
Sig.—Apply two or three times a day.

If these means fail, ether is given, the sphincter is stretched, and the tumors are cut away.

**Connective-tissue External Hemorrhoids (Skin Tabs).**—They are due to hypertrophy of mucocutaneous tissue at the anal margin. Usually they result from acute inflammatory external piles; sometimes they arise gradually as a result of chronic anal or rectal inflammation or irritation, and they may be due to varicose or thrombotic external piles (Tuttle). They produce no trouble when not inflamed. If they cause serious annoyance the treatment is extirpation.

**Internal hemorrhoids** are varicose tumors of the internal hemorrhoidal plexus, and are found internal to the external sphincter, just within the anus, and they prolapse easily. They are not simply varicosities, but new tissue has been formed, and they are, in reality, vascular tumors. They are covered with mucous membrane. *Capillary* piles are small, sessile, with a surface like a mulberry, and bleed freely. Children are not very liable to develop piles, excepting the capillary form. *Venous* piles are the most common variety. They extend from just above the anal margin of the rectum for an inch or more. They are purple in color, soft, irregular in outline, and are usually multiple. They bleed when irritated by hard fecal masses, but not so easily as the capillary piles. Each pile is composed of a varicose vein, some fibrous tissue, and a few arterial twigs. *Arterial* piles are very unusual. They are large, smooth, pedunculated, bleed easily and freely, and contain, besides a distended vein, arteries of some size.

Anything producing venous congestion in the rectum—constipation, diseases of the rectum, enlargement of the prostate, pregnancy, tumors of the womb, congestion of the liver, cirrhosis of the liver, certain diseases of the heart and lungs, sedentary occupations, relaxing climate, and stricture of the urethra —may cause hemorrhoids.

*Symptoms and Treatment.*—If there is neither bleeding nor protrusion the piles give no trouble. The first symptom is usually hemorrhage, and rectal examination by the speculum will make clear the condition. After a time, during defecation, the piles protrude; they may reduce themselves when the patient stands up, or it may be necessary to push them in. Pain does not exist in uncomplicated cases, and pain during or after protrusion means "abrasion, fissure, or ulceration" (Mathews).

*Palliative Treatment.*—This will not cure, but it will give great comfort. Some people only suffer at rare times when the liver is congested, and such subjects will not submit to operation. Remove, if possible, the cause (alcohol, irritating foods, want of exercise, etc.); restrict the diet; insist on regular exercise; give a course of Carlsbad salt, and follow this by the administration of bichlorid of mercury ($\frac{1}{24}$ gr. after each meal). Prevent constipation by a nightly dose of extract of cascara. After each bowel movement wash the parts with a soft sponge soaked in cold water, and syringe out the rectum with cold water and dry outwardly with a soft rag. If the hemorrhoids prolapse after restoring them and injecting cold water, insert a suppository containing 10 gr. of the extract of hamamelis and use another suppository at bedtime. A useful suppository for prolapse is that employed by Tuttle: it contains 5 gr. of ichthyol, 5 gr. of tannic acid, $\frac{1}{3}$ gr. of ext. of stramonium, $\frac{1}{3}$ gr. of ext. of belladonna, and 10 gr. of ext. of hamamelis. Bleeding may be arrested by suppositories, each containing 5 gr. of suprarenal extract. When the piles prolapse and inflame, rub Allingham's ointment on the parts (2 dr. each of ext. of conium and ext. of hyoscyamus, 1 dr. of ext. of belladonna, and 1 oz. of cosmolin). Mathews uses 12 gr. of cocain, 1 dr. of iodoform, $\frac{1}{2}$ dr. of ext. of opium, and 1 oz. of cosmolin. Grant uses an ointment containing 8 gr. of morphin, 12 gr. of calomel, and 1 oz. of vaselin. This is applied after bathing

the part with hot water. If the piles are protruding and reduction cannot be effected, put the patient to bed, give a hypodermatic injection of morphin, and apply hot poultices. If reduction cannot soon be effected, divulsion of the sphincter must be practised or radical operation must be resorted to.

*Operative Treatment.*—Give a saline the morning before and an enema the evening before the operation, and wash out the rectum well the morning of the operation. In treating by *injection of carbolic acid* the sphincter should be divulsed while the patient is under the influence of nitrous oxid gas unless the pile is thrombotic. "Under gas muscular relaxation does not obtain as in the use of ether. Hence dilatation under gas can be more rapidly induced, as we have the sphincteric rigidity as a guide in knowing exactly how much force may be employed in the individual case" (Lewis H. Adler, Jr., in "Jour. Am. Med. Assoc.," Jan. 21, 1905). The surgeon must be careful not to tear the parts. The tumors are drawn out or, if gas was not given, the patient strains them out. An injection is given by a hypodermatic syringe into the center of the pile, and as each pile is injected it is pushed into the rectum. Only one or two piles are injected at each séance, and the operation is not repeated for one week (Geo. W. Gay, in "Boston Med. and Surg. Jour.," Dec. 5, 1901). The dose for each pile is 1 or 2 min. of a 10 per cent. solution of carbolic acid. The injections relieve the condition, but are rarely absolutely curative and are not without

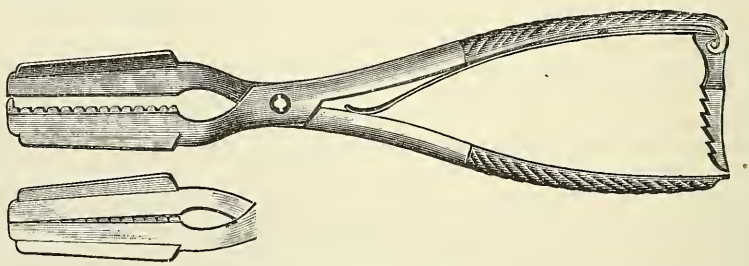


Fig. 782.—Brick's pile clamp.

danger, and may produce abscess, sloughing, hemorrhage, phlebitis, pyemia, stricture, and even death (W. T. Bull-Kelsey). Dr. Collier F. Martin ("American Medicine," August 27, 1904) maintains that the method is safe and satisfactory. He injects equal parts of a French preparation of phenol and distilled water, freshly mixed and filtered. From 7 to 15 min. are injected into a pile and only one pile is injected at a séance. In from five days to one week another injection may be given. Before beginning a course of injections the sphincter is stretched while the patient is under nitrous oxid and oxygen. It is not necessary to repeat this for future injections. During injection a special speculum is used. The pile protrudes into the speculum, is cleansed with a 1 per cent. solution of creolin, and the injection is thrown into the most prominent part of the pile. The speculum is withdrawn before pulling out the needle. This maneuver prevents escape of injection and arrests bleeding. I seldom employ the injection treatment. I never use it if the patient consents to the clamp and cautery operation or to ligation. The *clamp and cautery* is, in the great majority of cases, the operation of choice. It requires but a few minutes to do it; after it is done there is little or no postoperative pain, in very many cases retention of urine does not occur, and the patient usually is about again within ten days. The patient is anesthetized and the sphincter is carefully and thoroughly stretched. The stretching of the sphincter is very important. It gives free access to the parts, prevents subsequent spasm and pain, and lessens the likelihood of venous bleeding after operation. The

pile is caught by forceps and drawn outside of the sphincter. Many use Smith's clamp. It is applied with the ivory surface against the mucous membrane of the bowel. I use the clamp devised by Dr. J. Coles Brick (Fig. 782). From the bite of Brick's clamp the pile cannot slip, as the blades come evenly and firmly together. The pile is cut off and the stump is seared with the Paquelin cautery at a dull-red heat. Pile after pile may be thus treated, care being taken to leave some mucous membrane at each side of every pile. If this precaution is not taken, healing will be slow and stricture will result. After cauterization is complete a speculum is inserted and the blades are widely opened. Any bleeding-points are at once ligated. This is a most important precaution. Packing is never inserted. I formerly used it, but have given it up. It is of no service and produces severe pain and edema. The treatment from this point is identical with that advised below after the use of the ligature. *Excision* is preferred by Allingham. He stretches the sphincter, holds it open with a retractor, catches up the pile, cuts it off, and twists the bleeding vessels. Some prefer to pass a silk or catgut suture, cut off the tumor, and tie the thread (Fig. 783). *Whitehead's operation* (Fig. 784) is only to be performed in severe cases, when the piles are extremely large and form a protruding circular mass. Primary union is rarely secured. When first introduced the operation was viewed with favor, but experience shows it is sometimes followed by disastrous consequences.[1] Stricture not infrequently arises after its performance; fecal incontinence occasionally results, and anal anesthesia with inability to restrain the passage of gas is common. After this operation the anus is permanently more or less moist. The entire pile-bearing area of mucous membrane is dissected out and the cut margin of mucous membrane is pulled down and stitched to the surface. The sphincter may be dilated as a preliminary measure.

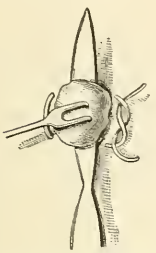


Fig. 783. — Extirpation of hemorrhoids (Esmarch and Kowalzig).

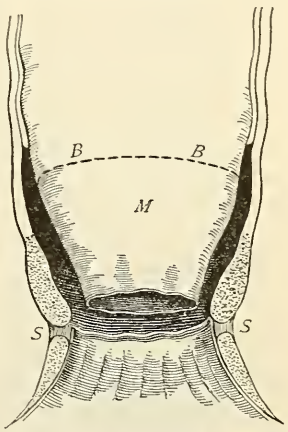



Fig. 784.—*S*, *S*, The lower circular incision along Hilton's white line; *M*, tube of mucous membrane dissected from the sphincter; *B*, *B*, dotted line showing the place for the upper circular incision (Edmund Andrews).

The *application of the ligature* is an easy and useful method. It is not so rapid as the cautery, is followed by more pain, healing requires a longer time, and stricture is more common. In this operation, after anesthetizing, stretch the sphincter and treat each hemorrhoid separately. Catch a pile with a pair of forceps or a volsellum, pull it down, and cut a gutter through the skin-margin if the pile is of the mixed variety; tie the small piles without transfixing, but transfix the large piles; tie with silk (coarse silk for the large piles, finer silk for the small piles); cut off each tumor beyond the thread and cut the ligatures short. Treat the other piles in the same manner. Irrigate with hot normal salt solution. Do not insert packing. Place a 2-gr. opium suppository in the rectum. Apply a gauze pad and a T-bandage over the anus. The opium locks up the bowels. The patient is kept on a light diet for three days, at the end of which time a saline may be given. Just before the bowels act remove the dressings and give an enema of warm water or of

[1] Andrews, in "Mathew's Medical Quarterly," Oct., 1895.

glycerin. After the movement wash the parts first with dilute peroxid of hydrogen and next with hot salt solution, dust with iodoform, and apply a gauze pad over the anus. Irrigate daily until healing is complete. After the tenth day examine with a speculum to see that the ligatures have come away; if any are found in place, remove them.

**Prolapse of the Rectum.**—If the mucous membrane is prolapsed the condition is commonly called *prolapsus ani;* if the entire thickness of the rectal wall is prolapsed, it is commonly called *prolapsus recti* (Fig. 785). The term "prolapse of the anus" is an incorrect and objectionable one, and we should designate such cases *prolapse of the rectal mucous membrane, incomplete* or *partial prolapse.* If all the coats of the bowel descend the condition should be called *complete prolapse* or *procidentia.*

**Incomplete Prolapse (Partial Prolapse).**—In this condition the mucous membrane of the rectum protrudes from the anus. In normal conditions the

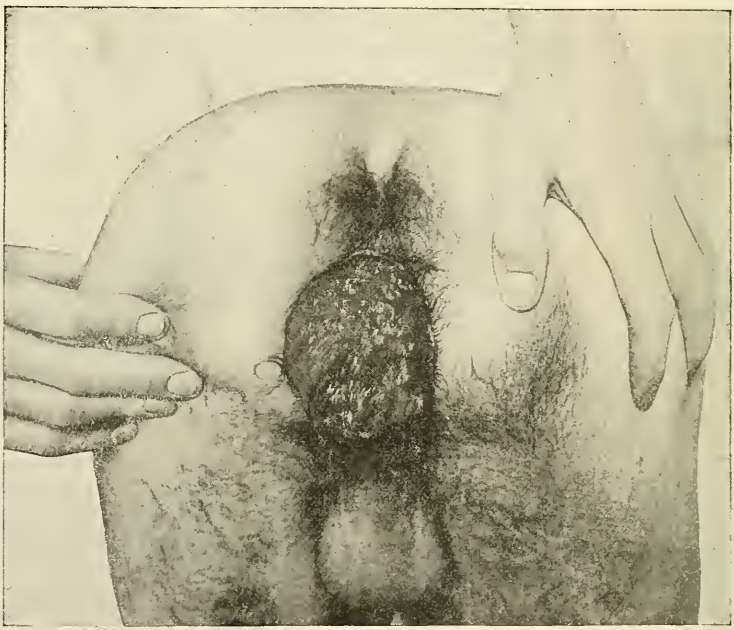


Fig. 785.—Rectal prolapse.

membrane protrudes during defecation and at once retracts when the act of defecation terminates. In the condition under discussion the mucous membrane remains protruded because the submucous tissues being stretched and relaxed find it difficult or are unable to draw the mucous membrane in again. In this condition a ring of mucous membrane or only a portion of its circumference may protrude. It is particularly common in early youth and in old age. Prolapse is apt to occur from excessive straining at stool and is commonest in feeble, ill-nourished children. A polypus may be the cause. Piles and worms may lead to prolapse. Straining from phimosis, stone in the bladder, or urethral stricture may be causative. Its development is favored by the use of articles of food which cause frequent movements of the bowels. If an individual sits a long time on the seat of the closet or on the chamber the development of prolapse is favored. The condition comes on gradually and is at first painless. For some time it reduces itself spontaneously after defecation, but reduction becomes more and more difficult. A common custom of sufferers is to push it

by the fingers above the grasp of the sphincter. Sometimes, but seldom, it becomes strangulated. A recent prolapse is pink, an older one is angry red, one which is tightly caught is purple, a strangulated one is deep purple and soon becomes black from gangrene. If the prolapse is of the entire circumference it shows radial folds of mucous membrane. It frequently bleeds. Prolapse, be it large or small, tends to recur again and again, and eventually the mucous membrane inflames, ulcerates, or sloughs. Prolapse of all the coats may ensue (see below). The condition is sometimes confused with hemorrhoids, but in prolapse the protruding mass is circular and has a depression in the center, whereas hemorrhoids are distinct masses. Further, hemorrhoids are very rare in children. Hemorrhoids often exist with prolapse of the mucous membrane and frequently cause it. In prolapse of the mucous membrane there is no sulcus between the sphincter muscle and the anterior portion of the protrusion, in complete prolapse there is.

**Treatment.**—*Palliative* treatment forbids straining at stool and amends an improper diet. Phimosis must be corrected; stone in the bladder must be crushed or cut for and removed; stricture must be dilated; hemorrhoids and polypi are to be removed. Give an enema of cold water just before going to stool in order to hurry the emptying of the rectum. If prolapse occurs, the protrusion must be bathed with cold water and restored. Constipation must be prevented (enemata of water or glycerin may be used), and after each movement several ounces of an infusion of white oak bark (1 ounce of quercus to a pint of water) should be injected. If a prolapse is caught firmly, paint it with cocain and adrenalin, place the patient in the knee-chest position, apply hot compresses, grease it with cosmolin, insert a finger into the rectum, and apply taxis around the finger (Mathews). If this fails, cover a finger with a handkerchief and insert the wrapped digit into the rectum; if this proves futile, invert the patient before manipulation. Do not give a general anesthetic unless it is imperatively necessary; the vomiting so often caused by ether and chloroform might reproduce the prolapse. After reduction apply a compress upon the anus, direct that it be worn when at stool, and before each act of defecation give an injection of cold water containing an astringent (tannin or fluidextract of hydrastis). Most cases in children can be cured without operation. Some cases in adults and obstinate cases in children may be treated by painting the prolapse with fuming nitric acid, greasing it with olive oil, and restoring it. Some cases require excision of the mucous membrane, the divided edge of this membrane being stitched to the skin. In other cases the protrusion is stroked longitudinally with the actual cautery and restored. When the surgeon comes to operate for recurring prolapse, it will often be found to have modestly withdrawn and he may be obliged to stretch the sphincter to bring it into view.

**Complete Prolapse (Procidentia).**—In this condition all of the rectal coats protrude. There are said to be three degrees of this condition. In the first degree the prolapse begins at the anal margin and its outer surface is continuous with the peri-anal skin, there being no sulcus between. It is usually a consequence of prolapse of the mucous membrane. In the second degree (genuine procidentia) the prolapse begins on a level or above the level of the lower margin of the peritoneum and projects out of the anus. In the third degree the prolapse begins at the origin of the rectum or in the sigmoid, and is, in reality, a protruding intussusception. Some cases come on suddenly. The prolapse of the first degree has no furrow between it and the perianal skin, and the folds are circular instead of longitudinal. It is usually treated as is prolapse of the mucous membrane. If simple methods fail, it will be necessary to do an operation to lessen the size of the anal orifice. In prolapse of the third degree the treatment is as for intussusception. It is prolapse of the second degree we

consider here. It is more common in adults than in children. There are many theories as to its causation. Esmarch regards it as due to inflammation of the rectum, which spreads to all the coats and also to the perirectal structures. Another theory (Jeannel's) is that the small intestine is in a state of ptosis, and by falling into Douglas's culdesac presses upon and causes atrophy of the levator ani muscle. The most probable theory is that the condition is, in reality, hernia (Waldeyer, Zuckerkandl, Moschchowitz). Moschchowitz ("Surg., Gynec., and Obstet.," July, 1912) makes a powerful argument to prove that the intestine in Douglas's culdesac causes a bulge of the anterior rectal wall, that this bulging part of the wall grows larger and descends, and that finally the entire circumference of the rectum is dragged down.

Complete prolapse is usually preceded by chronic constipation, great straining being necessary to effect defecation. Chronic rectal catarrh is also a common antecedent.

A mass sticks out of the rectum and there is a purulent discharge which often contains blood. The protruding mass (which is seldom over 6 inches in length) is covered externally with mucous membrane (which may be normal, inflamed, ulcerated, or bleeding) and is lined by normal mucous membrane (Moschchowitz, Ibid.). The opening is at the apex of a canal directed backward. The anterior part of the protrusion is greatly larger than the posterior part, the anterior part is usually tympanitic, and the posterior part dull on percussion (Moschchowitz, Ibid.). There is a deep furrow between the anterior portion of the sulcus and the anal margin. The furrow, as a rule, is about 1 inch deep.

Early in the case the prolapse is reduced spontaneously, later it must be reduced by the hand. It may become incarcerated or strangulated. The protrusion when strangulated becomes gangrenous.

*Treatment.*—In children the condition may often be cured by the methods used for prolapse of the mucous membrane. If a prolapse is caught tightly, apply hot compresses to reduce swelling, paint it with cocain and adrenalin, put the patient in the knee-chest position, and reduce as described under partial prolapse. General anesthesia is undesirable because of the probability of vomiting with consequent straining. After reduction apply a support. Electricity is used by some, injections of astringents under the mucous membrane by others. Operation is necessary for prolapse in most adults and in some children. As a preliminary, hemorrhoids, ulcerations, etc., must be cured. A multitude of operations have been recommended. Some try to constrict the anal opening (cauterization by the actual cautery, nitric acid or chlorid of zinc, removing a wedge-shaped piece from posterior rectal wall followed by suturing, twisting the rectum, injections of paraffin about the sphincter, and various other plans). Such operations are entirely inefficient. Some operate on the bowel higher up, striving to increase support. Moschchowitz also mentions "methods which pay particular attention to the fixation apparatus of the rectum." One plan is to operate through an abdominal incision, fixing the sigmoid to the abdominal wall—an unphilosophical operation "because the distal end of the sigmoid is fixed to the sacrum, and pulling can have very little, if any, effect upon the prolapse of the rectum" (Moschchowitz, Ibid.).

I have abandoned the operation of stitching the sigmoid to the abdominal wall (*colopexy*) because it is dangerous to thus anchor the gut and because, I believe, it always fails to cure. Mikulicz's operation of excision of the prolapse has elements of decided danger, but may cure the case. I apparently cured 1 case this way, but in another case the condition recurred.

**Moschchowitz's operation** is the most reasonable one. The abdomen is opened, the rectum is held taut, and the culdesac of Douglas is obliterated by silk or linen sutures. The pelvic fascia is included in the sutures.

**Ulcers of the rectum** are divided into the simple traumatic, the syphilitic, the tuberculous, the dysenteric, the gonorrheal, and the malignant. *Simple* ulceration is due to abrasion with fecal masses or a foreign body, the abraded area ulcerating. It may follow an operation for piles and also protracted labor ("Diseases of the Rectum," by Allingham), and is apt to be single. The base and edges of a simple ulcer are neither prominent nor hard and stricture rarely forms. *Syphilitic* ulceration is a tertiary lesion commonest in women. There are numerous small ulcers of the mucous coat or submucous tissue, but little indurated, with sharp-cut edges which are not undermined. These ulcers fuse and constitute one large irregular ulcer; fibrous tissue forms in the wall of the bowel, induration becomes noticeable, and stricture follows. There is profuse discharge and fistulæ are apt to form. Such ulcers may be surrounded by nodules of a bluish color. In many cases the first condition is stricture due to the formation of masses of fibrous tissue in the rectal walls, and ulceration occurs secondarily. In syphilis there may be a breaking down of a huge gummy mass or of multiple gummata. It has been proved by the microscope that tuberculous ulceration may arise in the rectum. *Tuberculous* ulceration presents a conical ulcer with overhanging edges and a pale-red base. There is some mucous discharge, some tenesmus, and a little pain. Tuberculosis is seldom *directly* responsible for stricture (see page 1186). Dysentery, catarrh, diabetes, Bright's disease, neoplasms, and foreign bodies may produce ulceration of the rectum.

**Symptoms.**—There may be merely uneasiness about the rectum, but sometimes there is severe burning pain on defecation, and perhaps for some time after the act. There may be constipation or diarrhea, the patient strains at stool, and the stools may contain blood, mucus, or pus. As a rule, there is diarrhea on rising in the morning, the first movement consisting of blood and mucus, and the next movement being fecal. The history should be carefully inquired into; tuberculosis should be sought for; the question of syphilis should be investigated. A digital examination enables the surgeon to feel the ulcer, and an examination by means of an ordinary speculum or an electric proctoscope brings it into view.

**Treatment.**—In *simple* ulcer empty the bowel by the administration of a saline cathartic, wash out the rectum with hot water after the saline has acted, introduce a speculum, touch the ulcer with pure carbolic acid or silver nitrate (40 gr. to 1 oz.), place the patient in bed, restrict him to a liquid diet, and every day inject iodoform and olive oil (10 per cent.) or insufflate iodoform into the rectum. If this fails, give ether, stretch the sphincter, incise the ulcer through its entire thickness, and cauterize with fuming nitric acid, caring for the case subsequently as we would a patient who had had piles ligated. In *tuberculous* ulcer improve the general health, send the patient to a genial climate, or at least into the sunlight and fresh air, prevent constipation, give nutritious food, especially fats, wash out the rectum every day with hot water, and insufflate iodoform or inject iodoform emulsion. Touch the ulcer once a week with silver nitrate (10 gr. to 1 oz.). In *syphilitic* ulcer give antisyphilitic treatment and treat the ulcer locally, as is done in tuberculous ulcer. *Dysenteric* ulcer requires injections of hot water, the touching of the ulcer with pure carbolic acid, insufflations of iodoform, and special treatment of the dysentery.

**Benign Tumors of the Rectum and Anus.**—These tumors may be of the connective, epithelial, or muscular tissue type. Of the first there are fibroma, enchondroma, lymphadenoma, lipoma, and myxoma; of the second, adenoma and papilloma; of the third, myoma and fibromyoma. Many benign tumors appear in polypoid form. A polyp is a tumor with a pedicle. A polyp may be a fibroma, myxoma, myoma, papilloma, or adenoma. Papilloma is a rare growth, usually has a broad pedicle, but may have no pedicle.

It does not occur in childhood. A papilloma bleeds profusely and causes a discharge of mucus. A polyp causes no pain unless ulceration occurs. It causes bleeding and mucous discharge. On coming from stool the patient feels as though the rectum still contains feces. A polyp may protrude during defecation. It can be detected by digital examination and can be readily seen through a proctoscope. There may be one polyp; two, several, or many polypi. Polypi are most common in children. (See Adler, in "Annals of Surgery," Dec., 1909.) The most common forms of tumor are the myxoma, hypertrophied solitary follicles, adenoma, fibroma, and lipoma. Myxoma is most common in children. Fibroma originates in the connective tissue or the submucosa and is very rare. It may arise at the upper end of a fissure. It has a long pedicle and may become very large.

**Symptoms** of tumor are dull, aching pain, tenesmus, frequent defecation, and sometimes ulceration of the mucous membrane. In a non-polypoid benign tumor there is absence of hemorrhages and of mucous discharges.

**Treatment.**—Remove the tumor after dilating the sphincter. In tying or snaring off polypi it should be borne in mind that the peritoneum may be invaginated in the pedicle and, therefore, no traction on it should be made when operating. Sessile growths are dissected out. The postoperative treatment is practically the same as for hemorrhoids.

**Cryptitis** (*Inflammation of the Crypts of Morgagni*).—These crypts, five to ten in number, are situated in the mucous membrane of the rectum, about 3 cm. from the anus. They occasionally become packed with mucus or feces. There is quite severe pain referred to the site, especially after defecation. In this condition the examination of the anus by the finger is extremely painful. The inflamed crypts may be detected by examination through the speculum.

**Treatment.**—Divide the affected crypts, curet away any granulations, and allow the parts to heal.

**Non-cancerous stricture of the rectum** may be congenital or acquired. There are two forms of acquired stricture: first, stricture due to external pressure; second, stricture due to primary narrowing of the rectal lumen.[1] Stricture due to external pressure is very rarely complete, and may be caused by bands of adhesions or a tumor growth. The second form may be produced by syphilitic tissue, ordinary inflammatory tissue, cicatrices after operations, sloughing, tuberculous, syphilitic or dysenteric ulceration, rectal gonorrhea, and traumatism. The usual seat of simple stricture is from 1 inch to 1½ inches above the anus. The deposit may be limited to the submucous coat or all the coats may be involved. It is very seldom that stricture arises as a result of abrasion from fecal masses or foreign bodies. It may follow an operation for piles if considerable tissue is removed, and is an occasional sequence of Whitehead's operation. Stricture due to dysentery is extremely rare, and no case has ever been reported to the United States Pension Office (Peterson). The existence of stricture as a result of rectal gonorrhea has not been positively proved. A majority of sufferers from rectal stricture have labored under syphilis, but it is not probable that the lesion is syphilitic in all or even in most of them. The stricture may be due to the formation of fibrous tissue and ulceration may or may not occur. It may be caused by the contraction and healing of a large ulcer. Some maintain that tuberculous stricture does occur. Mathews dissents from this view and points out that the disposition of tuberculous matter is to break down, and before the rectum can be strictured from tuberculosis it breaks down from ulceration. Peterson[2] says a large proportion of the victims of rectal stricture die of phthisis, and that one-third of so-called syphilitic cases are tuberculous. It may begin as an ulcer or as an infiltration of submucous tissue. Although a syphilitic or a tuberculous

[1] Reuben Peterson, in "Jour. Am. Med. Assoc.," Feb. 3, 1900. [2] Ibid.

lesion may cause rectal stricture, in most cases such lesions simply expose the tissues to infection, and benign rectal stenosis results from the infection. Tuberculosis may cause stricture, but does so indirectly rather than directly.

The **symptoms** of rectal stricture are constipation, pain on defecation, straining at stool, perhaps the presence of blood and mucus in the stools, an open anus, and the passage of stools flattened out into ribbons if the stricture is low down. In some cases there is fluid diarrhea, solid fecal matter being retained above the stricture. The stricture is found by the finger or by the soft-rubber bougie, used with the utmost gentleness and care. A stiff instrument or the rough use of any instrument would be dangerous and might rupture the rectum. In syphilitic cases, in tuberculous cases, and in benign cases the fibrous thickening is usually in the submucous coat, and in syphilitic and tuberculous cases the mucous membrane is apt to ulcerate. It is said that complete obstruction may arise. I have seen obstructive symptoms, but never *complete* obstruction in rectal stricture. Distention of the abdomen and colic are very usual.

The **treatment** of non-cancerous and primary narrowing of the rectal canal is rest, non-stimulating diet, warm-water injections, mild laxatives, and hot hip-baths. Cocain suppositories may be needed. Any existing disease is treated. Bougies are passed every other day. Use a soft-rubber bougie, warmed and oiled, and introduce it gently. If only the method of gradual dilatation is employed the patient must for the remainder of his life pass a bougie from time to time. For fibrous strictures forcible dilatation (*divulsion*) by a special instrument is employed or incision is practised. Incision (*proctotomy*) may be either external or internal. In internal proctotomy one or more incisions are made from the rectum through the stricture down to healthy tissue, the first cut being in the middle line posteriorly. External proctotomy, which divides the sphincters, is apt to leave incontinence as a legacy. Electrolysis finds some advocates, but on what grounds it is difficult to see. In some cases the rectum should be removed. In incurable cases perform inguinal colostomy.

**Cancer of the rectum** is the cancer of the bowel most often met with. According to Abbe ("Keen's Surgery") rectal carcinomata constitute three-fourths of all intestinal tumors. Its growth may be primarily malignant or may arise from an adenoma. The commonest growths are composed of cylindrical cells, and may be either soft or scirrhous. In cases secondary to epithelioma of the anus ordinary epithelioma arises.

In most rectal carcinomata the cells present a tubular arrangement surrounded by a more or less plentiful stroma of connective tissue. In soft tumors the connective tissue is scanty; in hard tumors it is plentiful.

Cancer is most common after the age of forty, but it not unusually occurs before the thirty-fifth year, and is sometimes seen as early as the twenty-fourth year or even earlier. "Of 115 cases of cancer of the rectum at the Rostock Clinic, 4 occurred in patients between fourteen and seventeen years of age" (Miles Porter, in "New York Med. Jour.," Feb. 10, 1912). Extensive ulceration occurs. If a hard ring encircles the rectum the lumen of the tube is greatly and progressively diminished. In cases of diffuse infiltration the lumen is not greatly lessened. In growths involving the anus the inguinal glands are involved and also the glands in the hollow of the sacrum. In growths limited to the rectum proper the glands back of the peritoneum in the sacral hollow are involved, and the inguinal glands are involved late or not at all.

**Symptoms.**—In the beginning and for a considerable time after there are no symptoms. There may be none for a year or more. Symptoms begin with ulceration or constriction. The symptoms of rectal cancer are like those of non-malignant stricture, except that the pain is usually greater and the hemorrhage more severe. Constipation is apt to alternate with diarrhea. The diarrhea

is usually in the morning. Unfortunately, in many cases symptoms are long trivial; in fact, pain may be absent until the disease is far advanced. Mucopurulent or bloody stools are often thought to result from dysentery or hemorrhoids, which latter condition, however, may be only an accompanying condition of rectal cancer. The above symptoms may, on the patient's say-so, have been accepted by the physician, without any local examination, as caused by hemorrhoids. The patient, again, may have only imagined the presence of hemorrhoids, since, according to his notion, the above symptoms must result from hemorrhoids, with which condition so many of his friends with like complaints are afflicted. Loss of strength, emaciation, and cachexia are generally noticeable only in the late stages of rectal cancer. Only in the very latest stages the characteristic odor is perceptible, the patient becomes septic, and abscesses attended by gangrene may form (Ernest Jonas, in "Interstate Med. Jour.," No. 4, 1906). The finger and the speculum make the diagnosis. In rectal cancer metastasis occurs late. The most favorable cases for operation are those in which the growth is small and movable. Accurately define the extent of the growth, and endeavor to make out if it has invaded the cellular tissue outside of the rectum, the prostate, the bladder, the sacrum, the uterus, etc.

**Treatment.**—In every case of cancer of the rectum the following questions must be considered: Shall we perform a radical operation in hope of producing cure or at least greatly prolonging life? In what cases should a radical operation be attempted? It is the proper procedure if there are no metastatic deposits, if the patient is in fair general condition and free from serious organic disease, and if the cancerous bowel is movable and not fixed by dissemination to adjacent structures. As W. Watson Cheyne ("Brit. Med. Jour.," June 13, 1903) says, a slight adhesion to the vagina is not a contraindication, because this portion of the vagina can be readily removed with the diseased rectum. Some surgeons will not attempt radical operation if they cannot pass a finger through the growth. I do not regard high position as forbidding operation, although, of course, it makes it more dangerous to life and less promising as to cure. Cheyne is of the same opinion. When the surgeon is first called to a case of cancer of the rectum it is usually found to be so far advanced as to be inoperable. In at least 75 per cent. of my cases radical extirpation was impossible when I first saw the case.

If a radical operation is determined on, the next question to answer is, Shall we, or shall we not, perform *preliminary colostomy?* If the cancer is very low down, involves the anal canal, and is to be removed from the perineum, preliminary colostomy is rejected by many. I believe that even in such cases it should be done. If the cancer is high up and we propose to attack it by Weir's method or the Quenu-Mayo method, preliminary colostomy should not be done. If Kraske's operation is to be performed, I believe preliminary colostomy is usually indicated. It enables us to cleanse the area upon which operation is to be performed, and to keep the wound clean, and gives us a much better chance of obtaining primary union. In cases in which the sphincter is retained and it is possible to anastomose the divided ends of the rectum together, colostomy is not necessary; and if an artificial anus has been made in such a case, another operation will be required to close it. As a matter of fact, I have found it always difficult and usually impossible to suture the divided ends of the gut together after Kraske's operation, and I now follow the advice of Keen, and always precede Kraske's operation by colostomy. The abdominal incision necessary to reach the bowel to do colostomy may be used to permit of exploration, but it is wiser to have a median incision for this purpose. I consider exploration as of the first importance. It enables the surgeon to examine the outer surface of the rectum, to detect glandular involvement, to find out if there has been metastasis to the liver, and to

determine with certainty whether or not the cancer is operable. Several times in cases of small and apparently operable cancer of the rectum I have found on opening the abdomen extensive glandular involvement or unsuspected metastasis to the liver, and once I found another cancer 4 inches above the one to which attention had been directed. It is my custom to make a median incision for exploration and then a small incision through which to bring out the gut for colostomy. A large abdominal incision for colostomy is objectionable. I strongly object to operating at all on rectal cancer without a preliminary exploratory operation. If radical operation is rejected (and about three-fourths of the cases, when first seen by the surgeon, are obviously beyond such aid), palliative treatment is desirable. The best palliative treatment is the operation of inguinal colostomy. If this is refused, what shall be done? One plan is to introduce a tube through the stricture daily, wash out the rectum with warm water, and after washing inject emulsion of iodoform (10 gr. to 1 oz. of sweet oil). Injections of chlorid of zinc (1 gr. to 1 oz. of water) lessen the foulness of the discharge. The bowels are opened regularly by laxatives, and if the growth causes obstructive symptoms it is scraped away with a sharp spoon. Opium is given to relieve pain. The advantage of this plan is that the patient does not suffer from the unpleasantness of an artificial anus. Sooner or later, however, the growth gets outside of the bowel, and terrible pain will arise from involvement of the sacral plexus. W. Watson Cheyne ("Brit. Med. Jour.," June 13, 1903) would restrict palliative treatment of this character to cases in which fungating masses grow from one side of the bowel.

If a growth encircles the bowel and produces symptoms of obstruction, *palliative colostomy* should be performed. This operation gives great comfort to the patient and allays pain by intercepting the feces before they reach the cancer. I am not convinced that it distinctly retards the growth of the cancer or notably prolongs life. Unfortunately, colostomy does not do away with pain if the sacral plexus is involved. I have had no experience with radium in inoperable cancer of the rectum and have never seen the *x*-rays produce any marked or lasting improvement. *Operative treatment* includes one of several procedures. Excision of the rectum from below (*Cripps's operation*) is practised by some if not more than 3 inches require removal, if the peritoneum is not invaded, and if the adjacent organs are free from disease. The peritoneum must not be opened in Cripps's operation. After the growth is removed the divided rectum is pulled down and sutured to the skin. As the sphincter is sacrificed the condition would be dreadful without an artificial anus. A perineal anus without a sphincter is vastly more distressing than an inguinal anus. I seldom do perineal excision (it does not permit any considerable removal of lymph-glands). When of recent years I have done it I have preceded it by exploratory laparotomy and the formation of an inguinal anus. In some cases in women it may be possible to remove a low growth without

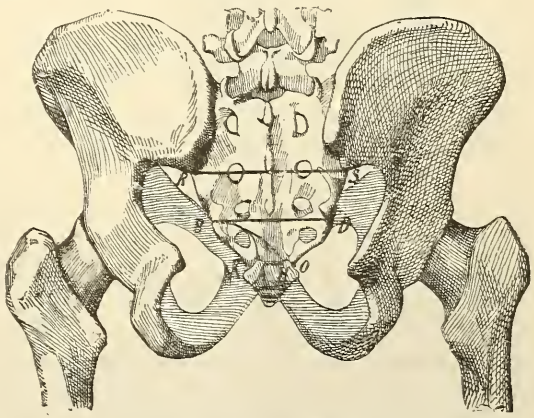


Fig. 786.—Different levels of resection of the sacrum: *K–O*, Kocher's line; *B–O*, Kraske's; *B–H*, Hochenegg's; *B–D*, Bardenheuer's; *R–S*, Rose's (Mass.).

damage to the sphincter through an incision in the posterior vaginal wall. Excision of the rectum after excising the coccyx and a portion of the sacrum (*Kraske's operation*, that is, excision of the rectum after sacral resection, Fig. 786, *B–O*) is a procedure which permits removal of the entire tube, portions of the colon, and even of adjacent parts. The peritoneum is opened deliberately in this operation, and is subsequently closed with sutures before the gut is opened. The glands from the mesocolon are always removed. The lower end of the upper segment of bowel is fastened in the wound, or, if colostomy has been previously performed, may be closed. In some few cases in which it is not necessary to remove the lower end of the rectum, the two portions may be anastomosed after resection of a part of the tube. Kraske's operation may be done by an osteoplastic method, the bone not being removed. It is well to precede a Kraske operation two weeks by an inguinal colostomy, which permits of cleansing the lower bowel of feces and lessens the chance of severe wound infection and delayed healing after the removal of the rectum: Preliminary colostomy may make the operation of extirpation more difficult by fixing the intestine, and thus interfering with the necessary drawing down of the gut (E. H. Taylor). If the growth is extensive and the mesocolon short, it may be best to perform right inguinal colostomy, but in most cases left inguinal colostomy is preferred (Gerster). The colostomy remains open during the patient's life, except in those rare cases of Kraske's operation in which the continuity of the rectum can be reëstablished after excision of the growth. In such cases the artificial anus may be closed some time after resection of the rectum.

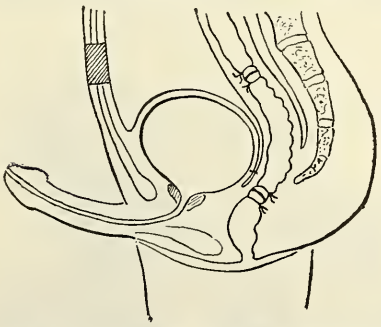


Fig. 787.—Tying off the tumor through an abdominal incision after separating peritoneum from sacrum and bladder (Weir).

Fig. 788.—Lower end of rectum everted through the anus and the upper end of bowel drawn out of the abdominal cavity (Weir).

Robt. F. Weir ("Med. News," July 27, 1901) has been so much impressed with the difficulties and dangers of Kraske's operation in a case of high carcinoma that he now employs it solely in cases in which there is freedom from disease for 2 inches immediately above the anus and in which the cancer does not extend more than 5 inches above the anus. In high cases he does the following operation: Open the abdomen above the pubes, separate the peritoneum so that the bowel and "contents of the sacral curve" are liberated behind nearly "to the tip of the coccyx and in front of the edge of the prostate." The tumor is then tied off with tapes (Fig. 787). The portion of the rectum bearing the tumor is removed, the lower end of the bowel is everted through the anus, and the upper end is drawn out of the abdominal incision (Fig. 788). The upper end is then caught with forceps and drawn through the everted lower end of the rectum (Fig. 789, *a*). The ends of the two everted portions (Fig. 789, *b*) are sewn together, the everted bowel is replaced, the divided peritoneum is sutured to shut off the peritoneal cavity, and posterior drainage is inserted (Fig. 790). In the Quenu-Mayo operation the object is

to remove all of the diseased glands as well as the cancer (Wm. J. Mayo, in "St. Paul Med. Jour.," April, 1906). The patient is placed in an exaggerated Trendelenburg position and the belly is opened by a median incision. The growth is studied to see if it is removable, and a search is made for enlarged glands which might cause us, and for secondary growths which would cause us, to abandon the operation. If we conclude to attack the growth, pack away all the intestine except the sigmoid, catch two clamps across the sigmoid, one of them being on the level of the sacral promontory. Divide the gut between them. Free the mesosigmoid by lateral cuts and bring the proximal stump out of the belly, ligate it, and apply a purse-string suture to invert it. A gridiron incision is then made on the left side and the proximal stump is pulled through it and is sutured there. Incisions are now made in the sides and in front to liberate the rectum, the inferior mesenteric artery is tied above and to the left of the promontory, the fat and glands are thoroughly removed from the sacral hollow, vessels being tied as cut, except the middle sacral and middle hemorrhoidal vessels, which are tied before division. The area is now packed with gauze and the patient is put in the lithotomy position. The rectum is packed with gauze, the anus is sutured, and the rectum is separated from the prostate and urethra or from the vagina from below upward to just above the levator

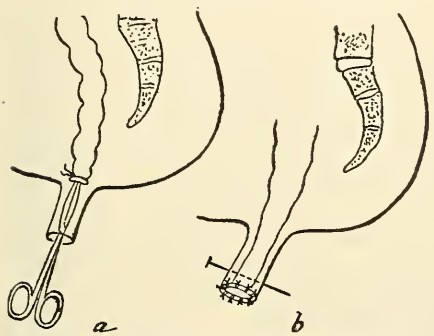



Fig. 789.—*a*, The upper bowel drawn out through the everted lower end of rectum; *b*, the ends of the two portions of the rectum sewn together (Weir).

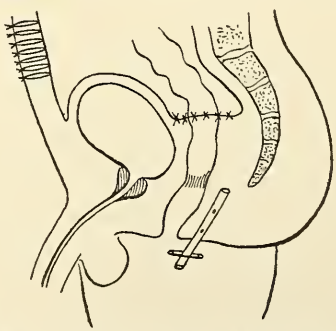


Fig. 790.—The united bowel replaced with posterior drainage and the divided peritoneum so sewn together as to shut off the general peritoneal cavity from the pelvis (Weir).

ani muscle. An assistant presses the cancer-bearing fragment carrying glands down from the abdomen and the surgeon removes it from the perineum. The peritoneum is sutured within the abdomen, room being left for a small drain which protrudes from the perineum. The perineal wound is narrowed by sutures and the wound in the belly is closed. In twenty-four hours the protruding end of the sigmoid is opened and an artificial anus is thus made.

(See Joseph A. Blake's views as to "The Operation of Choice in Carcinoma of the Rectum," "New York Med. Jour.," July 1, 1911.)

The mortality of Kraske's operation is from 12 to 15 per cent. Twenty-eight per cent. of Kocher's cases of extirpation of cancer of the rectum remain well from three to sixteen years after operation (W. W. Cheyne, "Brit. Med. Jour.," June 13, 1903).

## XXX. ANESTHESIA AND ANESTHETICS

**Anesthesia** is a condition of insensibility or loss of feeling artificially produced. An **anesthetic** is an agent which produces insensibility or loss of feeling. Anesthetics are divided into—(1) *general anesthetics*, as amylene, chloroform, chlorid of ethyl, ether, bromid of ethyl, nitrous oxid, and bichlorid

of methylene; (2) *local anesthetics*, as alcohol, bisulphid of carbon, carbolic acid, ether spray, cocain, eucain, stovain, ice and salt, rhigolene spray, and ethyl chlorid spray.

Anesthesia may be induced by a general anesthetic to abolish the usual pain of labor and of surgical procedures; to produce muscular relaxation in tetanus, herniæ, dislocations, and fractures; and to aid in diagnosticating abdominal tumors, joint diseases, fractures, and malingering.

Ether was first used as a surgical anesthetic by Crawford W. Long, of Georgia, in 1842, but he did not publish his cases until after William T. G. Morton, of Hartford, had given ether in public in 1846. Horace Wells gave nitrous oxid in 1844. Sir James Y. Simpson introduced chloroform in 1847.

**Death-rate from Anesthetic Agents.**—Sir Frederic W. Hewitt ("Anesthetics and Their Administration") combines the statistics of Julliard and Ormsby, with the following result:

| Anesthetic. | Total Number of Administrations. | Total Number of Deaths. | Death-rate. |
|---|---|---|---|
| Chloroform | 676,767 | 214 | 1 in 3,162 |
| Ether | 407,553 | 25 | 1 in 16,302 |

Hewitt regards the St. Bartholomew Hospital records as furnishing the most reliable statistics accessible. He takes them from 1875–1900. The fatality from chloroform was 1 in 1300; from ether (and gas and ether), 1 in 9319. It is to be noted that statistics covering many countries would indicate that chloroform becomes relatively safer in warm regions. In temperate regions it is relatively safer (compared with ether) in summer than in winter. Hewitt is of the opinion that ether is six times as safe as chloroform. Gwathmey ("Jour. Am. Med. Assoc.," Nov. 23, 1912) collects statistics from American sources. In 157,453 administrations of ether there were 28 deaths (about 1 death in 5623 administrations). In 16,390 administrations of chloroform there were 8 deaths (about 1 death in 2049 administrations).

Hewitt finds that during the last forty years only 30 fatalities are recorded as produced by nitrous oxid, and he thinks several of these should be excluded. It is practically certain, however, that many deaths, or at least some deaths, have not been recorded.

Seitz collected 16,000 instances of anesthesia by chlorid of ethyl, with 1 death. During a hospital experience of twenty-eight years I have seen anesthetics (particularly ether) given many thousand times. For five years I gave ether and chloroform for Prof. Keen. I have witnessed 3 deaths, each of which at the time was thought to be directly due to the anesthetic, and 1 of them was so caused. One death resulted from pouring a quantity of chloroform upon an Allis inhaler, the bandage of which was saturated with ether. At the time the chloroform was poured in the inhaler the patient had just been struggling and consequently was breathing deeply. One death resulted from giving ether on a thick cone made of several towels with paper between the folds. The towels were saturated with ether, the patient got no air at all, and was asphyxiated, as she might have been had the cone only been wet with water. The third case was a man who had an impacted hip fracture. He became cyanotic under ether while the impaction was being pulled apart and died. The death was supposed to be due to ether, but necropsy discovered fat emboli in the brain and lungs.

**Preparation of the Patient.**—Whenever possible prepare a patient before administering a general anesthetic, and prepare him, if the case admits of it, during two or more days. Heart disease is not a positive contra-indica-

tion to surgical anesthesia. It is quite true that anesthetics are dangerous to people with fatty hearts, but shock is also dangerous, and the surgeon stands between the Scylla of anesthesia and the Charybdis of shock. Gallant truly says that not enough attention is paid to the "character of the pulse and action of the heart before operation, by which to compare its work during anesthesia, and after the operation is over, and this neglect leads to unnecessary stimulation and overdriving a heart which is doing its average best."[1] Always examine the urine if the nature of the case allows time. If albumin is found, operation is not contra-indicated, but the peril of anesthesia is greater, and certain dangers are to be watched for and guarded against. If much albumin is present, postpone operation except in emergency cases. If sugar is found, the danger is considerable, as diabetic coma occasionally develops. The percentage of sugar does not determine the amount of danger. Coma may arise when only a little sugar is present, and may not arise when there is a considerable amount. The presence of aceto-acetic acid is more ominous than is the presence of sugar. Empty the intestinal canal by purgation a number of hours before anesthetization. It is well to give the bowel six to twelve hours' rest before operation. The usual custom is to give a saline cathartic the evening before operation and an enema early on the morning of the operation. Frequently the nature of the case or the necessity for haste does not permit of preliminary emptying of the intestine by the administration of cathartics. During the twenty-four hours preceding operation food should be taken in small amounts and in forms easily digestible. During the day or so before operation there is usually impaired digestion, and no undue strain should be put upon the stomach. In the morning allow no breakfast if the operation is to be performed at an early hour, but if the patient is very weak, order a little brandy and beef-tea. If the operation is to be about noon, give a breakfast of beef-tea and toast or a little consommé; *never* give any food within three hours of the operation, but brandy is admissible if stimulation is required. If the stomach is not empty at the time of operation, vomiting is almost inevitable, and portions of food may enter the windpipe; if the stomach contains no food, vomiting is far less likely to happen; and even if it occurs and vomited matter should enter the windpipe, it may do little harm, as it consists chiefly of liquid mucus. In cases of intestinal obstruction in which there has been stercoraceous vomiting there is much danger that vomiting will occur during anesthetization. In some cases of intestinal obstruction, during the administration of the anesthetic and during the anesthetic state, a stream of stinking brown fluid may flow without effort from the mouth. Vomiting or regurgitation of stercoraceous material is profuse, sudden, and dangerous. It may flood the bronchial tubes during inspiration and cause death by suffocation. In a case in which stercoraceous vomiting has occurred wash out the stomach before administering the anesthetic. If a patient with intestinal obstruction is too weak to permit lavage, a local anesthetic should be used instead of a general anesthetic. Vomiting while the patient is under the influence of an anesthetic is dangerous in any case, because of the great cardiac weakness which precedes and follows it. If a patient sleeps well the night before an operation, he will probably take the anesthetic better than if he sleeps poorly. Effort should be made to obtain a night's sleep. An excellent expedient is a hot ammonia bath, followed by a rub-down with weak alcohol.[2] It may be necessary to administer trional or bromid. About fifteen minutes before giving the anesthetic let the patient drink a glass of hot water. Water protects the stomach from the irritant effects of any anesthetic which may be swallowed. Before giving the anesthetic see that artificial teeth are removed and that the patient does not have a piece of candy or a chew of tobacco

[1] "Medical Record," Feb. 2, 1899. [2] A. Ernest Gallant, "Med. Record," Dec. 30, 1899.

in the mouth. Always have a third party present as a witness, because in an anesthetic sleep vivid dreams often occur, and erotic dreams in women may lead to damaging accusations against the surgeon. Place the patient recumbent. The effort should be to place him in as comfortable a position as possible if this position is consistent with operative necessities. Put a small pillow under him, so as to support the normal lumbar curve and prevent postoperative backache. See that the clothing is loose, particularly that there is no constriction about the neck and abdomen. Do not have the head high unless this position is demanded by the exigencies of the operation. The anesthetist must have a mouth-gag and a pair of tongue forceps at hand. It is very wrong to say that a mouth-gag and tongue forceps are never necessary. It is quite true they are often used when not needed, but this does not justify us in being without them when they are needed, and they may be needed very badly. The anesthetist should also have a pair of artery forceps and some small gauze sponges to swab out the mouth and throat. A hypodermatic syringe in *working* order, and solutions of strychnin, atropin, and brandy are to be in an accessible place, oxygen must be ready for administration, and it is well to have an electric battery adjacent. Accidents, it is true, are rare, but they may happen at any time, and hence the surgeon should always be prepared for them. Any danger which arises must be met promptly and decisively, or action will be of no avail. Many surgeons give a hypodermatic injection of morphin a short time before operation to steady the heart, to prevent vomiting during anesthetization, to shorten the stage of excitement, to prevent rigidity, and to aid the bringing about of insensibility with very little of the anesthetic. This method has been tried by many during the last forty odd years. It is used in drunkards (as their muscles tend to remain rigid in the anesthetic state), in those whom it is difficult to make completely unconscious, in neurotic individuals, and in badly frightened subjects. Its greatest use has been in operations about the mouth and face, for in these procedures an anesthetic was given on a towel or inhaler, was of necessity given intermittently, and a preliminary dose of morphin was found to keep the patients from rousing during the intervals. At present intratracheal anesthesia does away with the need of morphin in operations about the mouth and face. There are objections to giving morphin before anesthesia, and its use should be the exception and not the rule. It should not be used in children, in cases of stupor, or in cases in which the respiratory center is disordered. It depresses the respiration, lowers temperature, and thus perhaps increases operative shock, interferes with the pupillary phenomena of anesthesia, delays awakening from the anesthetic sleep, adds to subsequent abdominal distention and headache, and actually favors postanesthetic vomiting. Hewitt ("Anesthetics and Their Administration") says that several recorded fatalities were due to the combination. If the surgeon determines to give morphin, he gives $\frac{1}{6}$ to $\frac{1}{4}$ gr. twenty minutes before the anesthetist begins to give the anesthetic. Hewitt (Ibid.) says: "The anesthetic should be given until the usual signs of anesthesia commence to appear. . It should then be discontinued for a few moments and only reapplied as occasion may require. As little as possible of the ether or chloroform should be subsequently administered; the conjunctival reflex should be retained." In the clinic of the Jefferson Hospital the elder Gross long used as a routine the preliminary administration of opium, but during his later years he used it exceptionally. His successor, the younger Gross, used morphin hypodermatically exceptionally. In some cases we may anticipate trouble from the anesthetic. Cyanosis may occur in drunkards; in fat, thick-necked individuals of the Major Bagstock type, who are short of breath and congested in appearance; in individuals with some disease of the lungs, bronchi, pharynx, larynx, or trachea (empyema, emphysema, chronic bronchitis, croup, cancer of the larynx, etc).;

in individuals suffering from fatty heart or valvular incompetence. Buxton points out that an individual without teeth and with stenosis of the nares is apt to become cyanotic under an anesthetic, because the lips and pillars of the fauces are drawn in like valves during inspiration.

**Ether and Chloroform.**—The two favorite anesthetics are ether and chloroform. Only the very best ether or chloroform should be used. It is a good plan, in order to lessen bronchitis, to mix with ether turpentine of Pinus pumilio in the proportion of 20 drops to 6½ oz. (Becker, in "Centralbl. f. Chir.," June 1, 1901). Chloroform is more dangerous than ether in general cases, though it is more agreeable, less irritant to the lungs and kidneys, and quicker in its action. Chloroform, compared to ether, is relatively safer in warm than in cold countries. In fact, in the tropics it is a matter of considerable difficulty to use ether because of its great volatility. It should, however, be noted that Squire used ether successfully when the temperature was 120° F. in the shade ("Lancet," vol. i, 1913). Chloroform is preferred in campaigns, because less is required and transportation is easier. Recovery from chloroform is quicker and quieter than that from ether, but chloroform vomiting lasts longer than ether vomiting. Chloroform may induce sudden and even fatal syncope. Hare's experiments on animals indicate that chloroform may kill by respiratory failure occurring secondarily to failure of the vasomotor center; but certain it is that clinically a danger of chloroform is paralysis of the heart, and this condition may come on so rapidly that death may occur almost before an attempt can be made to save life. Leonard Hill has proved that most chloroform deaths that take place after considerable of the anesthetic has been taken arise from paralytic distention of the heart. Sudden death, when inhalations of chloroform have just commenced, may be due to the irritant vapor acting on the nasal mucous membrane, exciting a nasal reflex, and powerfully stimulating cardiac inhibition. If ether produces danger it does so usually through the respiration, and not the heart, and there is generally time to undertake means of resuscitation, which means are apt to be successful. Chloroform is preferred to ether by many surgeons for children under ten years of age, in whom ether causes a great outflow of bronchial mucus, which may asphyxiate; for people over sixty, entirely free from myocardial disease, at which age most persons have some bronchitis, and ether chokes them up with mucus. Ether also irritates the kidneys, which at the latter age are apt to be weak or diseased. Personally, I give ether even to infants (if they are free from bronchitis) and to old subjects without marked respiratory trouble. Chloroform is given if the actual cautery is to be used about the face, neck, or mouth, because ether vapor may take fire and chloroform vapor will not. Chloroform is preferred for labor cases, when moderate anesthesia only is required, and was preferred for operations on the mouth and nose before the advent of intratracheal anesthesia. In cleft-palate operations chloroform is usually preferred, because it causes but little cough and salivary flow. In ligation of a large artery which is overlaid by a vein ether greatly enlarges the vein, but this is no real embarrassment to an experienced surgeon. In goiter operations ether will decidedly enlarge the veins. Most goiters may be, and many should be, removed with the aid of local anesthesia only. Chloroform is particularly dangerous when there is myocardial disease, and is apt to produce cyanosis and embarrassed respiration. In valvular heart disease chloroform is more dangerous than ether, and even in functional heart trouble it is an undesirable anesthetic. It should not be used in those who smoke or chew tobacco to excess, or who overindulge in coffee or alcohol. Chloroform is more dangerous in shock than ether. A patient in dangerous shock requiring operation should, if possible, have the nerves coming from the part injected with cocain so as to prevent

further shock by introducing a "physiological block"—Crile (see page 262). Chloroform is preferred for patients with difficult respiration from any cause other than heart disease (in emphysema, bronchitis, or pulmonary tuberculosis), and for patients with kidney disease. I am convinced that etherization is sometimes responsible for a latent area of pulmonary tuberculosis becoming active. Some surgeons do not use ether in abdominal operations because they believe it may cause persistent oozing of blood, but this view is not in accord with the author's experience. Ether is the best and safest anesthetic for general use. It is much safer than chloroform in valvular disease and functional heart trouble. It is dangerous in myocardial disease, but not nearly so dangerous as chloroform. In valvular disease without heightened arterial tension it is reasonably safe, but in valvular disease with heightened arterial tension it is dangerous. Ether is dangerous when atheroma exists. Both ether and chloroform may induce changes in the blood.[1] In practically all cases they produce a diminution of hemoglobin and leukocytosis. In some cases they produce alteration in the shape of the corpuscles. These changes are especially marked in anemic blood. Ether produces distinct leukocytosis, probably toxic in origin. Both ether and chloroform seem to lessen the phagocytic activity of leukocytes, and hence to lower vital resistance (Ferguson, in "New York Med. Jour.," May 11, 1912). These blood changes indicate that prolonged anesthesia must militate against recovery from a severe operation. If a patient's hemoglobin is below 30 per cent., a general anesthetic should not be given. During the state of anesthesia the temperature drops from 1 to 3 degrees or more, hence the patient should be carefully covered during the operation. The question as to the effect of ether on the kidneys is much disputed. Most surgeons believe that it tends to cause albuminuria or increase existing albuminuria. Nitrous oxid is very dangerous when there is vascular degeneration, and it may induce apoplexy. It is also dangerous if the air-passages are narrowed as by a goiter. In giving ether or chloroform the administrator must devote his undivided attention to the task. He must note every symptom, must order or carry out proper treatment for complications, and must keep the operator informed as to the necessity for haste. The anesthetist must be a man who has a wholesome respect for ether and chloroform, although not afraid of them.

Can an anesthetic be administered to a sleeping person without waking him? I know that chloroform can be so given, for I have succeeded in giving it to a child without breaking the slumber. Probably, in most cases, an attempt will fail, but in some it will succeed. Stone ("Cleveland Med. Jour.," Jan., 1902) reports successful administration to sleeping children and also the chloroforming of a resident physician while asleep. Paugh ("Jour. Am. Med. Assoc.," May 18, 1901) reports three successes with children. Ether, because of the irritant nature of its vapor, would be more apt to arouse a sleeper than chloroform.

**Administration of Chloroform.**—Chloroform should be given only by a highly trained man. In fact, safety in giving chloroform is dependent upon skill and experience more than in giving ether. No one should think of allowing anyone but a physician to give chloroform. The most dangerous period is when the patient is incompletely anesthetized, but is going under. Most deaths happen at this time. In administering chloroform have at hand a mouth-gag, tongue forceps, artery forceps, small gauze sponges, a clean towel, a hypodermatic syringe, solutions of strychnin, atropin and brandy, an electric battery, and a can of oxygen. Use only *pure* chloroform. The

[1] See the author on the "Blood-alterations of Ether-anesthesia," "Medical News," March 2, 1895, and also the author and Kalteyer in "The Proceedings of the American Surgical Association for 1901."

patient must be recumbent. No special inhaler is required, but the drug may be given upon a thin towel, a napkin, or a piece of lint. The mask of Skinner (Fig. 791) is very useful. Junker's inhaler (Fig. 792) is used by many anesthetists. In operations about the face Souchon's instrument is serviceable. Souchon's apparatus is so arranged that chloroform may be given through a tube which is introduced through the nose, the instrument being well out of the way of the operator. Some surgeons cocainize the nares before giving chloroform, so as to prevent the supposedly dangerous nasal reflex (Rosenberg). It is advisable to smear the lips and face with cosmolin to prevent blistering. The chloroform vapor must be well mixed with air. The chloroform is sprinkled on the fabric from a drop-bottle. Raise the napkin well above the mouth, add 5 drops of chloroform, and tell the patient to take deep and regular breaths, but do not tell him to breathe forcibly. Forcible respiration may lead to cessation of respiration. Add a few more drops of chloroform, and when the patient grows so accustomed to it that it does not choke, turn the wet part of the fabric toward the face and place it near the mouth; do not touch the mouth with the wet lint, because it will blister. If the drug is given *gradually*, struggling is not usually violent or prolonged. Never pour on a large amount at one time. Keep the lower jaw pushed forward during the time the chloroform is being given. Cough

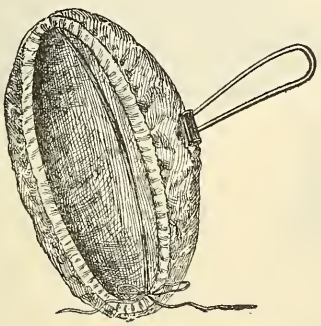


Fig. 791.—Skinner's mask.

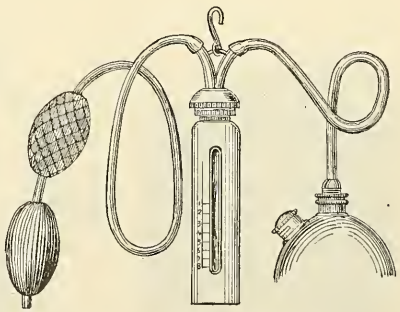


Fig. 792.—Junker's inhaler.

and vomiting at this time mean that the vapor is too strong. During the stage of excitement do not suspend the administration of chloroform unless respiration becomes difficult, in which case suspend it until the patient takes one or two respirations. If the patient struggles, do not hold him, and push the administration of the drug. He holds his breath while struggling, and as struggling ceases takes full, deep breaths. If the inhaler is saturated with chloroform, he may inhale a dangerous amount during the deep respiration after struggling. Chloroform given in considerable amount when the patient is breathing deeply from the effects of ether is unsafe. If chloroform is given subsequent to anesthetization by ether, it should be given gradually and well mixed with air. When the patient becomes anesthetized, give just enough of the drug to keep him so. While a patient is taking chloroform hiccup usually means that vomiting is going to occur. If vomiting occurs at this time more chloroform must be given to abolish the reflexes. Deep and sighing respiration and repeated swallowing indicate that more of the anesthetic is required. Stop the administration or give very little when shock becomes evident or when there is profuse hemorrhage. Chloroform vapor is not inflammable, hence it is safer than ether when a hot iron is to be used about the face and when there is a lighted lamp or a stove in a small room; but the presence of a naked gas-flame decomposes chloroform

into irritant products of chlorin ($COCl_3$), which cause the patient and the surgeon to cough.

**Chloroform and Oxygen.**—The use of this mixture was suggested by Neudorfer. Some anesthetists advocate the mixture of chloroform and oxygen, asserting that it does not produce spasm of the glottis or muscles of respiration, that it does not produce cyanosis or weakness of circulation, that it does not irritate the kidneys, is safer to life than pure chloroform, and is less often productive of severe and prolonged vomiting. These alleged advantages are probably stated with rather undue emphasis, although I do believe the mixture has less tendency to produce cyanosis than has the pure drug, does not so often induce vomiting, and is somewhat safer. Hewitt does not think that the method offers any "special advantages" ("Anesthetics and their Administration," by Sir Frederic W. Hewitt). If this method is used, a bag containing oxygen is attached to the hand-bellows attachment of a Junker inhaler, and oxygen is forced through the chloroform and flows to the face-piece.

**Administration of Ether.**—The administration should not be intrusted to a novice. The anesthetist should be one of your most trusted men. I do not believe in allowing a nurse to give ether. She cannot have sufficient knowledge to observe incipient trouble, to anticipate outward effects, and to

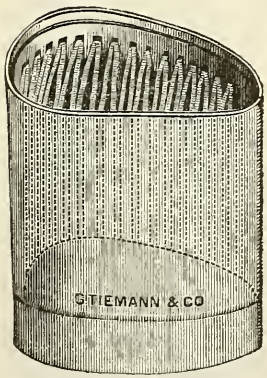



Fig. 793.—Allis's ether inhaler.

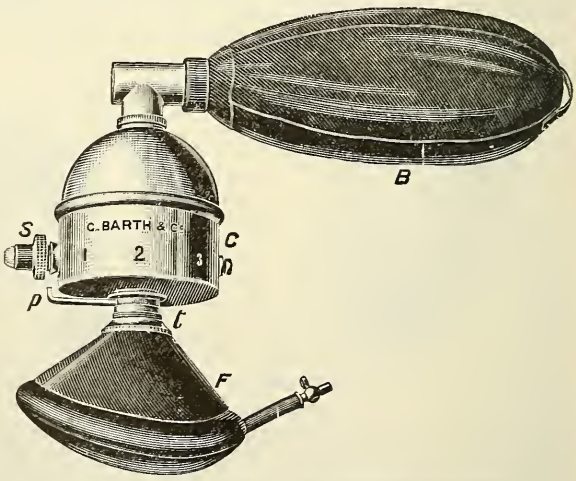



Fig. 794.—Clover's portable regulating ether inhaler.

at once do the correct thing when difficulty arises or calamity impends. The moral responsibility and the legal responsibility demand that a physician give the ether. Ether is best given from a partially open inhaler. The most satisfactory appliance is Allis's inhaler (Fig. 793). This inhaler secures a plentiful supply of air. Before being used the metal frame is scalded, dried, and threaded with a clean gauze bandage. The end of the frame which is to be toward the mouth is covered with one layer of gauze. The frame is then inserted in a clean metal case and the case is wrapped in a clean towel. The drug is given drop by drop, the drops following each other in regular sequence. This is known as the *drop method*. The drop method is the safest plan and the most comfortable to the patient. Instead of Allis's inhaler a piece of gauze or an Esmarch inhaler may be used. Some surgeons prefer closed inhalers. The Clover inhaler (Fig. 794) is popular in England. *F* is the face-piece; *C*, a reservoir of ether through which the air-current passes; *B* is an India-rubber bag. In this apparatus there is no provision for the entrance of fresh air. By turning the reservoir *C* on the tube *t* the amount of current passing over the ether can

be regulated. When this apparatus is used, the ether vapor breathed into the lungs is expired into the bag and is rebreathed. This inhaler, if used by a skilful man, is very useful; but any lack of watchfulness or skill will permit of cyanosis, and the very young, the senile, the anemic, and feeble are best anesthetized by the drop method described above.

An admirable detailed account of anesthetization by the closed method will be found in Sir. Frederic W. Hewitt's treatise on "Anesthetics and Their Administration," and in Mr. Dudley W. Buxton's treatise on "Anesthetics, Their Uses and Administration." When giving ether, have at hand the same drugs and appliances as when chloroform is given, and keep the lower jaw pushed forward during the administration. When using Allis's inhaler, take every care that none of the ether runs through on to the face and into the eyes. If it does it will irritate and perhaps blister. Even the vapor irritates the eyes, and boric acid solution should be used now and then during the administration to flush out the conjunctival sacs. It is wise to grease the face and lips with cosmolin. Place the dry inhaler over the mouth and nose, let the patient take several breaths, that he may gain confidence, begin to drop ether into the inhaler, let the patient take several more breaths, and so on, gradually increasing the rapidity with which the drops of ether are given. If he tends to struggle, diminish the amount of ether for a time, but do not hold him. Do not tell him to breathe forcibly. Forcible breathing is liable to cause a cessation of respiration. Never suddenly add a large amount of the anesthetic: it causes coughing and often vomiting. When the patient becomes thoroughly anesthetized, give a very little ether as often as is required to maintain unconsciousness. When bleeding is profuse or shock is marked, suspend the administration of ether or give very little of it. If he rolls his eyes from side to side, if the respirations are deep and sighing, if there are repeated movements of swallowing, more anesthetic should be given (Tarnowsky). Hiccup is often preliminary to vomiting, and always means that the reflexes are returning. If a hot iron is to be used about the face, remove the inhaler and fan away the ether before bringing the cautery near. Have any light set high up, as ether vapor is heavier than air, and no explosion is possible until it reaches the level of the flame. If the vapor takes fire, cover the patient's mouth and nose with a towel.

The patient should be kept in a condition in which he feels no pain, makes no movement, and is not rigid. In this condition the cutaneous reflexes are abolished, the breathing is regular and quiet, the color is good, and the pupils react to light, though slowly. Just enough anesthetic must be given to cause the patient to pass into this condition and remain in it. To give any more is to poison him. The amount necessary varies with the individual and the operation, and requires skill, experience, and attention on the part of the anesthetist.

The old idea that we must poison a man into dangerous coma has been abandoned. When the breathing becomes louder, more rapid, or spasmodic, it means that reflexes are returning, and a little more anesthetic should be given.

**Ether and Oxygen.**—This mixture is useful in certain cases in which respiratory difficulty exists, particularly in empyema. If during the administration of ether cyanosis tends to occur, it is often advantageous to give oxygen with the ether. The process of anesthetization by ether and oxygen is somewhat slower than by ether vapor mixed with air. It can be given by inserting beneath the Allis inhaler or pushing deep down into it, from above, a tube attached to a reservoir of oxygen and from which a stream of oxygen emerges.

**Intratracheal Insufflation Anesthesia (Method of Meltzer and Auer).**—A flexible elastic tube much smaller in diameter than the trachea is

carried down almost to the tracheal bifurcation. When air is forced in it emerges between the tube and the trachea. The addition of ether vapor to the air makes the maintenance of anesthesia easy. Ether can thus be administered by means of a tracheal tube, a Wolff bottle to contain the ether, and a foot-bellows. I have had it given in this way a number of times. Elsberg's apparatus (see Fig. 570) permits of the most desirable method.

The patient is first anesthetized by the ordinary method. When unconscious the head is dropped back over the end of the table, and the tube is passed into the trachea "under the guidance of the eye by means of a Jackson direct laryngoscope" (see Fig. 562). (See Brewer, in "Keen's Surgery," vol. vi.) In this form of anesthesia the stream of air carries off ether vapor and prevents accumulation in the tubes.

A degree of anesthesia necessary to maintain muscular relaxation will not apparently cause dangerous symptoms. If the ether is given for a long time in unnecessarily large amounts danger may be reached, but it is attained gradually and not suddenly and gives warning. If there should be an ominous fall in blood-pressure and respiratory failure, cut out the ether vapor and use the apparatus for artificial respiration. A patient remains very still under this method of anesthesia, awakes rapidly from it, and suffers little from shock and postoperative vomiting. Nitrous oxid and oxygen have been given by this method. Meltzer is studying chloroform so used ("Keen's Surgery," vol. vi). In operations about the head, mouth, and neck insufflation anesthesia keeps the anesthetist out of the operator's way. In operations about the mouth the method prevents the inhalation of blood or vomit. In goiter operations it is a safeguard against sudden suffocation. In intrathoracic operations it serves to prevent collapse of the lung. After the operation all ether vapor is driven out of the lungs by a stream of fresh air. Peck ("Annals of Surgery," July, 1912) maintains that the insufflation method is safe if certain rules are carefully followed. These rules are to avoid excessive pressure (which might rupture air-cells), be sure not to spray liquid ether into the tracheal tube, do not carry the tube into the gullet or beyond the tracheal bifurcation, and do no damage by rough introduction. Meltzer (Loc. cit.) refers to 3 deaths.

**Rectal Etherization.**—Pirogoff suggested this method in 1847 and Roux employed it the same year. The method is only used in operations about the face, tongue, nasopharynx, pharynx, mouth, and larynx; in other words, in cases in which, were the ether given by inhalation, the operator and anesthetist would interfere with each other. The rectum should be emptied by a purgative enema the day before the operation, and again the morning of the operation, and a short time before giving the ether the rectum should be irrigated with warm salt solution. A dose of laudanum is given a few hours before, or an injection of morphin and atropin twenty minutes before the administration of the ether. We should employ an apparatus of the type of Buxton's, which prevents liquid ether from passing into the rectum.

A tube containing ether is set in a vessel containing water at a temperature of 122° F. The ether tube is joined by a glass tube and rubber pipe to a glass globe, and the globe is connected by a rubber pipe to the tip, which is inserted into the rectum. If ether vapor condenses into liquid in the glass globe the globe should be at once emptied. During the administration abdominal distention occurs from unabsorbed ether, and from time to time the administration should be suspended temporarily to permit the gas to escape, otherwise too much will be given and prolonged stupor and postoperative colic may result. It takes much longer to obtain unconsciousness by rectal administration than by inhalation. The method must never be used if the intestines are irritated or inflamed (Dumont, in "Corr-Bl. f. Schweizer Aerzte," Dec. 15, 1908). The method has never come into general use. It irritates the large

intestine, often produces colic, and sometimes is said to lead to protracted stupor ("Anesthetics and Their Administration," by Sir Frederic W. Hewitt). In some cases we can begin the ether by inhalation and shift to the rectal administration when ready to operate. I agree with Baum that the method is more dangerous than the inhalation method. After one of Baum's cases intestinal hemorrhage occurred; in another, gangrene and perforation ("Zentral. für. Chir.," March 13, 1909). Dudley W. Buxton, however, has employed it in many operations about the face, mouth, and larynx, and in some operations for empyema, and commends it. Rectal etherization does not produce a sense of suffocation, the stage of excitement is short, and struggling is trivial or absent. Intratracheal insufflation anesthesia seems to have practically done away with all need for rectal anesthesia.

**Intravenous Etherization (Infusion Anesthesia).**—This method was devised by Burkhardt, of Würzburg, and is still on trial. A $7\frac{1}{2}$ per cent. solution of ether in normal salt solution is employed by Rood ("Lancet," March 23, 1912). He found that a 10 per cent. solution causes hemolysis and a 5 per cent. solution is inadequate. About three-quarters of an hour before operation he gives a hypodermatic injection of $\frac{1}{6}$ gr. of morphin, $\frac{1}{100}$ gr. of scopolamin, and $\frac{1}{100}$ gr. of atropin. When ready to operate, a vein is selected, exposed and opened, and the cannula is introduced. The solution is warm. From $\frac{1}{2}$ to 1 pint of the fluid is run in and anesthesia should be secured in from three to five minutes. Anesthesia is maintained by running in a constant succession of drops. If anesthesia becomes too deep the rate of flow is lessened, and vice versâ. This method of anesthesia keeps the anesthetist out of the way in operations on the head, neck, and mouth. It is claimed that it enables us to measure the dose of anesthetic much more accurately than does the respiratory plan, and that by it we avoid irritation of the mucous membrane of the respiratory tract. The respiration must be watched just as carefully as in respiratory anesthesia. Rood ("Brit. Med. Jour.," Oct. 21, 1911) has had 136 cases and no bad results.

**Hedonal** has been used for infusion anesthesia. It was suggested by Federoff, of St. Petersburg. Mr. Page, of London, has reported 75 cases ("Lancet," March 23, 1912). He uses a .75 per cent. solution in normal salt solution and gives it continuously. Ward ("Lancet," March 23, 1912) has reported a death from it.

**Anesthetic State from Ether or Chloroform.**—The inhalation of an anesthetic produces irritation of the fauces, often some cough, a profuse secretion of mucus, acts of swallowing, dilatation of the pupils, flushed face, and sometimes struggling (especially in children and in drunkards). If at the start the vapor is given in concentrated form, cough will be violent and will cause cyanosis. If the anesthetic is given gradually the cough soon ceases, the respirations become rapid and often convulsive, the pulse becomes frequent, and the patient passes into a condition of active intoxication with preservation of sight and touch, loss of hearing and smell, diminution of pain and sensibility, and often with illusions or hallucinations. In this stage the patient may struggle, and while efforts are being made to hold him, cyanosis may occur. From the stage of excitement just alluded to, many subjects (strong men and drunkards) pass into a stage of rigidity in which the muscles become firmly fixed, the breathing is impeded, the respirations are stertorous, and the face is bluish and congested. Too rapid forcing of the anesthetic tends to cause rigidity, and a skilled anesthetist endeavors to avoid its production, because it is dangerous. The next stage is one of insensibility; the pupils are contracted and react sluggishly to light. If anesthesia is deep the contracted pupils will not react to light; if anesthesia is profound the pupils dilate, but will not react to light. The conjunctival reflex

is gone; the lids are closed; if the arm is lifted and allowed to fall, it drops as a dead weight; the skin is cool and moist and often wet with sweat; the respirations are easy and shallow; the pulse is slow, and there is complete unconsciousness to pain. The loss of conjunctival reflex is the usually accepted sign that the patient is unconscious. In a young child this reflex is soon exhausted by touching the eye, and the sign is unreliable. If a baby is to be anesthetized, the administrator places his finger in the infant's hand. The child grasps the finger, and relaxes its grasp when unconscious.

Always bear in mind that dilated pupils reacting to light and associated with preserved conjunctival reflex mean that anesthesia is not complete; that contracted pupils reacting to light and without conjunctival reflex mean moderate anesthesia; that contracted pupils not reacting to light and without conjunctival reflex mean deep anesthesia; that dilated pupils not reacting to light and associated with lost conjunctival reflex mean dangerously profound anesthesia. Sudden dilatation with fixation is always very ominous. The pupillary phenomena are very valuable when present, but unfortunately they are absent at some stage of the anesthesia in many cases. Inequality of the pupils is not unusual and fixation of one pupil or of both may occur. A preliminary dose of morphin or atropin interferes with the pupillary phenomena. Weak pulse and pallor may be due to nausea, but always require instant attention. Vomiting may be due to forcing strong vapor upon the patient, but may also be due to his partially emerging from a state of insensibility.

Watch the pulse carefully to see if it becomes very weak, irregular, abnormally slow, abnormally fast, or if it suggests a fall of blood-pressure. Syncope may be due to nausea, shock, hemorrhage, or the giving of too much of the drug. Watch the respiration, and do not forget that the chest walls and belly may move when no air is entering the lungs; hence always *listen* to the breathing. *Cyanosis* is a dusky or bluish discoloration of the skin. This condition indicates want of oxygen in the blood. The individual may have been cyanotic or predisposed to cyanosis to start with; cyanosis may be due to pressure; to cough early in the administration; to struggling during the stage of excitement; to gathering of mucus in the respiratory tract; or to rigid fixation of the respiratory muscles. It may also be due to obstruction of the air-passages by some foreign matter, as blood or vomit, lodging in the bronchial tubes, windpipe, larynx, or pharynx; falling back of the tongue (*swallowing of the tongue*); closure of the epiglottis; or to the glottis being pushed against the pharyngeal wall by bending the head forward. Some patients with occluded nostrils may fail to get enough air because of closure of the lips. A patient may, while taking an anesthetic, lie perfectly quiet and appear to "forget to breathe." Ether and chloroform mitigate the causal mental phases of shock, but neither drug keeps nerves from conveying stimuli and each produces a fall of blood-pressure, chloroform directly by its action on the vasomotor center, ether by overstimulation of the vasomotor center (Buxton, in "Proceedings of the Royal Soc. of Med.," April, 1909). Each produces a fall of temperature. Buxton heartily condemns the once common belief "that evidences of shock during a surgical operation are a proof that an insufficient quantity of the anesthetic has been given and that the symptoms of shock can be abrogated by increasing the depth of the narcosis" (Ibid.). Heavy anesthesia by ether or chloroform produces or adds to shock. Shock is manifested by deathly pallor, weak, rapid, and irregular pulse, slow respiration, cold extremities, and a drenching sweat. Edema of the lungs occasionally arises during or after anesthesia.

**Treatment of Complications.**—*Vomiting* due to too much anesthetic is corrected by giving a few breaths of air; vomiting due to incomplete anesthesia is amended by giving more of the vapor. While the patient vomits hold the head

over the edge of the bed, and when vomiting ceases separate the jaws with the gag, and wipe out the vomited matter, mucus, and saliva. *Shock* is treated by diminishing the amount of the anesthetic that is being given, by the hypodermatic injection of atropin (atropin is particularly useful when there is a profuse sweat), by the administration of hot saline fluid by the rectum, by surrounding the patient with hot-water bottles, or by wrapping him in hot blankets, and by lowering the head of the bed. Syncope is sudden cerebral anemia and is usually due to a reflex cause. A tendency to *syncope* requires lowering of the head of the bed, suspension of the anesthetic, and hypodermatic injection of strychnin. In *extreme syncope*, which is most apt to occur from chloroform, do not wait for breathing to cease, but suspend the anesthetic, lower the head of the bed, open the mouth with the gag, catch the tongue, and make rhythmical traction while an assistant is making *slow* artificial respiration. If the patient does not *at once* improve, invert him completely, holding him by the legs and continuing artificial respiration by compressing the sternum (Nélaton). By continuing artificial respiration the blood is urged on through the heart. Give hypodermatic injections of atropin, ether, or even of ammonia. Put mustard over the heart and spine. Employ faradism to the phrenic nerve (one pole to the epigastric region, the other to the right side of the root of the neck). Let fresh air into the room, put hot-water bottles around the legs, apply friction to the extremities, wrap the patient in hot blankets, give an enema of hot salt solution, and hold ammonia to the nose. In some cases of anesthetic poisoning *direct heart massage* has been successfully employed. The method was suggested by Schliff in 1874. Frazier was operating for hydrocele when the patient's respiration ceased and his pulse disappeared. Frazier at once opened the abdomen and, with one hand on the chest wall and the other against the diaphragm, "massaged the heart at the rate of 15 to 20 movements a minute." In about two minutes cardiac contractions were perceptible. In about eight minutes resuscitation was complete ("Jour. Am. Med. Assoc.," May 20, 1911). In Conkling's case there was a chest wound exposing the lung. The heart ceased to beat. The surgeon grasped the heart between the fingers and made compressions. In less than a minute he detected a thrill, and in a few seconds more regular pulsation began ("New York Med. Jour.," Sept. 2, 1905). In Sencert's successful case an operation was being done for gall-stones when collapse occurred, and the surgeon stroked and kneaded the heart through the diaphragm. In a case recorded in the "Brit. Med. Jour.," Nov. 18, 1905, respiration and pulse had ceased three minutes when the abdomen was opened and the heart was kneaded. Recovery ensued. Müller, of Hamburg, advocates exposing and opening the pericardium to perform massage, introducing oxygenated salt solution into a vein, opening the trachea, and performing artificial respiration. Frazier ("Progressive Medicine," March, 1911) publishes statistics of 50 collected cases. These figures indicate that the subdiaphragmatic method is more successful than either the direct method or the transdiaphragmatic method. The table is as follows:

| | Successful. | Partially successful. | Failures. | Total. |
|---|---|---|---|---|
| Direct..... | 2 | 8 | 18 | 28 |
| Transdiaphragmatic.. | 0 | 1 | 2 | 3 |
| Subdiaphragmatic.... | 8 | 5 | 6 | 19 |
| | 10 | 14 | 26 | 50 |

Leonard Hill holds that in the failure which arises *soon after administration of chloroform is begun* the trouble is due to vasomotor paralysis with starvation of the nerve-centers. In such a case he applies abdominal compression and

inverts the patient, making artificial respiration at the same time. In the failure which occurs after *considerable chloroform has been taken* there are paralytic distention of the heart, fulness of the venous system, and loss of the compensations for the hydrostatic effects of gravity. In such a condition empty the distended heart of venous blood by raising the patient into an erect position, and after a moment place him recumbent and make artificial respiration.

*Forgetting to breathe* is met by removing the inhaler and waiting a moment; a breath will usually be taken soon; but if it is not taken, somewhat forcibly knead the structures in the arm-pit. If this fails, open the mouth and pull forward the tongue; this causes a reflex inspiration. Cyanosis is practically not encountered when oxygen is given with ether or chloroform. *Cyanosis*, if slight and due to cough or struggling, is met by removing the inhaler while the patient takes a breath or two of air. If position is responsible for cyanosis, correct it. In empyema, lying upon the sound side may produce it. Obstruction to breathing may be due to bending down the head. If due to stenosis of the nares in a person without teeth, hold the lips apart with a finger. If due to collection of mucus, wipe the mucus out of the mouth by means of bits of gauze firmly clamped in forceps; raise the shoulders, extend the head and place it on its side in order that mucus may run out of the angle of the mouth; and give a dose of atropin hypodermatically. If the amount of mucus is large and the secretion is persistent, it may be necessary, especially in children, to empty the respiratory passages by inverting the patient. In cases of excessive bronchial secretion we fear the development of pulmonary edema or postoperative bronchopneumonia.

Dudley W. Buxton points out that duskiness will often pass away if ether is removed, one or two inhalations of chloroform given, and ether then continued. If in any case cyanosis is severe or grows worse, suspend the drug, dash cold water in the face, force open the jaws, pull forward the tongue, make artificial respiration until a breath is taken, and then give oxygen for a time. If these means fail, stretch the sphincter ani and bleed from the external jugular vein. If a breath is not now taken, do tracheotomy. In respiratory or heart failure forced artificial respiration by Fell's method is of great value (see page 896). The pulmotor, if at hand, enables the surgeon to maintain regular artificial respiration for an indefinite time (see page 867). *Swallowing the tongue* is corrected by pulling the tongue forward. If it tends to recur, lay the head upon its side or keep the tongue anchored with forceps. *Closure of the epiglottis* is corrected by pulling the patient's head over the edge of the table and pushing strongly back upon his forehead. This maneuver lifts the hyoid bone, and with it the epiglottis. The epiglottis can be lifted by passing a spoon-handle or the index-finger over the dorsum to the base of the tongue and pressing forward. If, in obstruction to respiration, the above means fail, make artificial respiration at once (see page 864); if obstruction continues, perform tracheotomy.

*Edema of the lungs* is treated by instant venesection, the inhalation of nitrite of amyl, and the administration of stimulants and nitroglycerin hypodermatically. Sometimes during the anesthetic state the muscles of the belly become very *rigid*, a condition which greatly interferes with an abdominal operation. It may arise during cyanosis, and if so caused is amended, as cyanosis abates under proper treatment. In some cases it is due to the fact that sufficient anesthetic has not been given. If the air-passages are obstructed before operation, abdominal rigidity is apt to arise. In some cases it seems impossible to overcome it with ether. In such a case, if the anesthetist is a trusted man, anesthetize the patient with gas and ether and then give chloroform (Blumfield, in "Lancet," May 31, 1902).

**The Reaction from Anesthesia.**—When ether or chloroform is given, a considerable quantity is swallowed and either drug irritates the stomach and creates nausea and often vomiting. The longer the operation, the more of the anesthetic enters the stomach, and the greater the liability to subsequent vomiting. At the termination of a prolonged operation upon an adult, if the patient's condition admits of it, and if the nature of the operation does not forbid it, I like to have a stomach-tube passed and the stomach well washed out with warm water. The washings smell strongly of the anesthetic, and the procedure greatly lessens the severity and frequency of postoperative vomiting (Geo. S. Brown, in "Surg., Gynec., and Obstet., August, 1905). After the administration of the anesthetic has been suspended and the operation has been completed the temperature is usually subnormal. The patient must be watched until consciousness returns. If he is left alone, a change of posture may lead to arrest of feeble respiration, the assumption of the erect position may cause fatal syncope, and mucus or vomited matter may block the air-passages and cause suffocation. The best position to place him in is the recumbent, the head being level with the body or somewhat lower and the side of the face resting on the pillow. Shock is treated by ordinary methods (see page 262). The inhalation of oxygen is of great value in rousing a patient from the state of anesthesia, and will often prevent vomiting. If vomiting occurs, the head should be upon its side or should be held over the edge of the bed, and after the spell of vomiting the mouth must be wiped clean. The face should be washed with cold water and be fanned rather actively. It is the routine practice of some surgeons to administer vinegar by inhalation during the reaction from an anesthetic. This proceeding sometimes seems to prevent vomiting. Some patients awake from anesthesia as from a quiet sleep; others are noisy, turbulent, and violent. The duration of the period of reaction varies with the anesthetic used, the amount given, and the personal tendencies of the patient. The patient must not be allowed to sit up for several hours at least. No food is to be allowed for at least six hours. Unless the operation was upon the stomach, I do not forbid water, but allow the patient to drink freely of hot water. This dilutes any irritant material in the stomach and dissolves mucus, and if vomiting does occur it serves to wash the stomach out. All fat patients, all patients with respiratory difficulties, or in whom we apprehend respiratory complications should, if there is no contra-indication, be placed in a sitting or, at least, a semi-erect posture as soon as reaction from anesthesia is obtained. If this plan is followed, ether-pneumonia and other respiratory troubles will very seldom develop.

**After-effects of Anesthetics.—Vomiting.**—I am convinced that in many cases postanesthetic vomiting is due or is largely due to irritation of the stomach caused by swallowing considerable quantities of an irritant anesthetic. The liability to it is greatly lessened by washing out the stomach before the patient leaves the operating table, and allowing the patient (in suitable cases) to drink freely of hot water as soon as he returns to consciousness. Most patients vomit more or less, but if the man has been drinking hot water an act of vomiting washes out his stomach and he may not vomit again. Violent vomiting may occur in spite of all our efforts, and may persist for hours, greatly exhausting the patient and doing infinite harm, it may be, especially if the operation were upon the brain or an intra-abdominal structure. If vomiting continues, forbid food absolutely. Very hot water in doses of a teaspoonful should be given at frequent intervals. Drafts of hot water may relieve the condition by washing out the mucus from the stomach. Other remedies which may succeed are: inhalations of vinegar, hot black coffee by the mouth, a mustard plaster over the stomach, fresh air in the room, small pieces of ice placed

in the mouth and sucked, small doses of iced champagne, and drop doses of a 3 per cent. solution of cocain or 3-drop doses of a 5 per cent. solution of eucain. The best remedy for persistent vomiting is lavage of the stomach. Some persons, as Dudley W. Buxton points out, suffer greatly from nausea, although there is little or no vomiting. In such cases Buxton uses 1 min. of tincture of nux vomica in a teaspoonful of hot water every ten minutes until six doses have been taken. If this plan fails, he gives drop doses of wine of ipecac or minim doses of dilute hydrocyanic acid.[1]

*Vomiting from chloroform* is usually more difficult to check than vomiting from ether. In any case of persistent vomiting examine for acidosis (see page 1207).

**Backache.**—This is a very common and often a very distressing consequence of anesthesia. It is complained of soon after consciousness is regained, it may persist for several days, and it is a not uncommon cause of wakefulness. It is usually greatly aggravated by turning and twisting, and by attempting to rise up from the bed. The pain is located in the lumbar and sacral regions and is often accompanied by rigidity of the lumbar muscles. Various explanations have been given of it. One view is that it is due to renal congestion. Another, that it results from congestion of the spinal cord. I believe that the explanation of most cases is that given by John Dunlop ("New York Med. Jour.," July 10, 1909), viz.: "The patient during the operation lay upon a flat table without support to the lumbar curve, consequently the sacro-iliac synchondroses were strained. The backache may be largely prevented by placing a small pillow so that it will support the lumbar curve during anesthesia."

**Respiratory disorders** are more often noted after ether than after chloroform. Bronchitis may follow or bronchopneumonia (*ether-pneumonia*). Respiratory difficulties may be due to chilling the patient by bringing him from a warm operating room through a cold hall and into a cool bedroom. Bronchopneumonia is especially common in septic patients, and may be due in some cases to septic emboli and in others to aspiration of septic material into the bronchi (cases of cancer of the tongue and pharynx, and cases with stercoraceous vomiting). They are treated by ordinary methods. If chloroform is given when a gas-light is in the room the vapor is decomposed and certain highly irritant products are formed, which, when inhaled, produce laryngeal spasm and possibly bronchitis. The irritant material is probably $COCl_2$. The treatment is to admit fresh air freely into the room, and to have the patient inhale vinegar and, later, oxygen. Ether-pneumonia must not be confounded with postoperative pneumonia, described by Wm. H. Bennett.[2] This latter condition may arise from seven to fourteen days after operation in robust, gouty people, and is usually unilateral. Some cases of respiratory disorder result from chilling while in the operating room, or while coming from it, rather than from the anesthetic. If the patient is placed in a sitting position or, at least, semi-erect in bed, as soon as he reacts from the anesthetic the danger of serious respiratory disorder will be at a minimum.

**Renal Complications.**—After the administration of an anesthetic, blood, albumin, sugar, acetone, or diacetic acid may appear in the urine, and the secretion may become scanty or even be suppressed. It is usually maintained that chloroform is less apt to irritate the kidney epithelium than ether, but there has been much dispute on this point. If casts and albumin are present before anesthetization, the condition may be rendered worse if ether or chloroform is given. If neither casts nor albumin are present, they will not be so apt to appear after taking chloroform as after taking ether, but if they do appear after chloroform, they remain longer than after ether (Legrain). The truth of

[1] "Anesthetics," by Dudley W. Buxton.
[2] "Practitioner," December, 1896.

the matter probably is, that if the kidneys are healthy a small or moderate amount of either drug is not particularly irritant; but if the kidneys are diseased, a small amount, and even if they are healthy, a large amount, of either drug produces decided renal irritation. Chloroform is less irritant because less chloroform than ether is given to secure and maintain anesthesia. Scantiness or suppression of urine may be due to operative shock rather than to ether or chloroform. If the urine becomes somewhat scanty or if albumin appears in it, give non-irritant diuretics, diaphoretics and cathartics, and employ proctoclysis. The treatment of acidosis is set forth below. If the urine becomes very scanty, use hypodermoclysis. If postoperative suppression arises, it is the usual custom to give intravenous infusion of hot saline fluid, but I am doubtful of its value. Exposure of each kidney in the loin and incision of its capsule to relieve tension is justifiable and may do good.

**Acid Intoxication.**—This condition has been called *delayed poisoning*, *acetonuria*, and *acidosis*. Diabetic coma is due to acid intoxication. It is known that even in healthy urine there may be a trace, but a bare trace, of acetone. A diabetic individual deprived absolutely of carbohydrates is apt to get acetone and diacetic acid in the urine. In such cases carbohydrates cause the prompt disappearance of the acetone and diacetic acid. In a case which shows acetonuria before operation Bonn gives glucose as a prophylactic ("Brit. Med. Jour.," Feb. 25, 1911). Even people who do not suffer from diabetes may develop acetonuria after anesthesia. In certain cases in which dangerous symptoms arise after anesthesia the urine shows increased acidity, may contain albumin or casts, and contains acetone bodies (particularly β-oxybutyric acid), diacetic acid, or both of these substances. The blood contains an excess of free fat and shows diminution of alkalinity. Acid intoxication is very much commoner after the administration of chloroform than of ether, but may follow the giving of any general anesthetic. It may occur in individuals whose tissues contain areas of fatty degeneration, but it also occurs in those entirely free from degeneration; in fact, children particularly suffer in this way after the use of chloroform. The actual operation has nothing to do with the trouble, and sepsis is not causative. The drug used as an anesthetic causes acute fatty degeneration of the liver and other organs, quantities of toxins are formed, and these toxins cause the symptoms. Diacetic acid and β-oxybutyric acid are by-products of the process and are antecedents or precursors of acetone. The symptoms arise after the patient has emerged from anesthesia and reacted from shock. There is persistent vomiting of thin and foul fluid, the patient is extremely restless and much excited, there may be delirium, but dulness and heaviness may take the place of restlessness and excitement and coma may arise (J. A. Kelly, in "Annals of Surg.," Feb., 1905). Usually the temperature is subnormal, but sometimes there is elevated temperature. In many cases jaundice arises. There is an odor of acetone on the breath. Latent cases free from symptoms usually get well. Slight cases and even some grave cases recover, but most of the grave cases die in from one to five days. A knowledge of this condition explains some otherwise inexplicable deaths, and also some cases of persistent postoperative vomiting and of retarded convalescence. In acid intoxication there is fatty degeneration of the kidneys, of the liver, of the suprarenal glands, and of the gastric mucosa. The occurrence of such a condition is an impressive admonition that a surgeon should operate quickly, that as little of the anesthetic should be given as possible, that the urine should be carefully examined each day after operation for certainly several days, and that chloroform should not be used for prolonged administration. The indication for treatment is to saturate the patient with alkalis. Sodium carbonate is usually selected. It may be given by the rectum, by the mouth, by hypodermoclysis, or intravenously. Rectal administration produces irritation and diarrhea. In acetonuria without clinical

symptoms alkalinize the urine by 4 or 5 dr. of sodium bicarbonate daily, given by the mouth. When the urine becomes alkaline it is to be kept so. When symptoms appear give from 10 to 20 teaspoonfuls a day by the mouth. If symptoms do not soon disappear give the drug intravenously. Severe acid intoxication is treated as follows: Encourage skin activity by wrapping the patient in blankets and surrounding him with hot-water bags. Give carbonate or bicarbonate of sodium intravenously. A solution of bicarbonate of a strength of from 3 to 5 per cent. is used. Labbé gives from 1 to 2 liters. If the patient improves, the sodium bicarbonate is again given by mouth. Some surgeons give citrate of sodium. Drennan gives a salt of calcium. Bevan and Farill ("Jour. Am. Med. Assoc.," Sept. 20, 1905) reported 1 case and collected 27 from literature. In this series there were 2 recoveries. (On this subject see Lewis Beesly, in "Brit. Med. Jour.," May 19, 1906; J. A. Kelly, in "Annals of Surgery," Feb., 1905; A. D. Bevan and H. B. Farill, in "Jour. Am. Med. Assoc.," Sept. 20, 1905; Geo. E. Brewer, in "Transactions Am. Surg. Assoc.," vol. xx, 1902; Labbé, in "Arch. gen. de med.," Dec., 1911, and in "Presse méd.," Feb. 5, 1910; Braun, in "Brit. Med. Jour.," Feb. 25, 1911.)

**Postanesthetic Paralysis or Narcosis Palsy.**—Paralysis may arise during anesthesia as a result of cerebral hemorrhage or embolism.

It sometimes happens that when a person has come out of the anesthetic sleep a palsy of some part is found to exist, the condition being peripheral and not central in origin. Peripheral narcosis palsies are pressure palsies, although it is held by some that the anesthetic has a toxic influence which distinctly lowers the capacity of the nerves to sustain pressure. Certain it is that palsy sometimes follows what seems a degree of pressure inadequate to cause such a result. Narcosis palsies may be due to pressure of an extremity upon a table edge or to pressure upon nerves by placing the patient in certain positions.[1] When the Trendelenburg position has been employed, the flexures of the knees are in contact with the edge of the table, and paralysis of one or both external popliteal nerves may be induced. When the patient lies upon the side any nerve of the arm or forearm may suffer, but the circumflex and radial are most liable to be damaged. When the arm is elevated to the side of the head, or when it is drawn out strongly from the body, the brachial plexus may be compressed by the head of the humerus (Braun, in "Deutsche Med. Woch.," 1894). When the arm is in external rotation and is drawn backward and outward the median nerve is stretched, and when the forearm is flexed and supinated the ulnar nerve is stretched (Braun, Ibid.). In most cases the paralysis involves muscles supplied by the brachial plexus and is due to drawing the arm upward and backward over the head, a position which may squeeze the cords of the plexus between the collar-bone and the first rib. Garrigues shows that the plexus is particularly apt to be squeezed when it is stretched by the head being drawn to the opposite side or being allowed to fall back.[2] According to Büdinger the mounting up of the clavicle squeezes the plexus as its cords cross the first rib. This surgeon thinks that extreme abduction of the arm may squeeze the cords.

Postanesthetic peripheral paralysis is most common in the arm, but may occur in the leg or face. The prognosis is good, as a rule. Slight cases are soon recovered from; more serious cases, in which degeneration occurs, may not be recovered from for months. The treatment is that of any pressure palsy.

**Primary Anesthesia.**—Instruct the patient to count aloud and hold one arm above his head. Give the ether rapidly. In a short time he becomes mixed in his count and his arm sways or drops to the side. There is now a period of insensibility to pain lasting only about half a minute, and

[1] H. J. Garrigues, in "Am. Jour. Med. Sciences," Jan., 1897.
[2] Ibid.

during this period a minor operation can be performed. The patient quickly reacts from primary anesthesia without vomiting (Packard).

**Mixtures** are used by some because of the belief that a mixture might eliminate some unpleasant feature or some danger from a particular anesthetic.

**Mixture of Ether and Chloroform.**—This may be used in varying proportions. Hewitt at times employs 2 parts of chloroform to 3 parts of ether.

**Vienna mixture** contains 1 part of chloroform and 3 parts of ether.

**Mixture of Alcohol and Chloroform.**—All the chloroform mixtures produce the effects of chloroform, but we are giving the drug in an unknown amount. It was believed by Sansom, who devised this mixture, that the alcohol prevents concentration of chloroform vapor by retarding evaporation. When used, 1 part of alcohol is added to 4 parts of chloroform.

**Nitrous Oxid and Oxygen.**—(See page 1211.)

**A. C. E. Mixture.**—This mixture was originally used by Harley in 1864. It is often valuable in cases in which ether cannot be given. It is composed of 1 part of alcohol, 2 parts of chloroform, and 3 parts of ether. Its action is supposed to be between that of chloroform and ether. The objection to the A. C. E. mixture, as to any mixture, is that the materials do not evaporate in the ratio in which they are mixed, hence an uncertain amount of chloroform vapor is being inhaled (Buxton). This mixture is given by some from a Junker and by others from an open inhaler. Plenty of air should be given with it. The anesthetic acts similarly to chloroform.

**Billroth's mixture** contains 1 part of alcohol, 3 parts of chloroform, and 1 part of ether.

**Schleich's Mixture for General Anesthesia.**—Schleich, in 1895, introduced *a new anesthetic agent* which he claims is safer than chloroform. This surgeon maintains that a material is safe as an anesthetic only when almost all of the amount taken in at an inspiration is expelled on expiration. The anesthetic is unsafe in direct proportion to the amount absorbed; and the lower the boiling-point of an anesthetic, the less is absorbed, hence an anesthetic agent, to be safe, should have a low boiling-point. Schleich makes three solutions. The first contains (by volume) $1\frac{1}{2}$ oz. of chloroform, $\frac{1}{2}$ oz. of petroleum ether, and 6 oz. of sulphuric ether. The second contains $1\frac{1}{2}$ oz. of chloroform, $\frac{1}{2}$ oz. of petroleum ether, and 5 oz. of sulphuric ether. The third contains 1 oz. of chloroform, $\frac{1}{2}$ oz. of petroleum ether, and $2\frac{2}{3}$ oz. of sulphuric ether. No. 1 is used for light anesthesia, No 2 for medium anesthesia, and No. 3 for deep anesthesia. The anesthetic can be given from an open inhaler or a towel. The anesthetic state is quiet, reaction is rapid, and vomiting occurs in but half the cases. The superiority of this new anesthetic has not been proved. It sometimes causes dangerous symptoms, and has produced death. Some surgeons, who formerly approved of it, have abandoned it. It will certainly not displace ether or chloroform. Schleich's mixtures are now seldom or never used in the United States. Petroleum ether has no anesthetic power, and Meltzer shows that it is dangerous and tends to paralyze the respiratory muscles. Willy Meyer ("Med. Record," August 15, 1908) believes in the Schleich principle, but substitutes ethyl chlorid with a boiling-point of 59° F. for the petroleum ether. He uses 17 per cent. volume of ethyl chlorid with 83 per cent. volume of the molecular mixture of ether and chloroform.

**Ethyl bromid** was first used by Nunneley, of Leeds, in 1849. It was reintroduced in 1882 and again in 1896. It is still sometimes used for short operations. It is given while the patient is recumbent. The unconsciousness is obtained in from one to three minutes and is rapidly recovered from, and there is no after-sickness. The unconsciousness lasts about three minutes. Three dr. are given to a child, and 6 dr. to an adult. A towel is put over the face and the entire amount is poured on at once, and as soon as the patient is uncon-

scious the towel is taken away and no more of the drug is given (Cumston). Even if consciousness is regained too quickly to suit the purposes of the surgeon, it is not safe to give more of the drug, a notable objection which chlorid of ethyl does not possess. Cases have been reported in which sudden death has followed the administration of this drug, and it should not be given if there is disease of the heart, lungs, or kidneys.[1] Twenty-four deaths from bromid of ethyl are on record (Guadiana). If it kills, it acts like chloroform. It may be given *before* ether to prevent unpleasant effects, but it is usually not considered proper to give before chloroform. Zematski, however, has used it before chloroform in 2000 cases ("Vratch," August 25, 1901). I know of 2 unpublished deaths from it in Philadelphia. I never use it and regard it as unsafe. The drug rapidly deteriorates, and the deteriorated drug is very dangerous.

**Chlorid of ethyl** is a rapid anesthetic and statistics imply that it is a safe one. My faith in it has been greatly shaken by knowledge of 3 unpublished deaths in Philadelphia. It was first used by Heyfelder in 1848. A committee of the British Medical Association condemned it in 1880. Carlson and Thiesing reintroduced it in 1895 (McCardie, in "Lancet," April 4, 1903). It may be sprayed upon a mask covered by six to eight layers of gauze, so that the drug will not evaporate too quickly in the air. Many anesthetists give it in a closed apparatus, the patient respiring into and from a rubber bag. I believe it should be mixed with air and that concentrated vapor is a danger to the heart. The odor of the drug is agreeable. From 5 to 10 gm. of ethyl chlorid are given for a short operation if the mask is used. The patient must always be recumbent when taking it. Early in the inspiration the pulse and respiration become rapid. When unconsciousness comes they should be normal. The anesthetic state is induced, when the mask is used, in from two to three minutes, and as soon as it is obtained the patient is allowed to get air. If the closed inhaler is used unconsciousness is obtained more rapidly. Excitement does not precede unconsciousness. The anesthetic condition lasts from one to three minutes, and it is recovered from rapidly, usually without vomiting or unpleasant after-effects. If the patient recovers too rapidly for the surgeon's purpose, more ethyl chlorid can be given. It is to be noted that complete muscular relaxation does not occur, in many cases the conjunctival reflex is not completely abolished, and often the pupils do not dilate. It has no superiority over nitrous oxid, except as to cost and portability, and sometimes it fails to produce complete unconsciousness. A large dose rapidly given is dangerous, as it may cause cessation of respiration and spasm of the diaphragm. A contraindication to its use is any respiratory obstruction. In many cases there is spasm of the masseters. Concentrated vapor administered for a considerable time lowers the blood-pressure, induces cyanosis and asphyxia, and would eventually cause death by respiratory failure (McCardie, Ibid.). Lotheisser, in a study of 2500 cases of anesthesia by this agent, reports 1 death. Ware collected 12,436 cases, with 1 death ("Jour. Am. Med. Assoc.," Nov. 8, 1902). Seitz, of Konstanz, collected 16,000 cases, with 1 death. Miller ("Jour. Am. Med. Assoc.," Nov. 23, 1912) estimates that there is 1 death in 13,365 administrations. It is perhaps safer than chloroform, not nearly so safe as nitrous oxid, and not so safe as ether. The drug is used for a brief operation or examination. It can be given to infants a few days old with reasonable safety, and it has been administered many times to the aged. When it kills, it acts in a similar manner to chloroform. I have often given it *before* ether to prevent unpleasant symptoms and to hasten the advent of anesthesia, but it must *never* be given before chloroform. Recently I have practically ceased to administer ethyl chlorid.

[1] See Cumston, in "Boston Med. and Surg. Jour.," Dec. 20, 1894.

**Nitrous oxid gas** may be used to obtain anesthesia for brief operations. It is contra-indicated when high blood-pressure indicates vascular degeneration, because apoplexy may follow its administration. It should never be given when the air-channel is narrowed, as in Ludwig's angina, abscess in or below the tongue, and thyroid enlargement (F. W. Hewitt, in "Lancet," July 20, 27, and August 10, 1907). This gas is stored in steel cylinders, in which it is liquefied. The gas is passed into a rubber bag (Fig. 795), and is given to the patient by means of a tube and a mouth-mask, a wedge being placed between the patient's molar teeth, and the nostrils being closed by the anesthetist's fingers. The wedge must be held by a string, so that it cannot be swallowed. The patient becomes unconscious in about one minute, and we know the patient is anesthetized by the stertor and cyanosis and the insensitiveness of the conjunctivæ. The pulse should be watched, and if it flags the administration must be suspended at once. The striking phenomena are asphyxial, stertorous respiration, cyanosis, and even convulsions, dilatation of the pupils, rapidity of the heart, and swelling of the tongue.[1] Muscular relaxation is not as complete as in ether-anesthesia. Slowing of the heart is a danger sign. If nitrous oxid causes death, it does so by asphyxia or by asphyxia and cardiac inhibition. A person rouses very rapidly when the administration of nitrous oxid is suspended. It is a useful plan to give nitrous oxid first and follow this with ether (see page 1214). By this method the patient is anesthetized rapidly and pleasantly with the nitrous oxid, and the anesthesia is maintained by the ether.

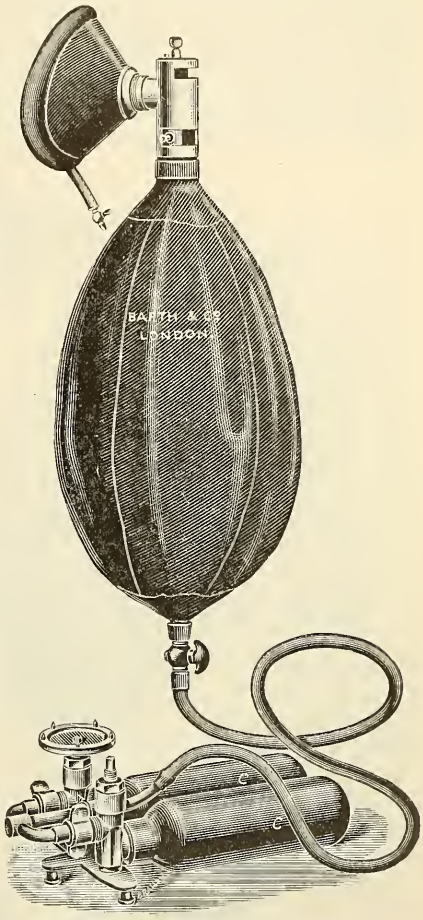



Fig. 795.—Hewitt's nitrous oxid apparatus.

It was formerly taught that nitrous oxid necessarily produces cyanosis, because the gas can only cause anesthesia by partially asphyxiating the patient. We know this is untrue, because if *nitrous oxid is mixed with oxygen* or atmospheric air anesthesia is obtained without cyanosis. Nitrous oxid is a genuine anesthetic agent. If a prolonged administration of nitrous oxid is desired, pure nitrous oxid can be given, a breath of fresh air being allowed, from time to time. By this method Preston has anesthetized many patients, the duration of the anesthesia being from ten to fifty minutes. A better plan is to give nitrous oxid and oxygen. I am satisfied that this combination does not occupy the place in surgery its merits entitle it to. One reason is the absolute necessity of having a specially skilled administrator. A trouble frequently encountered is persistent rigidity. This can often be prevented by a preliminary dose of morphin.

[1] See Hewitt, "Brit. Med. Jour.," Feb. 18, 1899.

Postanesthetic vomiting is rare (Teter, on "Thirteen Thousand Administrations of Nitrous Oxid with Oxygen," "Jour. Am. Med. Assoc.," August 7, 1909). Hewitt[1] formulates the following views as to the use of oxygen and nitrous oxid:

"In order to obtain the best form of anesthesia oxygen should be administered with nitrous oxid by means of a regulating apparatus (Fig. 796), the percentage of the former gas being progressively increased from 2 to 3 per cent. at the commencement of the administration to 7, 8, 9, or 10 per cent., according to the circumstances of the case. The longer the administration lasts, the greater may be the percentage of oxygen admitted.

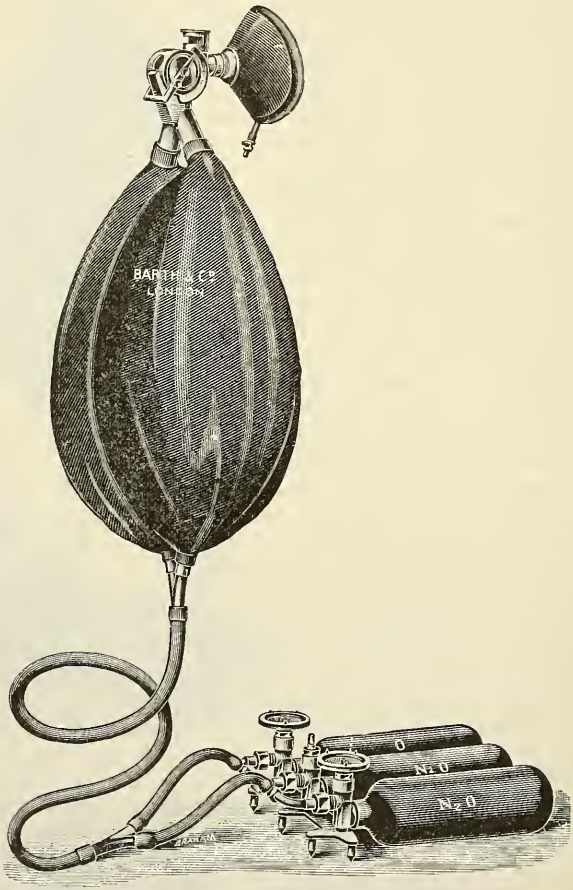



Fig. 796.—Hewitt's nitrous oxid and oxygen apparatus.

"The next best results to those obtainable by means of a regulating apparatus for nitrous oxid and oxygen are to be secured by administering certain constant mixtures of these two gases. Mixtures containing 5, 6, or 7 per cent. of oxygen are best for adult males, and mixtures containing 7, 8, or 9 per cent. are best for females and children. The next best results to those last mentioned are to be obtained by means of mixtures of nitrous oxid and air, from 14 to 18 per cent. of the latter being advisable in anesthetizing men and from 18 to 22 per cent. in anesthetizing women and children."

[1] "Brit. Med. Jour.," Feb. 18, 1899.

Crile uses nitrous oxid and oxygen as the anesthetic of choice for many operations, even major operations. He believes it produces less shock, less nausea, and less lowering of vital resistance to infection than does ether. He insists on the necessity of having a highly trained administrator and of keeping the patient *pink* during the administration. Respiratory failure is met at once by turning off the nitrous oxid and substituting pure oxygen. If relaxation is impossible (and it may be in a very muscular subject), ether is given until relaxation is attained and then a return is made to nitrous oxid ("Transactions of Southern Surg. and Gynec. Assoc.," 1909). Crile gives a small dose of morphia and scopolamin before operation to prevent too early postoperative appreciation of the "operative trauma."

The Gwathmey-Woolsey apparatus is a very useful one for nitrous oxid and oxygen administration (Fig. 797).

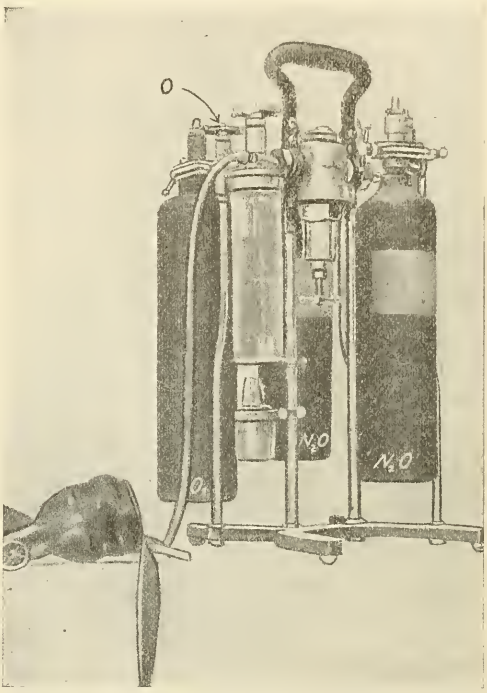



Fig. 797.—Gwathmey-Woolsey nitrous oxid oxygen apparatus with tanks in place: *O*, Regulating valve for oxygen; $O_2$, oxygen tank; $N_2O$, nitrous oxid tank.

**Bichlorid of Methylene.**—The composition of the so-called bichlorid of methylene is a matter of dispute. Some high authorities believe it to be a mixture of methyl alcohol and chloroform. It rapidly produces unconsciousness, and the patient returns quickly to consciousness when the administration is suspended. Some surgeons have thought highly of it, and claimed that it is pleasant, safe, and is not followed by vomiting as often as chloroform. The weight of opinion is that it is dangerous, death being similar to death from chloroform. It is given by means of a Junker apparatus.

**Anesthetic Successions.—Bromid of Ethyl Followed by Chloroform or Ether.**—(See page 1210.)

**Chlorid of Ethyl Followed by Ether.**—(See page 1210.)

**Chloroform Followed by Ether.**—Chloroform is sometimes given until the sensation becomes more or less obtunded, when ether is substituted. This is done to save the patient from the unpleasant sensations of etherization. It is a practice not to be commended, because it is precisely in the beginning that chloroformization is most dangerous.

**Ether Followed by Chloroform.**—When the patient cannot be relaxed or rendered unconscious by ether, or when some other complication develops, it is common practice to suspend ether and substitute chloroform. If the change is made, chloroform should be given cautiously. A large quantity should never be poured upon the inhaler at one time. The change should never be made when the patient is struggling, because the deep respirations which attend or follow struggling may lead to the rapid inhalation of a dangerous dose of chloroform vapor. Further, as Hewitt points out, when the patient is deeply under the influence of ether the change should not be made unless it is imperatively necessary.

**Nitrous Oxid Gas Followed by Ether (Gas and Ether).**—This very valuable method was suggested by Clover. I have used it repeatedly with great satisfaction. The patient is *made* unconscious by nitrous oxid and is *kept* unconscious by ether. Thus are avoided excitement, struggling, choking, and the very unpleasant sensations induced by ether. It lessens the amount of time requisite to obtain anesthesia and lessens the amount of ether used. More important even than this, the method is safe. It is more satisfactory in women and children than in men. In very muscular men and in very stout elderly men it should not be used. In many cases nitrous oxid causes a flow of mucus from the respiratory tract. Because of the frequency of this happening it is wise to precede gas and ether anesthesia twenty minutes by a hypodermatic injection of morphin and atropin (Van Kaathoven, in "Annals of Surgery," Sept., 1908), or of atropin alone. Many operators first anesthetize with nitrous oxid, using an ordinary dental apparatus, and then give ether on an ordinary inhaler. The anesthetist must bear in mind that ether must be given gradually, not suddenly, poured on in large amount. Others prefer to use a combined gas-and-ether inhaler. I use the Gwathmey-Woolsey apparatus (Fig. 797).

Hewitt ("Anesthetics and Their Administration") thus describes the administration by means of Clover's portable ether-inhaler fitted with a stop-cock and a detachable gas bag:

"If the patient be lying upon his back, his head should be turned to one side. The face-piece with the charged ether chamber is then applied during an expiration. Air will be breathed backward and forward. When the respiration is seen to be proceeding freely, and the face-piece fits well, the charged gas bag is attached to the ether chamber. Air will still be breathed, but not through the valves of the special stop-cock. When the valves are heard to be working properly 'gas' is turned on, and is likewise breathed through the valves. Three or four respirations (or about one-half of the contents of the bag) are allowed to escape. The valve action is now stopped by turning the tap at the upper part of the stop-cock. At the same moment at which the patient begins to breathe 'gas' backward and forward, the rotation of the ether chambers for the addition of ether vapor should be commenced. The administrator will, in fact, find that he can, in a few seconds from the commencement of the administration, rotate the ether chamber as far as '1' or '1½.' Should swallowing or coughing arise, he must rotate more slowly. Respiration soon becomes deep and regular, and more and more ether may be admitted. At about this juncture, if the apparatus has been fitting the face well, signs of nitrous oxid narcosis may appear, especially in those who are quickly affected by this gas. Should jerky breathing or 'jactitation' arise, one full inspiration of air may be admitted at the air-tap. It should be remembered, however, that in giving 'gas and ether' by this method, the object is to just steer clear of the clonus and 'stertor' of nitrous oxid narcosis and to gradually but increasingly mix ether with the gas.

"In muscular and vigorous subjects the quantity of gas above mentioned will be found to be, as a general rule, insufficient to lead to the usual signs of deep nitrous oxid anesthesia. The rotation of the ether chamber should be continued till the indicator points to '2', '3,' or 'F.'

"The mistake that is most commonly made is that of admitting air too soon. Should air be given during the first half or three-quarters of a minute, the patient will partially come round, hold his breath, set his teeth, and give a good deal of trouble. Duskiness of the features must be expected. Speaking generally, air should not be allowed until the patient is stertorous, when one breath may be given. In this manner the patient will continue breathing a mixture of nitrous oxid, ether, and air till the usual signs of deep ether-anesthesia appear,

when the gas bag may be detached, and the little bag ordinarily used with Clover's inhaler substituted."

Hewitt prefers to use a modified Clover inhaler, which permits of the introduction of ether after the inhalation of nitrous oxid has begun.

**Hypnotic Anesthesia.**—It is well known that Esdaile in India did numbers of operations upon patients in hypnotic anesthesia. Cloquet, as long ago as 1829, amputated a breast of a woman who was held free from pain by hypnosis. In 1851 Guérineau amputated the thigh of a hypnotized person and there was no sign of pain. But all subjects are not susceptible. Even susceptible subjects require to be hypnotized again and again for days before the operation. The method has its own dangers and has been entirely abandoned.

**Scopolamin-morphin Anesthesia.**—This method has been enthusiastically praised and I have used it with satisfaction in a number of cases, but I have grown afraid of it. In a patient in the Jefferson Hospital dangerous symptoms arose after a dose of $\frac{1}{100}$ gr. of scopolamin. Ely records a death from respiratory failure two hours after the administration of $\frac{1}{8}$ gr. of morphin and $\frac{1}{100}$ gr. of scopolamin ("New York Med. Jour.," Oct. 20, 1906). A number of deaths have been reported following its use and there are, beyond doubt, unreported cases. Four deaths in 2400 cases were certainly directly due to it (H. J. Whitacre, in "New York Med. Jour.," March 31, 1906). It has even been stated that the death-rate is 1 in 100 ("Semaine Medicale," Jan. 11, 1905). Scopolamin is chemically identical with hyoscin and must never be used unless fresh, as it decomposes in air and light. If given without morphin, it is inefficient. Large doses are certainly dangerous, and the combination should never be given in sufficient amount to induce anesthesia unaided. If used at all, it should only be as an aid to local anesthesia or to general anesthesia by ether or chloroform. I have used it as an aid to local anesthesia in 6 goiter operations and in 2 cases of removal of the Gasserian ganglion. It should not be used in heart disease (Hayem); in persons under sixteen or over sixty (Korff); in any one with a tendency to pulmonary edema or with any acute condition of the throat which interferes with respiration (A. C. Wood, in "American Medicine," Nov. 11, 1905).

It produces a drowsy, heavy state or actual sleep, and the patient can be kept unconscious with an extremely small quantity of ether or chloroform. For five or six hours after the operation the sleep continues, and in most cases there is no postoperative vomiting.

If it is used, a mixture is freshly made containing $\frac{1}{100}$ gr. of scopolamin and $\frac{1}{6}$ gr. of morphin, and this is given hypodermatically one-half an hour before the operation. During the operation the sleep may be maintained by small amounts of ether or chloroform. If symptoms of poisoning occur, artificial respiration and oxygen inhalations may be required, external heat is needed, and nitroglycerin, strychnin, or caffein should be given.

I agree with Kochmann that we are not as yet justified in recommending this method of anesthesia ("Münchener medizinische Wochenschrift," 1905, No. 17).

**Local Anesthesia.**—In every case requiring operation we should inquire whether local anesthesia should be used instead of general anesthesia. Many really extensive operations can be done under it, and its field has been greatly broadened by the knowledge that viscera innervated by purely visceral nerves are insensitive and sensation exists only in those which receive branches from the somatic nerves (K. G. Lennander, in "Mittheilungen aus dem Grenzgebeiten der Medicin und Chirurgie," 1902, Bd. x, Heft 1 and 2). Lennander shows that the parietal peritoneum is sensitive to pain, but not to touch—that the intestine, stomach, edge of the liver, mesentery, gall-bladder, urinary bladder, kidney parenchyma, lung, anterior wall of the trachea, tes-

ticle, and epididymis are insensitive, though the coverings of the testicle and epididymis are sensitive. My experience is that the viscera may be cut, sutured, and handled without any severe pain if they are not pulled upon. In removing an appendix the only pain felt will be when the meso-appendix is pulled upon or adhesions to the parietal peritoneum are separated. The advantages of operation under local anesthesia are freedom from the danger of anesthetic accidents, blood changes, and postanesthetic discomforts and dangers. The disadvantage is the knowledge of the patient as to what is taking place. He may become alarmed and turbulent, and may thus interfere with a necessary procedure at a vital moment. I have operated under infiltration anesthesia with satisfaction in the following cases: Gastrostomy, tracheotomy, rib resection, goiter, inguinal colostomy, typhoid perforation, abscess of the lung, gangrenous appendicitis, appendicitis in the interval, radical cure of hernia, strangulated hernia, suprapubic cystotomy, extirpation of the external carotid artery (Dawbarn's operation), amputation through the thigh, ligation of the thyroid arteries, varicocele, hydrocele, circumcision, and ligation of the femoral artery. There are many methods of local anesthesia.

Fig. 798.—Gebauer's ethyl-chlorid tube.

**Freezing.**—*Ice* and *salt* may be used. Take ¼ pound of ice, wrap it in a towel, and break it into fine bits; add ⅛ pound of salt; then place the mixture in a gauze bag and lay it upon the part. The surface becomes pallid and numb, and in about fifteen minutes decidedly analgesic. A *spray of rhigolene* freezes a part in about ten seconds. It is highly inflammable. *Ether-spray* anesthesia was suggested by Benjamin Ward Richardson. *Chlorid of ethyl* comes in glass or metal tubes (Fig. 798). Remove the cap from the tip of the tube and hold the bulb in the palm: the warmth of the hand causes the fluid to spray out. Hold the tube some little distance from the part, and let the fine spray strike the surface. The skin blanches and whitens, and is ready for the operation in about thirty seconds. Freezing is only of value in a trivial operation and when only a single cut or stick is required.

**Hypodermatic Injection of Cocain Hydrochlorate.**—Cocain was discovered by Gaedeke in 1855. In 1884 Köller, of Vienna, demonstrated the value of cocain as an analgesic in ophthalmic practice. In 1885 J. Leonard Corning, of New York, showed that cocain when applied to a mixed nerve in man abolishes nerve conduction, as it was already known to do in the lower animals. In 1885 Halsted and Raymond induced anesthesia in the nerve distribution by injecting cocain about the inferior dental and lingual nerves (perineural injection). This was done before pulling a tooth. Schleich introduced infiltration anesthesia. Braun, Van Hook, and Matas did much to develop local anesthesia. A tremendous impetus was given to infiltration anesthesia by Harvey Cushing's report on herniotomy under local anesthesia ("Annals of Surgery," 1900, vol. xxxi). He used a very weak solution of cocain (1 : 1000). Cocain hydrochlorate is soluble in water, but should not be boiled in water. To boil it impairs its anesthetic power. A tablet of the drug should be sterilized by dry heat and dissolved in sterile water. Always bear in mind that cocain is sometimes a decidedly dangerous agent. There are a number of deaths from cocain on record. The urethra is a particularly dangerous region, and so is the face. It is undesirable to use more than ⅔ gr. upon a mucous surface, and to inject

hypodermatically more than ⅓ gr. The drug must never be injected into a vein. Moderately severe cases of cocain-poisoning are characterized by great tremor, restlessness, pallor, dry mouth, talkativeness, and weak pulse. In dangerous cases there is syncope or delirium. Death may arise from paralysis or from fixation of the respiratory muscles. Cases with a tendency to respiratory failure require the hypodermatic injection of strychnin. In cases with tetanic rigidity of muscles hypodermatic injections of nitroglycerin or inhalations of the nitrite of amyl should be given. In cases marked by delirium, if the circulation is good, hyoscin is given. In any case stimulants are given, a catheter is used, and diuresis encouraged. Cocain-poisoning is always followed by a wakeful night. Cocain should not be used in any considerable amount if the kidneys are inefficient. In using cocain try to prevent poisoning. Because of the dangers inherent in cocain, have the patient recumbent. One minute before giving the cocain administer hypodermatically 1 drop of a 1 per cent. solution of nitroglycerin and repeat the dose once during the operation. In operating on a finger, after making the part anemic, tie a tube around the root of the digit before injecting cocain, and after the operation gradually loosen the tube. A hot solution of cocain is more efficient than a cold solution, hence hot solutions can be used in much less strength and are safer. The method of injection is as follows: A sharp needle is held at an angle of 45 degrees to the surface and is pushed into the Malpighian layer. One or 2 min. of a 2 per cent. solution are forced into the Malpighian layer, and a whitened elevation forms. The needle is withdrawn, at the margin of the wheal is reinserted, and more fluid is introduced, and so on until the region to be operated upon has been injected. After waiting five minutes the operation is begun. If, after cutting the skin, it is necessary to cut the subcutaneous tissue, inject a few drops of a 1 per cent. solution into the tissue. After the completion of the operation, if a rubber band was used, it is loosened for a few seconds, tightened for a few minutes, again loosened and readjusted, and so on several times (Wyeth). In this way only a small quantity of cocain is admitted into the circulation at one time and toxic symptoms are prevented. For operations upon the eye a 1 to 4 per cent. solution is employed; 1 drop of fluid is instilled every ten minutes until 3 drops have been given. Rarely use over a 10 per cent. solution on mucous membrane, although in laryngeal operations a 20 per cent. solution may be required. For the nasal mucous membrane a bit of wool soaked in a 5 per cent. solution is inserted or a spray of 4 per cent. solution is thrown from an atomizer into the nostrils. In the rectum, vulva, vagina, and uterus use a 5 per cent. solution; in the urethra, a 4 per cent. solution, and in the bladder, a 2 per cent. solution.

**Cocainization of a Nerve-trunk.**—Krogius, Halsted, and Raymond pointed out that if cocain is injected into the tissue about a nerve-trunk (perineural injection), anesthesia will follow in the area supplied by the nerve. The anesthesia will be produced in five minutes, and will last fifteen minutes. If cocain is injected about the root of the finger, all of the tissues of the digit will become insensitive. Injection over both supra-orbital notches renders the middle of the forehead insensitive. Injection over the ulnar nerve causes complete anesthesia of its trajectory. This plan is extensively used in Helsingfors.

It has been demonstrated by Crile ("Jour. Am. Med. Assoc.," Feb. 22, 1902) that the injection of cocain into a nerve-trunk (endoneural injection) interposes an absolute block to the transmission of afferent and efferent impulses and greatly lessens operative shock. In 3 cases I employed this method to secure anesthesia for amputation of the leg. None of the patients felt pain and shock was trivial.

In two amputations of the entire upper extremity, although the patient was under ether, the brachial plexus was cocainized to minimize shock and

shock was very slight. The cocain was injected directly into the trunks (endoneural injection). The combination of local anesthesia and general anesthesia is part of Crile's **anoci-association operation,** a plan to rule out all noxious or noci influences ("Jour. Am. Med. Assoc., July 13, 1912). He points out that though a person under ether is without feeling, the greater part of the brain is still awake, nerve impulses still reach the brain and cause functional depression and morphological alterations in the brain cells (Crile's "Ether Day Address," 1910). He would prepare a patient by filling him with calm confidence, giving him morphin and scopolamin previous to operation, administering nitrous oxid, and isolating the brain from the field of operation by infiltrating the region with 1 : 400 solution of novocain. At the conclusion of the operation the region may be injected with the hydrochlorid of quinin and urea.

**Eucain hydrochlorate** (β-eucain) is far safer than cocain used in full doses, and in many cases is to be preferred to it. It is injected in the strength of from 2 to 5 per cent. It is soluble in water and can be boiled without destroying its properties, and hence can be readily rendered sterile. It occasionally, though rarely, happens that the injection of eucain causes sloughing, especially at the extremities, in fatty tissue, in tendon-sheaths, and in bursæ. It can be used on mucous membranes.

**Stovain.**—This agent is a local anesthetic introduced by Fourneau. It is as powerfully analgesic as cocain, is only one-third as toxic, and is slightly germicidal. It is dissolved in cold water or salt solution, and a solution used of the strength of 0.5 per cent. Adrenalin can be given with it. (See Sonnenburg, in "Deutsche medicinische Wochenschrift," March, 1905.)

**Quinin-urea hydrochlorid** in from $\frac{1}{4}$ to 1 per cent. solution causes prolonged local anesthesia. A solution of 1 per cent. is too strong—it causes the wound to heal slowly and produces induration. The drug has no toxic effect and can be used in considerable quantity; it lessens bleeding, and, by producing an effect for many hours, lessens postoperative pain. Quinin-urea hydrochlorid is used by infiltration (see below). It is dissolved in water or normal salt solution.

**Novocain** of a strength in solution of from 1 : 200 to 1 : 400 is preferred by some surgeons. It may be given alone or mixed with stovain. It is given by infiltration. The drug is soluble in water and the solution can be boiled without being impaired in anesthetic power. It is one-sixth as toxic as cocain.

**Infiltration anesthesia** is in most instances the preferred method of local anesthesia. It is a term used to indicate a form of local anesthesia in which the tissues are not only injected, but are distended decidedly with a fluid anesthetic indifferent in nature. It is called *terminal anesthesia* because the anesthetic acts upon the terminal branches of sensory nerves. It is called *regional anesthesia* because the fluid injected affects a particular part of a sensory nerve in its course. Infiltration anesthesia was devised by Schleich, of Leipsic, who was dissatisfied with cocain, because it is not safe and sometimes fails to produce complete local anesthesia, owing to want of thorough diffusion. He found that salt solution (0.2 per cent.), if injected into uninflamed parts, produced anesthesia. To obtain this anesthesia the part must be distended by wide infiltration. If minute quantities of cocain, morphin, and carbolic acid are added to the solution, the anesthesia becomes more thorough and more prolonged, and can be obtained even in inflamed areas. Schleich uses three solutions:

No. 1, a strong solution, which is used in inflamed areas: cocain hydrochlorate, 3 gr.; morphin hydrochlorate, $\frac{2}{5}$ gr.; sodium chlorid, 3 gr.; distilled sterile water, $3\frac{2}{5}$ oz.; phenol (5 per cent.), 2 drops.

No. 2, medium solution, which is employed in most cases: cocain hydrochlorate, $1\frac{1}{2}$ gr.; morphin hydrochlorate, $\frac{2}{5}$ gr.; sodium chlorid, 3 gr.; distilled sterile water, $3\frac{2}{5}$ oz.; phenol (5 per cent.), 2 drops.

No. 3 is the weak solution used to infiltrate extensive areas: cocain hydrochlorate, $\frac{1}{6}$ gr.; morphin hydrochlorate, $\frac{2}{3}$ gr.; sodium chlorid, 3 gr.; distilled sterile water, $3\frac{2}{3}$ oz.; phenol (5 per cent.), 2 drops.

The addition of adrenalin chlorid to the cocain solution is an advantage, as it retards the circulation and hence favors analgesia and lessens bleeding during the operation. A satisfactory fluid for infiltration is 1 part of a 1 : 1000 solution of adrenalin chlorid and 9 parts of a 0.5 per cent. solution of cocain (Gangitans, in "Riforma Medica," Sept. 9, 1903). Eucain and adrenalin are preferred by some. Barker uses distilled water, 100 gm.; pure sodium chlorid, 0.8 gm.; $\beta$-eucain, 0.2 gm.; chlorid of adrenalin, 0.001 gm. After injecting Barker's fluid the surgeon waits for twenty minutes before operating.

The injections are begun *in* the skin, not *under* it (Fig. 799), and are made one after another until the area to be operated upon is surrounded above, below, and on all sides with Schleich's solution. At each infiltrated area a wheal forms in the skin. This infiltration can be made painlessly by touching with pure carbolic acid the point where the needle is to be inserted, or by freezing this spot with ethyl chlorid. After infiltration of the skin with the cocain solution the surgeon waits for a minute or two and then operates; incision is made, and when deeper tissues are reached they are infiltrated before incising them. If a nerve comes in sight, touch it with a drop of pure carbolic acid. Van Hook says that the anesthesia obtained by this method is due to artificial

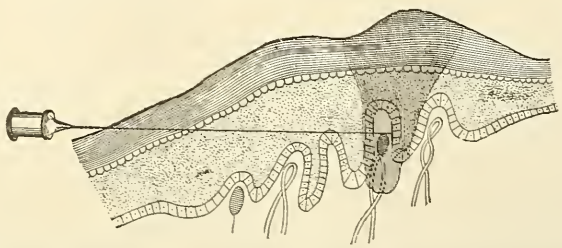


Fig. 799.—The syringe-point stops at the papillary layer, and the fluid lodges in the skin itself (Van Hook).

ischemia, pressure upon the tissues, the direct action of the drugs, and the lowered temperature.[1] The method is very efficient, and can be used for operations of considerable magnitude. Matas uses a special apparatus to infiltrate the tissues. The fluid is driven by compressed air, and widespread or *massive infiltration* is produced.

An ideal fluid to be injected should be isotonic with the blood, and should contain the smallest percentage of chemical agent necessary to render the part anesthetic. The solution recommended by Dr. James Mitchell, of Washington, D. C., seems to meet these requirements as closely as practically possible. Mitchell uses a tablet containing $\frac{3}{4}$ gr. of cocain and $\frac{1}{40}$ gr. of adrenalin. The tablets are dry sterilized, and just before operation are dropped into cups containing normal salt solution. Two strengths of solution are prepared: one tablet is dissolved in a cup containing 50 c.c. of the saline solution, one in a cup containing 100 c.c. The stronger solution is used for infiltrating the skin, blocking nerves, or for any particularly sensitive area; the weaker solution is employed for general infiltration of tissue. As Karl G. Lennander ("Local and Subarachnoid (Spinal) Anesthesia," in "Keen's Surgery," vol. v, page 1054) has pointed out, "Cocain has a greater affinity for the sensory than for the motor nerves. The effect of cocain in a certain proper concentration upon peripheral mixed nerves is to abolish, first,

[1] "Med. News," Nov. 16, 1895.

the sense of tickling, then the sense of temperature, thereafter the sense of pain, and, lastly, the sense of touch (pressure), and only thereafter the motor faculty, which may remain unaffected, although all the modalities of sensation have been paralyzed. During the retrogression of poisoning the faculty of motion is the first to return, then the sense of pressure, next the sense of pain, and lastly the sense of temperature." The addition of the adrenalin to the cocain solution is an advantage, in that it retards the circulation by contracting non-striped muscle-fibers in arteries and capillaries. The cocain is thus localized, its anesthetic action prolonged, and bleeding diminished during the operation. I do not believe that adrenalin in any way modifies the toxic action of cocain; indeed, Berry ("Am. Jour. Med. Sciences," Nov., 1905) seems to prove that it actually increases it. With the extremely weak solution recommended by Mitchell I have never seen the slightest physiological action of cocain. The total dosage of cocain given by infiltration should not exceed $1\frac{1}{2}$ gr. In 1 case I observed superficial sloughing of the skin when, by an operating-room error, three tablets had been dissolved in 50 c.c. of saline. I believe the slough was due to the high percentage of adrenalin.

The field of operative achievement under infiltration anesthesia has broadened to such an extent that it is difficult to definitely limit its possibilities when it is in the hands of one skilled in its use. It is necessary for the surgeon operating by this method to use an essentially different technic from that commonly employed in operations under a general anesthetic. He should be calm, equable, content to wait, and patient in spite of delay. He must be skilled in clean operative dissections, and accustomed to refrain from handling tissues unnecessarily. Failure is certain if the operator is not constantly engaged in this kind of work, for he must have learned, above all things, gentleness and willingness to spend plenty of time, and must have acquainted himself accurately with the sensibilities of the various tissues encountered in different operative fields. For the occasional operator to attempt major operations under infiltration anesthesia is to court failure. The patient should be informed that operation is to be a matter of intelligent co-operation, and in major operations it is wise to give a hypodermatic injection of morphin ($\frac{1}{4}$ gr.) a half-hour before beginning. This is usually sufficient to allay the apprehension natural in one about to undergo an operation. A tactful assistant should be detailed to sit by the patient's head and engage him in conversation, sponge his face, administer small sips of water, and, in other words, be what may be called the "psychic" anesthetist. The patient should not see any of the preoperative preparations; instruments should be carefully excluded from the range of his vision. Needless conversation and other noise should be avoided.

The special instruments required are: two Record syringes of 2 c.c. capacity and one of 5 c.c.; two fairly fine needles $1\frac{1}{2}$ inches in length and two coarser needles 2 inches in length; a pair of Mayo's $5\frac{1}{2}$-inch straight dissecting scissors with keen edges; dissecting forceps with teeth, and fine-pointed artery clamps. For example, let us take an appendectomy in a patient with a frank tuberculosis of the lung; a case in which general narcosis would cause damage and might prove fatal. The patient, having been prepared for operation (care having been taken to administer morphin a half-hour previously), is placed upon the operating table and made as comfortable as possible. The head is arranged upon a pillow, the arms are disposed comfortably at the sides, and a folded blanket is placed under the lumbar spine. The patient's wishes may be consulted as to whether he desires a piece of gauze placed over his eyes or not. Usually the patient, unless addressed, keeps his eyes closed. The psychic anesthetist engages the patient in conversation, the surgeon must show full confidence, be cheerful, and endeavor to communicate this frame of mind to his subject.

The easiest method of approach to the appendix under infiltration anesthe-

sia is McBurney's muscle-splitting incision. Begin infiltration by taking the 2 c.c syringe filled with the stronger cocain and adrenalin solution. Use a fine needle. Pinch up a small piece of skin, informing the patient that you are going to stick him slightly with a fine needle. Holding the needle almost parallel with the skin, barely insert the point under the epidermis and, at the same time, press on the piston of the syringe so as to obtain an infiltrated spot simultaneously with the introduction of the needle. The pain caused by the first introduction of the needle is trivial and even that degree is absent during subsequent insertions. A small wheal is produced, the integument assuming a blanched, pig-skin appearance. From the center of this infiltrated circle the needle may now be inserted painlessly to its full length *in* the skin (not *under* the skin), parallel with the surface. Do not insert the needle too deeply at this time. As the point of the needle travels the solution should be fed from the syringe. Ordinarily 2 c.c. of solution will infiltrate $1\frac{1}{2}$ to 2 inches of skin. After the superficial infiltration a coarser and longer needle should be used to infiltrate subcutaneous tissues. The incision may now be carried down to the aponeurosis of the external oblique. Infiltrate the muscular and aponeurotic portion of the external oblique. Make a split in the aponeurosis by a knife and continue by Mayo's scissors. Gentle clips with the scissors cause less pain than knife dissection. Retract carefully the aponeurotic layer. Infiltrate the internal oblique and split it. The transversalis and peritoneum are infiltrated with the stronger solution, which we employ at this point for the first time since the infiltration of the skin. These layers are then incised. Incisions in all layers of the abdominal wall must be of ample size to avoid unduly strong retraction. Up to this stage the patient should experience no pain unless one has thoughtlessly clamped a vein without previous infiltration with the stronger solution. The visceral peritoneum may be cut, clamped, burned, and stitched with impunity, but undue traction upon the mesentery immediately causes general cramp-like abdominal pain with consequent rigidity of the abdominal wall. The appendix, therefore, is sought for by gently following the anterior longitudinal band intra-abdominally rather than by attempting to pull the cecum out of the abdominal incision. Once the base of the appendix is found, the rest is easy. Traction on the meso-appendix must be gentle, and before tying it infiltrate it with the stronger solution. The crushing ligation and inversion of the appendix is not accompanied by pain. Occasionally the necessary traction on the mesocolon will cause the patient to have nausea, which is relieved as soon as the traction ceases. Suture of the abdominal wall now follows without special incident, except that very small gutta-percha tissue drains should be inserted just under the skin at one or both ends of the incision when infiltration anesthesia has been used. These drains take care of any possible oozing that may occur after the effect of the adrenalin has worn off.

By the use of infiltration anesthesia I have operated with satisfaction in the following cases: Tracheotomy, tuberculous glands of the neck, rib resection, goiter, drainage of a cerebral cyst, ligation of the thyroid arteries, gastrostomy, inguinal colostomy, typhoid perforation, abscess of the lung, chronic and acute appendicitis, appendiceal abscess, jejunostomy, radical cure of inguinal, femoral, and umbilical hernia, strangulated hernia, incisional hernia, resection of the bowel, suprapubic cystostomy, extirpation of the external carotid artery (Dawbarn's operation), ligation of the femoral artery, cholecystostomy, suture of fractured patella, amputation of the arm, amputation of the thigh, amputation of the leg, removal of stone from the pelvis of the kidney, posterior gastroenterostomy, hysteropexy, kidney abscess, psoas abscess, and large numbers of minor conditions.

Patients after abdominal section under infiltration anesthesia very rarely

vomit at all, never have as much postoperative pain as those who were under a general anesthetic, and seldom have backache. Distention of the bowel is uncommon. The catheter is rarely required, and, of course, the blood changes, renal difficulties, and postoperative pneumonias are much less frequent than after general narcosis. Infiltration anesthesia cannot be ignored in such cases as diabetes, Addison's disease, sepsis, advanced Basedow's disease, diseases of the cardiac muscle, liver, both kidneys, etc. In children and high-strung individuals infiltration is usually impossible; in operations for extensive malignant growths, or during which complete muscular relaxation must be obtained, this form of anesthesia is absolutely contra-indicated.

To those interested in infiltration anesthesia I would suggest a careful study of Spalteholz's admirable illustrations of nerves (especially those of the extremities),[1] together with the perusal of the following writings:

H. Braun: "Die Lokalanästhesie, ihre wissenschaftlichen Grundlagen und praktische Anwendung," Leipzig, 1907. (A very full bibliography accompanies this excellent work.)

Harvey Cushing: "Observations Upon the Neural Anatomy of the Inguinal Region Relative to the Performance of Herniotomy Under Local Anesthesia," in "Annals of Surgery," 1900, vol. xxxi.

Theodor Kocher: "Text-Book of Operative Surgery," third English edition, by Harold J. Stiles and C. Balfour Paul, vol. i, page 16.

Karl G. Lennander: "Local and Subarachnoid (Spinal) Anesthesia," in "Keen's Surgery," vol. v, page 1045.

James F. Mitchell: "The Production of Local Anesthesia for Surgical Purposes," in "American Practice of Surgery," by Bryant and Buck, vol. iv, page 231.

**Bier's Intravenous Method of Local Anesthesia.**—This plan was described by Bier at the German Surgical Congress of 1908.[2] It permits of serious operations upon the limbs, operations for which ordinary methods of local anesthesia would prove quite inefficient. Suppose the surgeon intends to resect an elbow-joint: Mark the position of the veins on the asepticized extremity. Apply an Esmarch bandage from the tips of the fingers to well above the elbow-joint. This is the expulsion bandage. A thin soft-rubber band is applied around the arm above the Esmarch bandage, the bandage is removed, and a like band is applied below the elbow. The anesthetic is injected into a superficial vein of this bloodless area (the basilic or cephalic). The tissues above and about the vein are infiltrated. The vein is exposed, the syringe of the cannula is introduced, and ligatures are used as though we were going to give an ordinary intravenous injection of salt solution, except that the cannula is pointed to the periphery. The fluid used is a 0.25 or 0.5 per cent. solution of novocain. The syringe, containing 50 c.c. of fluid, forces the solution downward into the veins and the limb swells.

If the stronger solution is used, 50 c.c. are enough; if the weaker solution is used, 100 c.c. will be required. If resecting the knee, the injection should be made into the internal saphenous vein and twice the amount would be necessary as advised for the elbow.

After injecting the strong solution operation may be begun at once. After injecting the weak solution we should wait ten minutes. The bloodless area between the bands becomes anesthetic very promptly after the injection, the bones as well as the soft parts. The peripheral portion of the limb beyond the area between the bands becomes anesthetic after a short time and motor paralysis may follow. Such paralysis is eventually recovered from. If anesthesia between the bands is not attained within five minutes there has been some failure in technic (Bier, "Edinburgh Med. Jour.," 1910, v, No. 2). The

[1] "Hand Atlas of Human Anatomy," by Werner Spalteholz. Edited and translated by Lewellys F. Barker.

[2] Bier: "Ueber einen neuen Weg Lokalanästhesia au den Gliedmaassen zu erzeugen," "Arch. f. klin. Chir.," 1908, lxxxvi, No. 4.

analgesia is entirely satisfactory and passes away as soon as the band is removed. When the operation has been completed, wash out the vein with salt solution in order to prevent toxic effects.

By this method the anesthetic passes through the vein walls and becomes fixed in the tissues, and when the bands are removed it returns very gradually to the circulation, hence greatly larger doses may be given than should be admissible by any other method. Adrenalin should not be given with the novocain. Toxic symptoms are rare. The method is contra-indicated if there is arteriosclerosis. (See Carroll Smith, in "Jour. Am. Med. Assoc.," March 23, 1912; Page and McDonald, in "Lancet," Oct. 16, 1909; Hitzrot, in "Annals of Surgery," 1909, vol. i.)

**Anesthesia by Infiltration with Sterile Water.**—When the tissues are well infiltrated with warm or cold sterile water, anesthesia ensues promptly. I have not found it as complete as when cocain or eucain is employed, even when a considerable amount of fluid is introduced. Gant uses it in rectal operations and commends it strongly ("New York and Phila. Med. Jour.," Jan. 28, 1904).

**Spinal Analgesia.**—J. Leonard Corning in 1885 discovered that cocain injected between the spines of the eleventh and twelfth dorsal vertebræ produces analgesia of the lower limbs ("New York Med. Jour.," Oct. 31, 1885). From this observation spinal anesthesia springs. Bier produced complete anesthesia of the entire body except the head by the injection of a small amount of cocain into the subarachnoid space of the spinal cord. A solution of cocain of a strength of from 0.5 per cent. to 1 per cent. is used by some, but cocain cannot be boiled without impairment of its anesthetic power, and carbolic acid must be added to it in small amount. Hence cocain so prepared is not certainly sterile, and the carbolic acid added may induce harmful symptoms. (See Neugebauer, in "Wien. klin. Woch.," 1901, Nos. 50, 51, 52.) Some surgeons use a solution of eucain which can be boiled, but it is not so rapid and certain as cocain. Some use tropacocain (Illwicz). A solution of this drug can be boiled, is less poisonous than cocain, and somewhat slower in action. Experimenters tell us that ½ to 1½ gr. of cocain may be given, but it is not wise to give over 0.5 gr. I have used stovain in a number of cases. Some combine it with adrenalin, but the combination is not desirable in the subarachnoid space. The dose is 1 c.c. of a 5 per cent. solution. The analgesia lasts from one-half an hour to an hour or more, and was followed in my cases by retention of urine. Some have used novocain alone or combined with adrenalin.

A. W. Morton ("Jour. Am. Med. Assoc.," Nov. 8, 1902) takes chemically pure crystalline hydrochlorate of cocain, places it for fifteen minutes in a dry temperature of 300° F., and puts it in sterile tubes until wanted. The dose depends upon the locality in which he wishes to induce analgesia, and varies between 0.3 and 0.5 gr. The required dose is placed in the barrel of the sterile syringe and is dissolved in cerebrospinal fluid drawn into the syringe for that purpose. I now follow the plan of Mr. Arthur E. Barker. He believes that the specific gravity of the fluid containing the drug plays an important part in its localization within the canal ("Brit. Med. Jour.," March 16, 1912). He uses a fluid as nearly as possible isotonic with the blood. It consists by weight of 5 parts of stovain, 5 parts of glucose, and 90 parts of distilled water. Adrenalin is never added. It may do harm, it can do no good. This fluid seeks the lowest level it can find and mixes but little with the cerebrospinal fluid. The average dose is 1 c.c. of the 5 per cent. solution.

The syringe used is of glass and of a capacity of 2 c.c. The needle is the hollow one of Bier. The needle must be sharp, else it would not go through the dura. Barker's fine blunt cannula goes through the lumen of the needle.

The fluid to be injected is kept in sealed Jena glass ampoules, and should not

be more than a week or two old. The fluid is drawn into the syringe from the ampoule. The patient lies upon his side with the head and shoulders well raised and with the back curved. The back has been previously sterilized. The dressings are removed and the region to be punctured is resterilized. The spines of the third and fourth lumbar vertebræ are located, and the needle is entered in the midline beneath the spine of the third or fourth lumbar vertebra and is pointed upward and forward. The surgeon determines that he has punctured the subarachnoid space by lessened resistance and the appearance of fluid at the needle-opening. The injection is made slowly, the needle is withdrawn, and the puncture sealed by collodion. In performing the operation care must be taken to prevent the escape of the cerebrospinal fluid.

If the patient remains upon the side the nerve-roots of the dependent side are rendered analgesic some time before those of the uppermost side. If he is turned for a time upon the other side the nerve-roots of that side will quickly become anesthetized. To turn him upon the back will do the same thing, but less rapidly. If he is placed in a sitting position the rectum and anus are quickly rendered anesthetic.

The usual position for operation is on the back, but he may perhaps be upon his side. When the patient is placed upon his back promptly after injection the anal region becomes anesthetic in from one to two minutes, the lower extremities in from three to six minutes, and the upper extremities in from fifteen to thirty minutes. The anesthetic condition lasts from one to three hours or even longer, and is due to the contact of cocain with the nerve-roots (A. W. Morton, "Jour. Am. Med. Assoc.," Nov. 8, 1902).

After cocainization of the spinal cord surgical operations can be performed on many regions without causing pain. Among the operations which have been performed are resection of the knee, resection of the ankle, osteotomy, amputation of the leg, amputation of the thigh, hysterectomy, perforation of gastric ulcer, intestinal obstruction, strangulated hernia, excision of bowel, acute appendicitis, gastro-enterostomy, oöphorectomy, removal of ovarian cyst, and removal of rectum.

Spinal analgesia is not growing in popularity. It is regarded by most surgeons as a method to be used in exceptional cases. It should never be used as a routine procedure, and it will not displace ether or chloroform. By it analgesia can usually be secured. A. W. Morton (Ibid.) used it 673 times without a failure, and 60 of these operations were above the diaphragm. If we desire to obtain analgesia of the upper portion of the body the patient must be placed in the Trendelenburg position after the fluid has been injected. Most operators have had failures, especially above the diaphragm. In Sonnenburg's 1117 cases there were 78 utter failures ("Jour. de Chir.," Oct., 1908). Bier says that failures occur in 4 per cent. of cases; Moynihan says in 14 per cent.; Legueu says in one-seventh of the cases. In Barker's last 100 cases there were 3 failures. No one should attempt it who is not well trained in aseptic methods, because infection of the cord or its membranes will prove fatal. Untoward effects are common, and they may arise during or after the operation.

Sonnenburg had them in 193 out of 1117 cases. Among the untoward effects reported are grave collapse, temporary paralysis of the abducens nerve, of the facial nerve, of the hypoglossal nerve, meningitis, retention of urine, chills, elevation of temperature, incontinence of urine, persistent paraplegia, pain in the back and legs, perhaps lasting for weeks or even for months, nausea and vomiting during and after the operation, sweating, overaction of the heart, dimness of vision, cramps in the limbs, dyspnea, violent headache, involuntary evacuation of feces, and cardiac overaction. Many of the immediate symptoms are probably due to the absorption of the drug injected. The headache is due to tension and is relieved when some cerebrospinal fluid is with-

drawn by lumbar puncture. In 20 per cent. of Barker's cases ("Brit. Med. Jour.," March 16, 1912) there was more or less transient headache. In 16.3 per cent. there were nausea and vomiting.

Headache, vertigo, weakness, paresthesia, neuralgia may, in a few cases, persist for months or even years (Hohmeier and König, in "Archiv. für klin. Chir.," Oct. 8, 1910).

Whether or not permanent harm ever comes to the cord is not certain. Bristow ("Brooklyn Med. Jour.," 1902, xvi, page 410) reported the case of a man, fifty-five years of age, on whom he operated for hemorrhoids after spinal cocainization. An examination one month later indicated degeneration of the posterior and lateral columns of the cord (spastic lower extremities, ataxic gait, increased knee-jerk, ankle-clonus, and inability to retain urine). Marx ("New York Med. Record," Dec. 22, 1900) states that 1 case in his experience, after cocainization of the spinal cord, developed typical locomotor ataxia. Dandois ("Jour. de Chir. Brux.," April–May, 1901) reports a case upon which he had operated for traumatic rupture of the urethra. Spinal cocainization was employed. Paraplegia developed and lasted two months. Several cases of hemorrhage into the subarachnoid space are on record. Legueu states that persistent paraplegia and persistent incontinence of urine may arise ("Rev. de Chir.," Oct., 1908).

Is there any danger of death from spinal analgesia? If the operation is not performed with scrupulous aseptic care it is very dangerous. Even when performed by the best surgeons death may occur. Tuffier places the mortality at 3 in 2000, but excludes from consideration 3 deaths ("La Presse Médicale," vol. lv, 1901, page 190). Reclus finds 6 deaths in less than 2000 cases (Address before the Paris Academie de Médicine, March 19, 1901). Hahn, in 1708 cases collected from literature, found 8 deaths ("Mitt. a. d. Grenzgeb. d. Med. u. Chir.," 1900, iii, 337). The mortality is usually supposed to be about 3 in evey 1000 cases. Wm. N. Perkins ("New Orleans Med. Jour.," Jan.–Sept., 1902) collected 2345 cases with 16 deaths or 1 death in 146 administrations. Strauss's table shows 46 deaths in 22,717 cases (quoted by Hardonin in "Archiv. Générale de Chir.," August, 1908). In Barker's 2354 cases there were only 3 deaths "which could be in any way put down to the spinal method of anesthesia" ("Brit. Med. Jour.," March 16, 1912). Hohmeier and König ("Archiv. für klin. Chir.," Oct. 8, 1910) collected 2400 cases of spinal anesthesia: 12 deaths were due to it directly; 4 of them died of paralysis of respiration; 7 of the fatal cases were over seventy years of age. One victim was only thirty-two.

Cocain seems to act like a toxin on the pia and arachnoid. Examination of fluid withdrawn after the performance of cocainization shows that it contains polymorphic leukocytes (Ravant and Aubourg, in "Gaz. Hebd. de Méd. et de Chir.," June 27, 1901).

My belief is strong that the method should only be used for operations below the diaphragm, and I hold this belief in spite of the claim of Jonnesco that he operates with the aid of spinal anesthesia on any part of the body. It is most successful in operations below the umbilicus. I very seldom use it for operations above that level.

In a case in which, because of heart disease, pulmonary disease, kidney disease, or some other condition in which a general anesthetic is inadmissible, spinal cocainization is justifiable. It should be reserved exclusively for cases in which other forms of anesthesia are positively contra-indicated. The method should not be employed on those under fifteen years of age or on the subjects of central nervous disease. Barker disapproves of spinal anesthesia in "cases of extreme asthenia due to carcinoma, and of advanced toxemia depending upon septic peritonitis or obstruction, especially in later life" ("Brit. Med.

Jour.," March 16, 1912), although some surgeons regard such conditions as particularly demanding spinal in preference to general anesthesia.

A solution of Epsom salts has been used by Blake, Haubold, and Willy Meyer. It was discovered (Meltzer and Auer, "Am. Med.," Nov. 25, 1905) that subcutaneous injections of salts of magnesium produce local anesthesia. The same investigators later pointed out ("Med. Record," Dec. 16, 1905) that subarachnoid spinal injections of Epsom salts produce widespread and complete anesthesia. A 25 per cent. solution is used and 1 c.c. of this is given for every 25 pounds of body weight. After a wait of three or four hours the drug causes paralysis and analgesia of the legs and pelvic region. Sensation and motion do not return for from eight to fourteen hours. Retention of urine may last two days. The pulse and blood-pressure are unaffected, but the respiration is slowed. Large doses would endanger life by respiratory arrest. In view of the fact that in some cases the effect of the drug is inordinately prolonged, it is wise, when the operation is completed, to puncture the theca of the cord again and wash it out with salt solution. Guthrie and Ryan ("Amer. Jour. of Phys.," August 1, 1910) deny that magnesium salts have specific anesthetic properties and claim that anesthesia following their injection is due chiefly to asphyxia.

## XXXI. DISEASES OF THE SKIN AND NAILS

**Dermatitis venenata** is a dermatitis resulting from irritants. It may be caused by wearing garments containing arsenic. A common cause is rhus-poisoning. Rhus-poisoning arises from the poison-oak, the poison-ash, the poison-ivy, and some other species of sumach. Actual touching of the plants is usually, but not always, necessary. Some suffer if they simply come near them. Some people are immune to rhus-poisoning, some are slightly susceptible, some are strongly predisposed. It is believed that toxicodendric acid is the irritant agent. The condition is most apt to arise when the skin is moist from perspiration.

The **symptoms** are burning, itching, redness, and edema of the affected parts. The hands and forearms are most apt to suffer, but any part may be attacked. If the penis and scrotum suffer from rhus-poisoning there is great swelling from edema. An eruption on the hands may inoculate the penis when that organ is handled. A vesicular eruption begins between the fingers. The eruption becomes violently inflammatory, and in the form of fierce red edematous inflammation spreads widely over the body. There may be slight fever. The condition usually begins to abate in two or three days and desquamation follows.

When one, knowing from experience that he is predisposed, feels the inaugural itching, he should at once apply to the parts a 1 per cent. solution of lactic acid in 95 per cent. alcohol (R. F. Ward, in "New York Med. Jour.," Dec. 26, 1908).

The **treatment,** when a moderate area is involved, comprises the application of cloths wet with a wash of lead-water and laudanum, or a saturated solution of acetate of aluminum (R. F. Ward, Ibid.). If an extensive area is involved, apply grindelia robusta (4 dr. to 1 pint of water) or moisten the surface frequently with sweet spirits of niter. Oxid of zinc ointment, containing 10 gr. of carbolic acid to 1 oz., gives great relief.

**Furuncle (Boil).**—(See page 137.)

**Aleppo Boil** (*Endemic Boil of the Tropics, Delhi Boil, Oriental Sore,* etc.).—Papules appear upon the exposed parts of the body. These papules, which ulcerate, do not cicatrize for at least a year, and leave ineradicable scars. The

condition is due to a protozoan. Man is infected by means of flies, lice, or other insects. The Aleppo boil was once apparantly confined to India, Arabia, Persia, Egypt, Algeria, etc. Of late it is said to have appeared in Panama, the Philippine Islands, and Hawaii.

**Erysipelas.**—(See page 199.)

**Erysipeloid.**—(See page 198.)

**Clavus, or Corn.**—A corn is a tender painful, and circumscribed thickening of the epidermis, and is commonest over one of the joints of the toes. *Hard* corns are situated on exposed parts of the digits; *soft* corns appear between the digits, where the parts are kept constantly moist. Corns are caused by pressure.

**Treatment.**—The wearing of well-fitting boots will usually cause a corn upon the toe to disappear. Soak the feet often in water containing bicarbonate of sodium, dry them, and apply a circular corn-plaster to the corn to take off the pressure of the boot. Another method is to touch the corn with iodin every night and pare away the hard tissue every morning. An old and valuable plan is to paint the corn every night and morning with a mixture composed of salicylic acid, 40 gr.; extract of cannabis indica, 10 gr.; and collodion and flexible collodion, of each, 2 dr. After several days of the treatment soak the parts in hot water and scrape away the mass. *Soft* corns are treated by washing the feet often with ethereal soap, drying, gently removing the sodden epithelium, dusting the toes and between them with borated talc, and placing absorbent cotton between the digits. Incurable soft corns require the removal of the skin from the adjacent sides of the two toes and suturing the toes together (thus converting two toes into one). In inflamed corns employ rest and lead-water and laudanum, and let out pus when it forms. Remember that in old persons the cutting of a corn may cause senile gangrene. In the inflamed and painful feet of a person who has corns nothing gives so much relief as washing the feet with ethereal soap, soaking in hot water, and wrapping the feet for half an hour in cloths wet with a mixture composed of linseed oil and lime-water, each, 2 oz., and spirits of camphor, 1 dr.

**Warts.**—(See page 379.)

**Onychia** is inflammation of the matrix of the nail. Syphilis often causes severe onychia which requires specific treatment (see page 326). A *run-around*, or paronychia, is suppuration of the matrix at the root of the nail, and of the skin about it, of traumatic origin. It requires incision, trimming away of the buried edge of the nail, and packing with iodoform gauze (see page 724).

**Malignant onychia,** which is inflammation and ulceration of the entire matrix, occurs only in a person of dilapidated constitution. This condition requires removal of the entire nail, cauterization of the matrix, dressing with iodoform gauze, and the internal use of stimulants, tonics, and nourishing diet.

**Ingrowing toe-nail** (see page 157) is sometimes due to lateral hypertrophy of the edge of the nail, but usually to forcing of the soft tissues over the margin of the nail. An irritable ulcer arises. The condition is treated by splitting the nail, removing the ingrown piece, the soft tissue at the margin and the adjacent matrix, and dressing antiseptically.

## XXXII. DISEASES AND INJURIES OF THE THYROID GLAND

The thyroid gland is an essential organ. It possesses functions of the first importance. It has a great influence upon nutrition. It acts by means of its secretion, which is an iodothyroglobulin.

An excess of this secretion if unneutralized in the body causes hyperthyroidism (see page 1236). A diminution of this secretion causes hypothyroidism

(see below). If there is no thyroid or a functionally inactive thyroid from birth, the child is a cretin (see below). A great deal of the gland can be parted with without harm. Charles H. Mayo estimates that one-sixth of the gland can furnish enough secretion for the body needs ("Illinois Med. Jour.," Feb., 1913). The older a person is, the less thyroid is apparently needed or, perhaps, the less thyroid, the older a person actually is. "According to Lorand, the deferring of old age requires the continued presence of some of the thyroid throughout life" (Charles H. Mayo, Ibid.). The thyroid of a woman is apt to enlarge at puberty and to become swollen from congestion before a period of menstruation. During pregnancy or at the menopause women are apt to exhibit symptoms of hypo- or hyperthyroidism. Charles H. Mayo (Ibid.) points out that increased secretion does not, of necessity, cause symptoms. A considerable excess may be neutralized in the body. In such a condition a shock may at once induce symptoms. Entire loss of the thyroid in an adult causes myxedema. Kocher pointed out that its complete removal in a young or middle-aged person usually causes *operative myxedema* (*cachexia strumipriva*) and perhaps *tetany*. Removal of the gland in an elderly person does not cause these curious conditions. Later knowledge indicates that removal of the thyroid with the parathyroids certainly produces myxedema or tetany, unless aberrant thyroids exist and compensate. Removal of the thyroid without the parathyroids does not induce tetany, even when there are no aberrant thyroids. The thyroid probably furnishes an internal secretion which destroys certain toxic products of metabolism. It is thought that the parathyroids furnish an antitoxin to poisons formed during digestion.

**Wounds** cause violent hemorrhage which is difficult to arrest. Ligatures may cut out and forceps will not hold. The hemorrhage is arrested by suture-ligatures, purse-string sutures, the actual cautery, or removal of the bulk of the gland.

The thyroid gland may be absent at birth. **Congenital atrophy** or **congenital hypertrophy** may exist.

**Acquired atrophy** leads to *hypothyroidism* and *myxedema*. Hypothyroidism may arise during any process destructive of the cellular activity of the thyroid gland. It is seen not unusually in women of from twenty-five to forty who have borne children. It sometimes occurs in men. The patient usually grows fat, is sterile, and neurasthenic. The temperature is subnormal. Complaint is made of headache, backache, shortness of breath, and indigestion. In women amenorrhea is the rule. In severe cases there is myxedema. (See Robert L. Pitfield, in "New York Med. Jour.," August 27, 1910.) *Myxedema* is a condition characterized by the presence of a firm subcutaneous swelling in the face, neck, and limbs; slow speech; mental dulness, and subnormal temperature. The condition is identical with that produced by removal of the entire gland.

**Cretinism** is a result of hypothyroidism. It is a form of infantilism and idiocy due to absence of the gland or atrophy of glandular elements in the thyroid. When atrophy of the parenchyma alone exists the size of the gland may be actually increased. The body is dwarfed; bone development is very defective, the face, neck, and extremities resemble those parts in myxedema, and a low grade of idiocy exists. Myxedema and cretinism are treated by the internal administration of thyroid extract.

**Thyroid Feeding and Grafting.**—Hypothyroidism, with or without myxedema, is greatly benefited by thyroid feeding or the administration of thyroid extract. Some experimenters have transplanted thyroids into thyroidectomized animals. The results show striking but temporary improvement. Such transplanted material eventually disappears. Experiments have been made and are being made on thyroid grafting in the treatment of cretinism and myxedema. Grafts have been placed under the skin, in bone at the junction of

the epiphysis with the diaphysis, in the spleen, and in other regions. Encouraging cases have been reported, but the results are temporary.

**Congestion of the thyroid** may be caused by violent exertion, prolonged effort, febrile maladies, and venous obstruction. It is treated by removing the cause and applying heat locally. Tracheotomy may be required.

**Inflammation of the thyroid** (*acute* or *inflammatory goiter*) may be induced by a septic or febrile malady, rheumatism, muscular strain causing vascular rupture, a wound, or contusion of the thyroid. Usually but one lobe is affected. The ordinary symptoms of inflammation are present. In addition there are dysphagia, dyspnea, venous congestion of the face, epistaxis, nausea and vomiting, and possibly delirium. It may terminate in resolution, suppuration, or fibrous induration.

**Tuberculosis of the thyroid** is usually a part of general miliary tuberculosis. It is very seldom that a local caseating focus occurs, but such cases have been reported.

**Syphilis of the Thyroid.**—Early in the secondary stage there is apt to be slight and painless thyroid enlargement. In the tertiary stage gummata may form.

**Tumors of the thyroid** are of various sorts. Among them are adenomata, cystic adenomata, sarcomata, and carcinomata. Eight cases of teratoma are on record (Isabella C. Herb, "Am. Jour. Med. Sciences," June, 1906). Malignant disease is unusual. I have operated on but 2 cases: 1 of cystic carcinoma in which operation was rapidly fatal, and 1 of round-celled sarcoma. The latter patient was living and apparently well four years after lobectomy. Malignant disease may arise in the normal, but is more apt to arise in a goitrous thyroid. In over 50 per cent. of the reported cases there is a history of antecedent goiter. Malignant disease is more common in women than in men and is very seldom met with before the age of thirty. It is most common between forty and sixty. One should always suspect malignant disease of the thyroid gland when the growth appears rather suddenly in a patient over forty years of age. If the growth is irregular in outline and is accompanied by pain and difficulty in swallowing, the diagnosis becomes reasonably certain. Later in the case there are symptoms due to pressure upon and infiltration of the nerves; the growth becomes firmly anchored and the lymph-glands adjacent to the thyroid become involved; there may be tracheal bleeding, and perhaps fever and eventually cachexia develops. Sarcoma or carcinoma may occur and it is seldom possible to determine clinically with which we are dealing. The cancer may be a scirrhus or an epithelioma, but is usually an adenocarcinoma. A sarcoma may be either of the round cell or spindle cell type. In malignant disease of the thyroid, metastasis occurs early in a great majority of cases, the

Fig. 800.—Sarcoma of thyroid gland.

lungs being first involved, and then the bones and other structures; though it has been stated that in adenocarcinoma the lungs are likely to escape and that solitary bone-metastasis is not infrequently noted. Sarcoma (Fig. 800) may involve one lobe, but carcinoma (Fig. 801), even at an early stage, is apt to involve both lobes (Berry, "Diseases of the Thyroid Gland"). These growths soon penetrate the gland capsule, become anchored to surrounding parts, and involve the vocal cords, trachea, and even the great vessels of the neck. Malignant growths if not cystic are apt to be hard and nodular and they grow rapidly. At first the gland moves with deglutition, but later becomes anchored to surrounding parts. In malignant disease of the thyroid it is usual to find difficulty of swallowing and paralysis of the vocal cord on the side of the growth. Malignant disease is rapidly fatal. Many die within six months and few survive over eighteen months. Radical operation is proper only before the growth breaks through the capsule, although at any stage it may be necessary to operate in order to prevent suffocation.

Fig. 801.—Cystic carcinoma of thyroid gland.

A **goiter**[1] is an enlargement of the thyroid gland not due to a malignant tumor or to inflammation. The enlargement may affect a small portion of the gland, one lobe, both lobes, or both lobes and the isthmus, and it may occur either sporadically or endemically.

There are a number of forms of ordinary goiter. The most common is what is called *simple* or *parenchymatous* goiter (Fig. 802). In this condition all portions of the gland enlarge, and the goiter is consequently bilateral. It does not appear first in one lobe and at a considerably later period in the other, but each lobe is enlarged equally or nearly equally. Parenchymatous goiter is often spoken of as simple goiter, and is sometimes, though not with entire accuracy, designated hypertrophy of the thyroid gland.

The common goiter of adolescence is "an edematous condition of the gland due to watery colloid" (C. H. Mayo, "Illinois Med. Jour.," Feb., 1913).

*Adenomatous goiter* (Fig. 803) is a condition due to the growth of encapsuled adenomata in the thyroid gland. There may be a single adenoma, but frequently there are multiple growths. One or both lobes may be involved. The goiter, however, usually seems to begin in one lobe; and if both lobes enlarge, one generally does so at a period distinctly subsequent to the enlargement of the other. In some cases growth seems to originate simultaneously in both lobes, but even then the growths seldom increase equally. Adenoma may develop in a healthy thyroid gland, but adenomatous growth is usually associated with some parenchymatous growth.

[1] For a study of the "Pathological Anatomy of Goiter" see W. C. MacCarty, in "New York State Jour. of Med.," Oct., 1912. This study is founded on 2500 cases from the Mayo Clinic.

*Cystic goiter*, or *bronchocele*, is a condition in which the chief mass of the enlargement is composed of a cyst or of multiple cysts. When cysts form, the thyroid gland is usually hypertrophied or adenomatous; occasionally, however, cysts form in a non-hypertrophied thyroid. The great majority of cysts are due to cystic degeneration of adenomata; some are formed by the running together of overdistended thyroid vesicles, and some few follow blood-extravasation into the thyroid tissue. The liquefaction is due to mucoid or colloid degeneration, and the fluid of the cyst is sometimes clear and thin, sometimes viscid, and often coffee-ground in appearance.

A *fibrous goiter* is a fibrous induration. It is likely to arise in old bronchoceles, which may actually pass into a calcareous condition. By the term *malignant goiter* is meant malignant disease of the thyroid gland, either carcinoma or sarcoma. As stated above, such cases are not really goiters. When hemorrhage takes place into a goiter the condition is often spoken of as a *hemorrhagic goiter*. A *colloid goiter* is a form of parenchymatous goiter in which there is an extremely large amount of colloid material. *Exophthalmic goiter* is

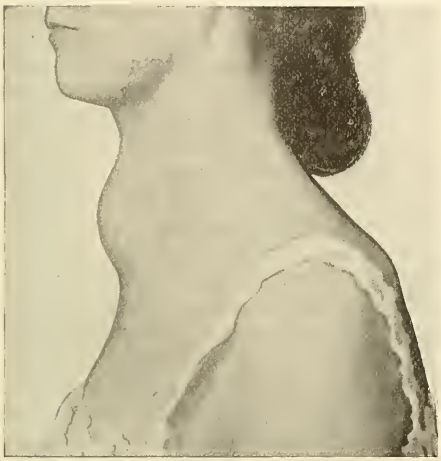


Fig. 802.—Parenchymatous goiter.

Fig. 803.—Adenomatous goiter.

discussed on page 1235. Occasionally a simple or an adenomatous goiter because of degenerative changes or overstimulation forms toxic material or an excess of secretion and causes symptoms of toxemia. "These cases may have all the nervous symptoms and heart complications of a bad case of Basedow's disease without the protruding eyes" (C. H. Mayo, "Illinois Med. Jour.," Feb., 1913). This evolution gives rise to what the French call a *Basedowified goiter* (Morestin, in "Rev. de Chir.," Nov. 10, 1899). A goiter that develops with great rapidity is sometimes called an *acute goiter*, and one that induces marked dyspnea is designated a *suffocating goiter*. Syphilitic, tuberculous, and amyloid enlargements are extremely rare, but occasionally occur. Further, a goiter may be back of the sternum, that is, *substernal* or *retrosternal*. A very movable goiter, which is now above and now below the sternal notch, is called a *wandering* or *diver goiter*. A goiter within the thorax is called *intrathoracic;* and such a goiter may be *retrosternal*, *retrotracheal*, or *retro-esophageal*. When a number of persons in the same region are attacked with goiter the condition is frequently referred to as *epidemic goiter*. When the condition is common in a certain district it is called *endemic goiter*. When a person living in a district in which the disease is rare develops goiter, we speak

of the condition as *sporadic goiter*. It has long been known that *accessory* or *aberrant thyroids* exist. The term "aberrant" is better than "accessory" because in some reported cases the thyroid proper was absent (V. L. Schrager, in "Surg., Gynec., and Obstet.," Oct., 1906). Aberrant thyroids are masses of tissue composed of structure identical with the thyroid gland, and distinct and separate from the thyroid gland proper. Median aberrant thyroids are found about the hyoid bone and are formed from remnants of the thyroglossal duct. Lateral aberrant thyroids are found and develop from the remains of the lateral anlages of the thyroid (Ibid.). Aberrant thyroids vary in number: there may be none, one, several, or chains of them. An aberrant thyroid may enlarge with the thyroid, may not enlarge even though the thyroid does, or may enlarge when the thyroid proper remains normal. When cachexia strumipriva does not develop after complete thyroidectomy, including the parathyroids, the patient has been saved by enlargement and functionation of accessory thyroids.

**Causes of Goiter.**—It is known that goiter is extremely common in the valleys at the foot of certain mountain ranges in Switzerland, southeastern France, northern Italy, the Austrian Tyrol, and in the Himalayas and the Andes. In a portion of England it is so common that it is referred to as the Derbyshire neck. It seems evident that the disease is due to the introduction of some poisonous element into the system; but what this element is, is not known. Some writers maintain that individual liability is developed by habits of life; others think that susceptibility depends upon hygienic surroundings; and some attach great importance to hereditary influence. The probability is, however, that the disease is due to the existence of some poisonous substance in the drinking-water. Some observers have blamed snow-water; many have laid the cause of the trouble at the door of water impregnated with salts of lime; but the real cause has not been demonstrated.

Some observers believe that simple goiter is due to bacteria in the intestinal canal, an important function of the gland being to save the body from poisons which reach the blood from the intestines, an excess of poison or certain powerful toxins causing the gland to enlarge.

An ordinary parenchymatous goiter seems to be a species of hypertrophy. A number of years ago I suggested the view that the gland has undergone such an enlargement and has become distended with colloid material because the human body has demanded more of the secretion of the gland than the normal gland has been able to supply; as a consequence, the normal gland has enlarged its capacity and increased its output.

**Signs and Symptoms of Goiter.**—One may determine that a growth is in the thyroid gland or is connected with it by studying a number of facts. A goiter, as a rule, follows the movements of the larynx and the trachea during deglutition, and this sign may be obtained in the great majority of instances. There are, however, rare conditions, such as hyoid cyst, in which a movement of the mass takes place during the act of swallowing, although the thyroid gland is not involved. Then, again, a malignant or an inflammatory growth of the thyroid usually becomes anchored to the surrounding tissues and does not show this mobility. Certainly, however, in the great number of cases an enlarged thyroid moves with the larynx and the trachea during swallowing.

Goiters vary greatly in size. Cases in which the goiter was as large as an adult's head, and some cases in which the goiter hung in front of the breastbone, and reached to below the level of the ensiform cartilage, have been described. A very large goiter may have a stalk.

When the entire gland, as well as the isthmus, is enlarged, or when the isthmus alone is involved, the swelling may appear to be in the median line of the neck. If the condition begins in one lobe, the growth will, for a time at least, be distinctly one sided; though when such a growth has attained a

large size, it may displace the windpipe and come itself to the middle line of the neck.

A goiter of any considerable size pushes the sternocleidomastoid muscle externally and anteriorly, and the muscles that run from the sternum to the hyoid bone and to the thyroid cartilage overlie the front of the growth. The carotid artery is displaced externally and posteriorly. The relation of the jugular vein to the carotid artery is usually profoundly altered. The artery, as already stated, goes externally and posteriorly, while the vein is actually pulled anteriorly and is flattened out upon the side or the anterior surface of the goiter; hence the vein comes to lie to the inner side of the artery. This curious alteration in relationship is due to the fact that the common carotid artery has no branches, and therefore is pushed externally with ease; but the internal jugular vein receives branches that lie in the tumor, pull upon the vein, and prevent its displacement with the artery (Lücke).

Berry alludes to the fact that the tumor, unless it is very small, usually reaches the upper level of the sternum, and frequently passes below this level; and that only extremely large goiters hang in front of the sternum, but that it is not at all unusual for prolongations from a goiter to extend for quite a distance into the mediastinum. A substernal goiter is productive of very dangerous symptoms and offers many difficulties in diagnosis. A goiter will occasionally wander, now appearing in the neck and again disappearing behind the sternum.

Some goiters are said to pulsate. This takes place in exophthalmic goiter, the vessels of the goiter pulsating as do the other vessels of the body; but in the ordinary simple goiter what is called pulsation of the goiter is usually the transmitted pulsation from the carotid artery.

Some of the most important symptoms of goiter are due to pressure and to the displacement of anatomical structures. Pressure upon the veins at the root of the neck causes great enlargement of the veins above the goiter and in it. Pressure upon the recurrent laryngeal nerve may induce characteristic symptoms (spasm of the glottis or paralysis of a vocal cord), but the dyspnea of goiter is due to pressure upon the trachea and not to interference with the recurrent laryngeal. Paralysis of a vocal cord is rare in non-malignant, common in malignant, goiter. Pressure upon the cervical sympathetic may cause contraction of the pupil and narrowing of the palpebral fissure (Berry). Pressure upon the cervical plexus or the brachial plexus causes paresthesia, anesthesia, or paralysis in the parts supplied by nerves from the compressed plexus. Pressure upon the larynx and the trachea may cause very great displacement, and any such displacement is productive of marked dyspnea. This displacement is usually to the side; and it may cause such a flattening out of the tracheal rings that when the tumor is removed the trachea collapses and the patient perishes of suffocation.

A parenchymatous goiter usually begins insidiously and grows slowly. It occasionally ceases to grow for a considerable period of time, and may even shrink. It frequently enlarges temporarily during menstruation or pregnancy, and occasionally attains an enormous size by changing into the cystic form. Alterations in its consistency and outline may be due to the developing of adenomatous masses.

In making a diagnosis between the different forms of goiter, one should remember that a fairly symmetrical, bilateral growth is probably parenchymatous; if it was symmetrical from the start the probability of its being parenchymatous is enhanced; that sudden enlargements are produced by hemorrhage; that cyst formation may lead to very great enlargement, and possibly to fluctuation; that if a non-malignant goiter induces dyspnea, it almost invariably does so by pressing upon the larynx and the trachea, whereas a malignant

goiter may do so by interfering with the nerves of the part or by infiltration of the trachea; that a non-malignant goiter very rarely produces difficulty in swallowing, but that a malignant goiter frequently does so; and that cough often exists if there is pressure upon the larynx or the trachea, such a cough being metallic in nature and unassociated with impairment of the voice.

In any goiter there may be cerebral symptoms, such as anemia, syncope, or even convulsions. Rapidly growing goiters are often fatal, and slowly growing goiters are very rarely so. A malignant goiter grows with great rapidity, becomes adherent, infiltrates, and quickly produces metastases, and both sarcoma and carcinoma produce metastases by way of the venous system.

**Metastasis of Non-malignant Goiter.**—An ordinary goiter which presents no sign of being malignant may suddenly be disseminated. The deposits are apt to take place in the bones and in the lungs. Tumors have been removed without any thought of thyroid trouble being responsible, and examination has shown thyroid structure. Patel collected 18 cases of thyroid metastasis ("Tumeurs bénignes du corps thyroide donnant des métastases," "Rev. de Chir." No. 29, 1904). The bones most apt to receive metastases are the bones of the cranium, the lower jaw, the vertebræ, the pelvis, and the long bones. In 4 of these 18 cases the spine was affected. Dercum has reported a case of thyroid metastasis to the spine ("Jour. of Nervous and Mental Dis.," Mar., 1906). Colloid goiters are particularly prone to metastasis. Some surgeons maintain that if a metastatic deposit grows and destroys bone, the primary tumor should be regarded as malignant, no matter what histological studies indicate.

**Treatment of Goiter.**—Iodid of potassium and arsenic internally have been advised. An ointment of red iodid of mercury[1] locally is advocated by some writers. It should be rubbed in while the goiter is exposed to the direct rays of the sun. The administration of thyroid extract may do much good in a case of parenchymatous goiter, but it is useless in other forms of the disease. It should be associated with the local use of tincture of iodin or ointment of red iodid of mercury. McCarrison ("Proceedings of the Royal Society of Medicine," Feb., 1912) grew cultures from the feces of individuals with goiter. He prepared a mixed vaccine. He also isolated certain bacteria and prepared vaccines from them. Cases of goiter were treated with the vaccines. It is claimed the vaccines, especially the mixed vaccine, will cure recent parenchymatous goiter. The vaccine is said to act more promptly than thyroid extract. In times past it was customary to treat cystic goiters by aspiration and injection with a solution of iodin. Electrolysis has been used for soft goiter, the negative pole being pushed into the growth, the positive pole being applied to its surface. In many cases the *x*-rays prove of benefit. In considering the propriety of operation remember that a goiter which begins at puberty may pass away. We should operate on every non-malignant goiter which is increasing in size steadily or rapidly. Operation is justifiable even if there is not pressure because the mortality is very small, and it saves the patient from the possibility of malignant change, of hemorrhage, and of inflammation. I always used to operate under local anesthesia. There is less bleeding under local anesthesia than under ether. It is a great advantage to have the patient conscious, because by asking him to speak during the operation the surgeon can tell if the recurrent laryngeal nerve is being approached or touched. Two cases of violent hemorrhage in conscious patients taught me a lesson. Each patient was conscious that something was wrong, became terrified and uncontrollable, and greatly increased the danger and difficulty. I now give ether (by intratracheal insufflation) unless there is very high blood-pressure, serious tracheal obstruction, or disease of the heart, lungs, or kidneys (C. H. Mayo, "Illinois Med. Jour.," Feb.,

[1] This ointment consists of 1 part of red iodid of mercury to 28 parts of vehicle (white wax and almond oil).

1913). When ether is to be given I precede its administration twenty or thirty minutes by $\frac{1}{6}$ gr. of morphin and $\frac{1}{120}$ gr. of atropin, given hypodermatically. In some cases I follow Charles H. Mayo's plan and combine local anesthesia with light general anesthesia. In some cases intraglandular *enucleation* is performed, in other cases *extirpation*. Occasionally these two methods are combined (Bergeat). Some surgeons advise simple division of the isthmus. Ligation of the thyroid arteries has been recommended. *Exothyropexy* is the operation of exposing the thyroid gland, dislocating it through the wound, and leaving it in this situation. Exothyropexy is now almost never performed, on account of the safety of the operation of thyroidectomy. Atrophy of the gland follows exothyropexy. Enucleation, if possible, is the desirable operation. It may easily be employed for the removal of a single adenomatous, colloidal, or cystic area. *Thyroidectomy* (extirpation) is employed when enucleation is impossible. The entire thyroid is not removed for an innocent growth: at least a portion of a lobe is left behind, otherwise operative myxedema will probably arise. Unilateral extirpation (*lobectomy*) is the method usually chosen. In cancer and in some cases of sarcoma complete extirpation may be attempted. The operation in malignant disease will occasionally prolong life, but it will rarely effect a cure. In malignant disease tracheotomy may be rendered necessary by urgent dyspnea. The operation is often very difficult because the growth may cover the trachea, the trachea may be deviated a considerable distance from its proper position, and the veins are very large. After the performance of the operation it is usually impossible to use an ordinary tracheotomy tube, and in such a case König's long, flexible tube (Fig. 804) is employed.

Fig. 804.—König's tracheotomy tube.

**Endotracheal Goiter.**—Such a goiter may be due to misplaced thyroid tissue, or may be a prolongation from the thyroid gland. Occasionally the growth is situated in front; but, as a rule, it is attached to the posterior surface of the larynx or trachea. The first symptom is dyspnea, which increases and becomes very severe. An examination with a laryngoscope makes the condition evident. Endotracheal goiter is much more common in the female than in the male. It is believed to begin usually at about the age of puberty, though it may exist for a long time unnoticed. In some reported cases the patients were forty years of age before being seen. The proper treatment for an endotracheal goiter is the performance of a preliminary tracheotomy and then extirpation.

**Exophthalmic Goiter** (*Graves's Disease; Basedow's disease; Pulsating Goiter*).—This condition was first described by Graves, of Dublin, in 1835. It is a condition of chronic overactivity of the gland (hyperthyroidism) in which the secreting structure of the gland is increased in amount. The increase of parenchyma may perhaps be general, may perhaps be in separated areas (C. H. Mayo, "Illinois Med. Jour.," Feb., 1913). The symptoms are very variable, depending upon the amount and nature of the thyroid secretion and also upon the nervous tendencies of the victim. It is vastly more common in women than in men, and is most common between the ages of twenty and forty. It may arise at puberty. It has been stated that child-bearing has little influence in its causation, but I have seen the development of it in a woman three times in three different pregnancies. There is no proof of heredity, but it is not unusual to find more than one member of a family with it. It is not particularly prone to appear in ordinary goitrous families, although a person with an ordinary goiter

sometimes develops all the nervous symptoms and heart symptoms of Graves's disease (see page 1231, Basedowified Goiter). It may arise after emotional excitement or depression, fright, shock, hemorrhage, or an acute illness. It may develop during the existence of locomotor ataxia, paresis, epilepsy, neurasthenia, hysteria and other nervous troubles, and abdominal and pelvic diseases. A shock or fright does not cause hypersecretion. That existed before, but there were no symptoms because the body was neutralizing the poison. Shock develops symptoms by abolishing neutralization (C. H. Mayo, "Illinois Med. Jour.," Feb., 1913). Crile ("Am. Jour. Med. Sci.," Jan., 1913) believes that "Graves's disease is not a disease of a single organ or the result of some fleeting cause," that it originates in fear, and is excited by "some stimulating emotion intensely or repeatedly given, or some lowering of the threshold of the nerve receptors, thus establishing a pathological interaction between the brain and thyroid." Digestive toxemia is thought by many to be the underlying cause. It is frequently associated with marked anemia the result of excessive vomiting. The disease is a toxemia and the real cause of the symptoms is hypertrophy of the thyroid and excessive secretion of the gland (*hyperthyroidism*). This view is rendered more probable when we recall that a condition known as myxedema possesses many symptoms directly opposite to those of Graves's disease and that myxedema is due to deterioration, great diminution or absence of thyroid secretion, and the unopposed action of adrenal secretion. The administration of thyroid extract to an individual may produce some symptoms observed in exophthalmic goiter and partial thyroidectomy may improve or cure Graves's disease. Iodothyroglobulin is, perhaps, the poisonous element.

Fig. 805.—Exophthalmic goiter and total blindness from protrusion of eyes (Hansell).

An apparent objection to this view is that Graves's disease may exist without detectable thyroid enlargement, but this objection loses force when we recall that the thyroid may be somewhat enlarged, though we cannot detect the increase. It is probable in exophthalmic goiter that whether or not there is an excess of thyroid products passing into the circulation, toxic materials of some sort are formed in the gland and are taken into the lymph and blood. The real cause of exophthalmic goiter is not positively proved, but it seems probable that the disease is due to the action on the sympathetic system of large amounts of thyroid material, of some poisonous product of thyroid activity, or of some toxin the thyroid fails to destroy.

In exophthalmic goiter the vessels of the gland are not dilated—in fact, they are "usually smaller and less numerous than in a parenchymatous goiter of the corresponding size" (Berry, on "Diseases of the Thyroid Gland"). The surface of the gland is smooth. On section, the cut surfaces seem solid and

very little colloid is visible. The enlargement is due to growth of the glandular epithelium, either general or in localized areas, and this epithelial proliferation may be induced by different exciting causes.

In exophthalmic goiter the lymphatics within the lobules are usually obliterated, but the lymphatics around the lobules are present in increased number and are of exaggerated size. Sometimes the thyroid becomes fibrous, and in such cases myxedema is apt to arise. In a typical case there are rapid pulse or *tachycardia*, protrusion of the eyeballs or *exophthalmus* (due to a collection of fat back of each eye), and enlargement of the thyroid gland or *goiter*. Either thyroid enlargement or exophthalmus may be absent—in fact, in some rare cases both are absent. The pulse-rate in most cases is from 90 to 140. Exophthalmus is present in at least 80 per cent. of cases. The enlargement of the thyroid is bilateral. Unilateral enlargements are instances of Basedowified goiter—that is, are cases in which toxemia arises in the course of an ordinary goiter (see page 1231). A systolic bruit is usually audible over the thyroid region, and the large vessels at the root of the neck pulsate strongly because of arterial dilatation. The cardiac symptoms are of great importance. Acute cardiac dilatation occurs during tachycardia, and for a time, at least, disappears as tachycardia abates. Even trivial fatigue brings on temporary dilatation. Dilatation may become permanent (and does after one year), valvular insufficiency may arise, or cardiac hypertrophy may occur (Grocco, in "Riv. Crit. di Clin. Med.," Jan. 2, 1904). *Von Graefe's sign* may be present; this is inability of the lids to follow the eyes in looking down. *Stellwag's sign* is retraction of the upper lids. The lids in some cases cannot be completely closed, and when the eyeball is suddenly turned up, the lid and brow may fail to act together. *Moebius's sign* is inability to maintain the eyes in convergence. In some cases ocular palsies exist, in others there is photophobia or nystagmus. Patients may suffer from neuralgia, colic, choreic movements, tremor, flushes of heat, and gastric crises. Tremor is practically always present when the arms and forearms are extended, the palms of the hands are turned down, and the fingers are spread apart. Widespread tremor is apt to arise from any excitement, shock, or surprise. Dyspnea often exists and albuminuria and polyuria are not uncommon. Hemoptysis, hematemesis, or mental disturbance is sometimes noted. The patient is usually greatly depressed mentally, sometimes is excited, and may have outbreaks of violent hysterical excitement or even of mania. The usual expression is one of fright. There may be insomnia, elevated temperature, excessive sweating, or sudden attacks of diarrhea. All symptoms are increased by fear or fright. Exophthalmic goiter is sometimes associated with osteomalacia. This fact is important in connection with MacCallum's observations on the action of the parathyroids in controlling calcium metabolism. Kocher emphasizes the importance of the blood-picture. In nearly all cases there is lymphocytosis. Halsted ("Annals of Surgery," August, 1913) says of his cases: "Almost invariably the proportion of lymphocytes was increased, once being as high as 65 per cent. But in 1 case, the most serious of all, the total percentage of lymphocytes was only 9." Halsted regards enlargement of the thymus as probably responsible for the lymphocytosis, and the thymus is enlarged in at least 75 per cent. of marked cases. After operation lymphocytosis gradually diminishes (Halsted, Ibid.). The duration of a case is entirely uncertain. It is usually very chronic, with remissions or actual intermissions. Sometimes the patient gets entirely well, but this result is rare. There is often a partial cure, which may at any time be followed by a renewed outbreak. Sometimes the condition passes away rapidly, but abatement is usually gradual. Some cases get progressively worse and die. Certain cases are acute and these are apt to result fatally. A man in the Jefferson Hospital died in five weeks after the first symptoms were noted. He was de-

lirious for several weeks. Another died in four weeks in spite of ligation of the two superior thyroids. Very grave cases of exophthalmic goiter are probably often associated with disease of the thymus (Rehn). C. H. Mayo ("Illinois Med. Jour.," Feb., 1913) says: "While the large majority of cases can be easily diagnosed from the nervous symptoms, tachycardia, goiter, and eye symptoms, there are a few cases in which it is difficult to diagnose true hyperthyroidism from pure neurasthenia, myocarditis, or Bright's disease, as well as a few cases in which there may be a complication by affection of the hypophysis, thymus, or adrenals."

**Treatment.**—Thyroid extract does harm. Medical treatment in a severe case should comprise rest in bed, the use of an ice-bag over the heart, and the administration of adrenalin. When the patient gets about again, he must avoid alcohol and all forms of excitement. Gentle exercise is desirable, but never violent exercise. Diet is to be nutritious, but non-stimulating. Electricity is said to be of benefit. Experiments in organotherapy are being tried in this disease. Thymus extract has been used. Ballet and Enriquez assumed that the thyroid gland furnishes an antitoxin to certain body poisons, that an excess of thyroid secretion over the amount required to neutralize toxin causes the condition known as Graves's disease, and that the symptoms of Graves's disease should disappear if sufficient toxin is administered to antidote the excess of thyroid secretion (Hubert Richardson, in "Am. Medicine," August, 1906). The two observers mentioned above obtained blood-serum from thyroidectomized dogs and injected it into individuals suffering from Graves's disease and claim that they noted improvement. In 2 of their patients, however, tetany developed. Lanz has used the milk of thyroidectomized goats instead of the serum of thyroidectomized dogs. The serum of thyroidectomized sheep, powder made from the dried goiter of a cretin, and the powdered flesh of thyroidectomized sheep have been used (Hubert Richardson, Ibid.). What is known as *thyroidectin* is the dried serum of an animal from which the thyroid gland has been removed. John W. Rogers and S. P. Beebe have made some extremely interesting studies on the production and application of a serum. Rogers makes two sera, using one or the other, according to the needs of the case. One serum, called the normal serum, is obtained from sheep or rabbits after injecting them with the combined nucleoproteins and thyroglobulin of healthy thyroids; the other, called the pathological serum, is obtained from the animals after injecting them with combined nucleoproteins and thyroglobulin obtained from the thyroids of Graves's disease. In 1 severe case I have seen rapid improvement and apparent cure follow the use of Rogers's serum. The value of serum treatment is as yet undetermined. It is certainly not free from danger and some deaths have followed its use One cause of diverse results after the use of goat serum may be found in the fact that some of the animals were probably incompletely thyroidectomized. The goat possesses aberrant thyroids and these must be removed as well as the gland proper.

There are upon the market three preparations to combat hyperthyroidism:

1. Thyroidectin (or thyreoidectin) is a powder made from the dried blood-serum of thyroidectomized animals. It is given in 5-gr. capsules. The dose is 1 or 2 capsules three times a day.

2. Beebe's serum is the serum of thyroidectomized animals.

3. The antithyroidin of Moebius is the serum of sheep's blood, the animal having had its thyroid gland removed at least six weeks before the serum was obtained.

Bilateral extirpation of the cervical ganglia of the sympathetic and division of each nerve below the ganglion has been employed, it is alleged, with benefit (Jaboulay). I have not employed the operation for this disease. Ligation of the thyroid arteries may do good. Its chief use is preliminary to thy-

roidectomy. Partial thyroidectomy is the operation commonly employed in severe cases; it cures within six months from 50 to 75 per cent. of the cases operated upon. One lobe, the isthmus, and a portion of the other lobe are removed. Some cases do not improve; others improve slowly and relief is only partial. It is the operation which I prefer. The Mayos have obtained a splendid series of results from this operation. It is their custom to apply the *x*-rays daily for several weeks and then to operate. The rays produce decided but temporary improvement. The operation is intracapsular extirpation of one lobe. Ether is given to most cases (by intratracheal insufflation). In some cases thyroid intoxication follows operation. In other cases very rapid growth follows incomplete removal, and the operation seems actually to have done harm. Sudden death occasionally follows the operation. The removal of an exophthalmic goiter is difficult; the capsule and blood-vessels rupture from slight force. All cases should not be operated upon. (See Operation for Exophthalmic Goiter on page 1243.) The *x*-rays frequently have a very beneficial effect upon the symptoms of exophthalmic goiter. They probably destroy some of the secreting glandular epithelium, and thus diminish the amount of the thyroid secretion and alter its character. They also induce fibroid changes, and 80 per cent. of cases are relieved. Frequently they cause great improvement, occasionally they will produce a cure. It is my custom to use the rays preliminary to operation in order to decrease the vascularity of the part, to lessen the amount and diminish the toxic quality of the thyroid secretion, and to modify the various symptoms.

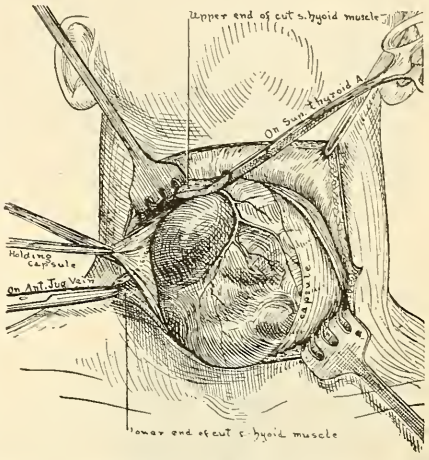



Fig. 806.—Enucleation of cystic goiter; capsule opened (C. H. Mayo).

**Operations on the Thyroid Gland.**—The removal of a goiter is a major operation and one beset with difficulties and dangers. Nevertheless it is a very successful operation. I lost a case of supposed adenomatous goiter which turned out to be a metastatic hypernephroma. The patient died from secondary hemorrhage. I lost a case of carcinoma of the thyroid and a case of simple adenoma. My mortality in ordinary goiter has been under 2 per cent. In 1200 cases of ordinary goiter the Mayos had a mortality of 1 per cent. following extirpation. Kocher's mortality is 0.4 per cent. Certain anatomical points are to be borne in mind. The internal jugular vein is frequently found to the inner side of the carotid artery and lying on the goiter (because the vein has branches which run to the goiter and hold the vessel against the thyroid). The recurrent laryngeal nerve ascends along the side of the trachea back of the gland, and is against the esophagus before it passes through the cricothyroid membrane. On the right side the nerve is very close to the inferior thyroid artery, sometimes passing over it, sometimes under it. On the left side it is deeper and not so near the artery. The parathyroids are behind the thyroid and usually behind the capsule.

In view of the fact that one or both recurrent laryngeal nerves may be paretic from pressure, all cases should be examined with the laryngeal mirror before the operation to determine this point. If the paresis is on one side only, the sound vocal cord may possibly compensate by advancing across the

midline. In such conditions the paresis may be first observed following operation, leading the operator to think that he has injured a nerve (C. H. Mayo, in "Surg., Gyn., and Obst.," July, 1907).

*Intraglandular Enucleation* (*Socin's Operation*).—By this operation an adenoma or cyst of the thyroid gland is removed, the encompassing glandular tissue being left in place. The capsule of such a growth is glandular tissue. The operation of enucleation is not suited to the removal of multiple tumors and it cannot be performed for parenchymatous goiter or exophthalmic goiter. Intraglandular enucleation is performed as follows: The thyroid is exposed by an oblique or by a transverse incision. An incision is made through the capsule of the thyroid gland and through the glandular tissue until the cyst or solid tumor is reached. As a rule, the tumor can be recognized by the fact that its color differs from the color of the thyroid tissue. The tumor is turned out by the fingers, a special scoop, the knife handle, or a dry dissector. In some cases a cyst can be most easily removed if, after exposure, it is incised and emptied and its wall is then grasped by strong forceps. A solid tumor should, if possible, be removed intact. The wound is packed temporarily with gauze, the edges of the cavity are grasped by forceps, the gauze is

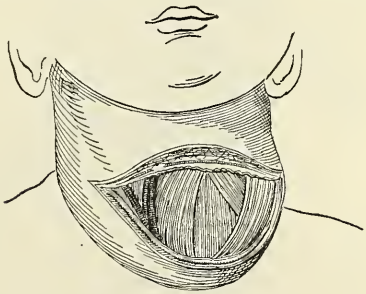


Fig. 807.—Kocher's transverse incision exposing the muscles and median veins of the neck (Kocher).

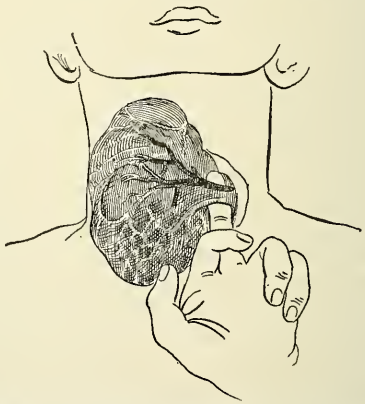


Fig. 808.—Isolating the accessory veins (Kocher).

removed, and every bleeding point is carefully ligated. The wound is closed by three layers of sutures—"one in the gland, one in the muscles, and a third in the skin" (James Berry, on "Diseases of the Thyroid Gland"). If the tumor is large, drain for twenty-four hours; otherwise, do not drain.

Enucleation is a very successful operation if performed upon properly selected cases, and can be performed rapidly, but the arrest of bleeding is often tedious and troublesome.

*Extracapsular Extirpation.*—This term means removal of the entire gland (*complete thyroidectomy*) or a portion of the gland (*partial thyroidectomy*) with the glandular capsule, the operation being an extracapsular procedure. Usually but one lobe is extirpated (*lobectomy*), or one lobe, the isthmus, and a portion of the other lobe. This method enables the operator to tie the chief vessels before he cuts them; as his vision is not obscured by bleeding, he can avoid cutting the glandular capsule (which would provoke copious bleeding), and he keeps a safe distance away from the recurrent laryngeal nerve.

If the patient suffers from grave respiratory trouble with myocardial disease, a general anesthetic is contra-indicated. If ether is used, after unconsciousness is obtained the ether is given by intratracheal insufflation. The patient

is placed recumbent, with the shoulders a little raised and the neck laid upon a sand-pillow so as to throw the head back as far as is consistent with comfortable respiration.

An oblique incision, a horseshoe-shaped incision, or a transverse collar incision (Fig. 807) may be made. I usually employ an incision shaped like an incomplete horseshoe, the convexity being downward. Layer by layer the tissues are divided. Vessels are carefully tied as divided or before division. The muscles which run from the sternum to the hyoid bone may in some cases be separated, but the extirpation of a large goiter requires transverse division

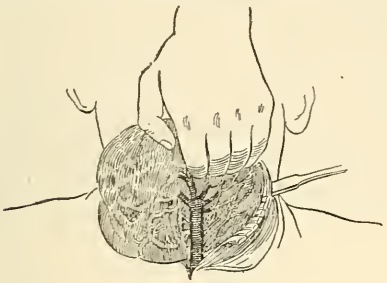


Fig. 809.—Exposure of veins at lower end before ligation (Kocher).

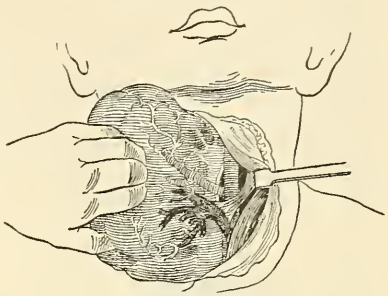


Fig. 810.—Dislocation of the goiter toward the right (Kocher).

of the muscles high up. The capsule of the lobe is exposed, and is separated from external parts (Figs. 808, 809, and 810). The upper portion of the gland is cleared. The superior thyroid vessels are found, tied with two ligatures each, and divided between the ligatures (Fig. 811). The clearing of the gland is carried on toward the median line and some rather large veins are encountered and tied (Fig. 813). The lower portion of the lobe is cleared and the inferior thyroid vessels are found. Near this point the recurrent laryngeal

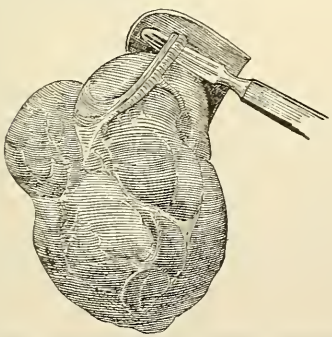


Fig. 811.—Isolation of the superior thyroid artery and vein (Kocher).

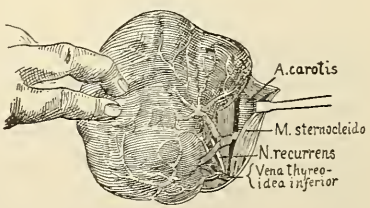



Fig. 812.—Ligation of the inferior thyroid artery (Kocher).

nerve lies and may be located. If the operation is being done under a local anesthetic adjacency to the nerve is readily determined, because if the nerve is pulled upon, or if it is pressed upon or touched by a blunt instrument, the patient's voice becomes metallic. A deliberate attempt is made to locate the nerve, and the patient is engaged in a conversation requiring answers while the surgeon is investigating. The lobe is lifted from its bed and dislocated from the wound and the inferior thyroid vessels are tied close to the border of the gland in order to avoid the recurrent laryngeal nerve (Fig. 812). The vessels are tied and

cut across as were the superior thyroid vessels. The isthmus is next exposed, clamped, ligated, and cut across, every care being taken to prevent colloid from being squeezed into the wound (Fig. 814). After dividing the isthmus, any bleeding point is ligated and the stump is cauterized. The divided muscles are sutured with catgut, a drainage-tube is inserted, and the superficial wound is closed with sutures of silkworm-gut.

*Intracapsular Extirpation.*—This operation is warmly advocated by the Mayos. The preservation of the posterior portion of the capsule protects the recurrent laryngeal nerve and greatly lessens the risk of injuring the parathyroids. Ether is given unless there is grave respiratory difficulty with myocardial degeneration, and half an hour before administering the ether the patient is given a hypodermatic injection of $\frac{1}{4}$ gr. of morphin and $\frac{1}{100}$ gr. of atropin. The ether is continued by intratracheal insufflation. When anesthetized, the patient is placed in the reversed Trendelenburg position and the shoulders are elevated (C. H. Mayo, in "Surg., Gyn., and Obst.," June, 1907). Kocher's transverse collar incision is made. Muscles are separated or divided as in ordinary extraglandular extirpation. If the ribbon muscles are divided the cut is made near their upper insertion to save their nerve supply and prevent the muscle scar from

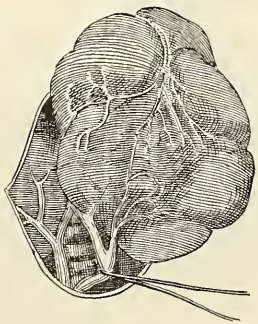


Fig. 813.—Isolation of the venæ thyreoideæ imæ (Kocher).

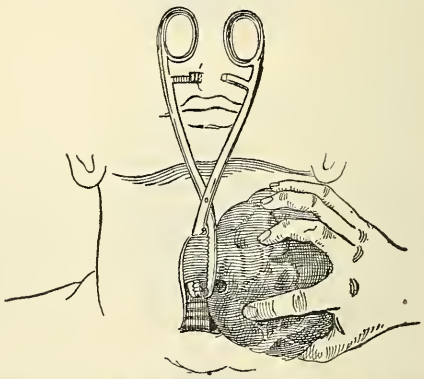


Fig. 814.—Isolation and clamping of the isthmus (Kocher).

being in line with the skin scar. The gland is elevated. The vessels entering and leaving the raised lobe are double clamped and tied. The capsule is incised along the outer side of the gland, is pushed back with gauze, and the lobe is drawn toward the midline, and vessels are caught by forceps, the tissues being grasped in line with the midline of the body (C. H. Mayo, Ibid.).

Every structure bearing any resemblance to a parathyroid is allowed to remain. The isthmus is clamped, divided, and closed by suture. The muscles and skin are sutured. Drainage is required after removal of large growths, a separate incision being made to permit of it.

**Dangers in Goiter Operations.**—During any operation for goiter sudden death may occur. In some cases a general anesthetic is responsible. In others suffocation arises from pressure upon or bending of the trachea or collapse of the trachea as the goiter is lifted from its bed. In rare cases dangerous dyspnea arises from irritation of the laryngeal nerves, and cardiac inhibition may be induced in the same manner. The parathyroids may be injured or removed and tetany may result. Rough handling and flooding the wound with colloid may be followed by great and even fatal hyperthyroidism. The trachea or esophagus may be opened. The recurrent laryngeal nerve may be injured. Air embolism seldom occurs. Reactionary or secondary hemorrhage is usually due to slipping of the ligature on the superior

thyroid artery "caused by including a piece of muscle" (C. H. Mayo, in "Surg., Gyn., and Obstet.," June, 1907).

**Acute Thyroidism (Hyperthyroidism).**—When colloid from the thyroid is squeezed into the wound during the operation or leaks into it later, it is absorbed and may produce serious symptoms or even death. This is most apt to happen in exophthalmic goiter. The symptoms always appear within forty-eight hours and usually within twenty-four. Sometimes they arise quickly after operation. In some cases in which this happens the patient never reacts from the operative shock, but develops a very rapid pulse and intense dyspnea, and dies in a few hours. In less severe cases there is a period of circulatory excitement, dyspnea, and elevated temperature (*thyroid fever*). The surgeon seeks to prevent acute thyroidism by limiting leaking of colloid, by cauterizing the stump, by washing the wound with adrenalin solution, suturing the capsule over the raw stump of the gland, and inserting drainage.

**The Operation for Exophthalmic Goiter.**—The operation of thyroidectomy is not to be performed during an acute exacerbation. To do it then will very probably cause death. Delirium is a contra-indication. So are gastric crises. The operation is very dangerous when there is great cardiac dilatation. After the disease has existed one year it is highly probable that marked dilatation exists.[1] Hence, early operations are much safer than late ones. If a case is seen early, and if the *x*-rays (and perhaps serums) fail to cure, operation should be recommended. In any case with serious symptoms the surgeon seeks to modify those symptoms before thyroidectomy by means of rest, the ice-bag over the heart, drugs, the *x*-rays, and perhaps serums. If under this treatment the dangerous symptoms disappear or greatly abate, thyroidectomy may be performed.

If the dangerous symptoms are little modified or not modified at all by medical treatment, preliminary ligation of one of the thyroid arteries is indicated. Ligation of an artery will not cure a case, but will probably greatly improve it. The operation is strictly palliative and preliminary. Tie the right superior thyroid. Wait one week. If the symptoms are not greatly improved, tie the left superior thyroid and wait a while. In severe cases tie both superior thyroids at one sitting. The operation is done under infiltration anesthesia. It is not entirely free from damage and may cause death. Halsted ("Annals of Surgery," August, 1913) ties one inferior thyroid or both inferior thyroids. He says that by the inferior operation we obtain a better cosmetic effect, leave a scar largely beyond the incision which will subsequently be used for lobectomy, and tie vessels which are larger than the superior arteries. In 1 of his cases four arteries were tied in 4 operations before lobectomy. C. H. Mayo[2] points out that the ligature of the superior thyroid should be applied very close to the pole or should actually include some of the thyroid tissue, "so that a reversed circulation in anastomotic branches with the inferior thyroid artery may not occur."

When it is esteemed safe the surgeon prepares to perform partial thyroidectomy. In most cases ether is given, preceded by a hypodermatic injection of morphin and atropin. After the patient is anesthetized by ether the tracheal tube should be introduced and the unconsciousness be maintained by insufflation anesthesia. If there is grave cardiac dilatation and we determine to operate, infiltration anesthesia is relied upon.

At the operation an entire lobe (except a thin slice), the isthmus, and a portion of the other lobe should be removed. The more of the gland removed, the better the prospect of cure (Halsted, "Annals of Surgery," August, 1913).

[1] C. H. Mayo, in "Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912; Halsted, "Annals of Surgery," August, 1913.

[2] "Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, August, 1912.

At least one-half of one lobe must be left in order to prevent myxedema. The parathyroids are protected by leaving a small slice of the posterior portion of each lobe (Halsted, "Annals of Surg.," Aug., 1913). After the removal of one lobe the other usually undergoes considerable atrophy. After thyroidectomy, lymphocytosis gradually decreases, and an enlarged thymus generally disappears (Ibid.). Thyroidectomy may cure the case; it may greatly improve it; may fail or nearly fail, or may cause death. Cure is gradual and may be attained only after several months. From 50 to 75 per cent. of patients are cured by the operation. Many others are vastly and permanently improved. Some are improved, but are subject to temporary relapses. In Halsted's 39 cases there was not a death from operation.

In the first 16 cases operated upon by the Mayos the mortality was 25 per cent. At present the factors which forbid operation are recognized. In 1355 operations for exophthalmic goiter their mortality was under 4 per cent. They have had 278 consecutive cases without a death, a truly wonderful record.[1]

The elder Kocher ("Jour. Am. Med. Assoc.," April, 1910) reports upon 535 partial thyroidectomies for exophthalmic goiter. The mortality was 3.1 per cent: 3 died from the anesthetic, and now he uses local anesthesia preceded by Crile's plan of psychic narcosis; 3 others died from kidney disease which existed at the time of operation. Now he will not operate if kidney disease exists. There were 3 thymus deaths; 5 with status lymphaticus died suddenly while being prepared for operation. Such patients should not be operated upon unless they are greatly improved by medical treatment. Kocher, like Halsted, says that the degree of improvement depends upon the amount of gland removed. In such cases use medical treatment and the *x*-rays first. If this plan fails, tie one or more of the thyroid arteries before doing thyroidectomy.

My personal experience in exophthalmic goiter is small; in fact, I know of no one in Philadelphia who has performed any great number of operations for it. I have performed thyroidectomy for exophthalmic goiter 14 times, with 2 deaths.

**The Parathyroid Glands and Tetany.**—These glands were discovered by Sandström in 1880, and their vital functions were pointed out by Gley. He showed that removal of the parathyroids causes tetany.

The parathyroids are brownish red and are larger in adults than in infants. They are constant in man, never being congenitally absent.

They are usually four in number and are ordinarily placed external to the thyroid capsule. In some cases, however, one or more of them may be found embedded in the thyroid gland, but even when they appear to lie within the thyroid they are always separated from it by a capsule of connective tissue.

While there are usually four parathyroids, there may be only three, or there may be six, seven, or eight. Accessory parathyroids may be found over wide areas. One was discovered by Rogers and Ferguson in the middle of the posterior portion of the pharynx; and there was found in the thorax by Ogle a gland that was partly parathyroid.

From their situation the parathyroids are divided into superior, or external, and inferior, or internal. Walsh describes these glands in adults as being each from 6 to 7 mm. in length, 3 to 4 mm. in breadth, and 1½ to 1 mm. in thickness. Each of these glands is supplied by a terminal artery, and the arterial supply is very largely obtained from the inferior thyroid artery or from the branch of anastomosis between the superior and inferior thyroid vessels.

If the parathyroid glands are extirpated from an animal, tetany usually develops (*experimental tetany*). This is positively the case in dogs. These glands certainly have most important functions in the metabolism of the body.

[1] C. H. Mayo, in "Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912.

They are essential for normal metabolism (Mayo and McGrath, in "Annals of Surgery," Feb., 1912). MacCallum and Voegtlin ("Johns Hopkins Hosp. Bull.," March, 1908) believe that damage or removal of them causes great changes in calcium metabolism.

Very little is known of diseases of the parathyroid glands. A few cases of tumor have been reported, each being an example of either work-hypertrophy or adenoma. One of these cases was reported by the author in "Surg., Gyn., and Obst.," Jan., 1909. In this case the tumor was removed under the impression that it was an adenoma of the right lobe of the thyroid gland. The pathological report, however, showed it to be a parathyroid. No trouble of any kind followed the operation, though at a later period enlargement of the opposite side of the neck occurred. This has been let alone through fear that it may be a left parathyroid. In spontaneous tetany histological changes have been found in the parathyroids and there is some evidence that traumatism may produce functional insufficiency.

Some persons have maintained that deficiency of parathyroid secretion is the cause of paralysis agitans, but this idea has been warmly combated and lacks evidence. Some observers believe that there is a deficiency of parathyroid secretion in exophthalmic goiter.

In view of the well-known fact that removal of or damage to the parathyroids may result in tetany (*hypoparathyroidism*), provided that several of these glands or all of them are damaged or removed, it becomes the duty of the surgeon, when operating for goiter, to exercise the utmost care that he does not remove any of these bodies. The safest way to avoid them is to retain the posterior portion of the capsule of the thyroid gland. If, during an operation, any small body that resembles a parathyroid is detected, it should be let alone; or if such a body has been accidentally removed, it should immediately be implanted into the capsule of the lobe of the thyroid gland that has been left undisturbed. It is highly improbable that the removal of even both parathyroids on one side will cause tetany if there are two normal parathyroids on the opposite side.

On account of the possibility of the development of tetany the question of the transplantation of parathyroids from animals becomes extremely important. Halsted has made some very valuable experiments upon the auto- and isotransplantation of the parathyroid glands in dogs ("Annals of Surgery," Oct., 1907; "Jour. of Experimental Med.," vol. xi, No. 1, 1909). He has shown that if the parathyroid be transplanted into an animal with normal parathyroids failure will follow, and the transplanted glandule will disappear. If, however, there is parathyroid deficiency in the animal into which the transplantation is made, the glandule will attach itself and grow. He obtained the best results by placing the parathyroid beneath the posterior sheath of the rectus muscle of the abdomen. Some observers have placed it in the spleen; others, within the peritoneum and in various other regions.

One of the dogs experimented upon by Halsted and reported upon in "Jour. of Exper. Med.," vol. xi, No. 1, 1909, lived in perfect health "for fifteen months and was in good health until the performance of the final operation, at which a parathyroid autograft" was removed. The dog died in three months from hypoparathyroidism (Ibid., No. 3, 1912).

If tetany follows thyroidectomy, it may be treated by the intravenous, rectal, or stomach administration of a 5 per cent. solution of lactate of calcium (MacCallum and Voegtlin, Loc. cit.). Beebe and Berkeley have prepared a parathyroid serum which they claim is efficient. Charles H. Mayo maintains that in tetany either the serum or the calcium lactate should be used, in the hope of tiding the patient over until parathyroid glands can be secured and implanted ("Annals of Surgery," July, 1909). The calcium salt may be given

intravenously, subcutaneously, by the stomach, or by the rectum. Joseph H. Branham ("Annals of Surg.," August, 1908) has reported tetany following thyroidectomy cured by the subcutaneous injection of parathyroid emulsion. The emulsion was made by grinding up fresh glands of beeves in a mortar, and then pouring 400 c.c. of sterile salt solution into the mortar. The preparation was filtered through sterile gauze and was administered beneath the patient's breast.

When tetany begins to develop in a patient there are usually headache, dizziness, and pain in the extremities. The muscles in one or both forearms and hands are liable to be affected. The hand flexes at the wrist, while the fingers are extended, and in some cases the forearm flexes at the elbow. Now and then the fingers will flex at the metacarpophalangeal joints, but the distal phalanges will remain extended. These spasms are painful. Similar spasms may occur in the feet and toes. In most severe cases the trunk muscles and those of the chest, throat, and eye may be involved.

Trousseau, years ago, showed that a spasm may be brought on in the affected limb by pressing upon the nerve-trunks and blood-vessels; this is called the *Trousseau sign.* Pressure upon the facial nerve may induce the spasms, and it is called *Chvostek's sign. Erb's sign* is a great increase in the galvanic irritability of the motor nerves. *Hoffmann's sign* is excessives sensitiveness of the sensory nerves. *Schultze's tongue sign* ("Münch. med. Woch.," Oct. 31, 1911) is said to be invariably present in adults with tetany. If the side of the tongue is tapped the organ responds by grooving itself on that side. If the tongue is lifted by a spatula and the dorsum is tapped a constriction like a waist forms.

Tetany due to absence of the parathyroids will inevitably prove fatal if not treated actively.

Before we understood the necessity of guarding the parathyroids tetany after goiter operations was not uncommon. The preservation of the posterior portion of the capsule has almost abolished this peril. In 3203 operations on the thyroid performed at the Mayo Clinic in St. Mary's Hospital, Rochester (up to Dec. 1, 1911), there was but 1 case of tetany and the symptoms in that were slight and transitory.

## XXXIII. THE CAROTID GLAND; THE THYMUS GLAND

**The Carotid Body or Gland.**—This structure was discovered by Haller in 1743. It has no known function. It is about the size of a grain of rice and lies in or very near the carotid bifurcation, being adherent usually to the common carotid, occasionally to the external carotid. Tumors may form in this little body. The type found is the perithelioma. Such a tumor grows slowly, is hard, is movable from side to side, but not up and down, and is lifted with each beat of the carotid artery. In the long run it tends to become malignant. As the tumor grows it comes to surround the common carotid artery.

**Treatment.**—Operation on an advanced case is very difficult, will probably require ligation of the common carotid, and cause grave injury to important nerves. In one of the author's patients the common carotid was tied and the patient developed laryngeal palsy and hemiplegia. Early operation may permit of removal of the tumor without tying the common carotid artery. In a second case the author was able to accomplish this. My first case led me to agree with Reclus that these tumors should be let alone unless they are productive of dangerous symptoms. I have come to the conclusion that the time to operate is early, long before there are dangerous symptoms. To wait for obvious malignancy is to court failure or disaster.

**The Thymus Gland.**—This bilobed ductless gland becomes fully developed toward the end of the second year of life. From that period it remains stationary for a time and then undergoes retrogression. At puberty retrogression becomes rapid. Finally, the gland almost entirely disappears, although a small vestige of it usually remains through life. Studied in the very young child, it is found to consist of a thoracic portion and a cervical portion. The chief part of the gland lies directly back of the sternum and upper four costal cartilages of each side in the superior and anterior mediastinal spaces. On each side are the pleura and lung. Posteriorly it lies upon the pericardium, superior vena cava, innominate vein, and pulmonary artery. The two lobes from the gland rise into the neck in front of the trachea. The left lobe overlaps the common carotid; the right lobe, the innominate artery. A strand of fibrous tissue usually passes from the lobe which ascends highest to the corresponding lobe of the thyroid gland. The lobes of the thymus receive numerous veins from the thyroids. The inferior thyroid artery is the chief source of arterial blood. The thymus is composed of many lobules separated by septa. The undegenerated thymus contains both lymphoid and epithelial structure.

The cells of the reticulum are epithelial. In the medulla of the lobules are the concentric corpuscles of Hassall which are derived from the epithelium of the reticulum. The small thymic cells are probably lymphocytes. In Hammar's words: "The thymus is an epithelial organ which is permeated with lymphocytes" (quoted by Charles H. Mayo and Bernard F. McGrath in their thorough and impressive study of "The Surgical Importance of the Thymus." See "Collected Papers by the Staff of St. Mary's Hospital," Mayo Clinic, 1912).

The function of the thymus is as yet undetermined. Some believe it is merely a lymphatic structure and furnishes no internal secretion. Others believe that it furnishes an internal secretion of great, perhaps of essential, importance in development. Some observers believe that the thymus destroys toxins, and that deficiency of thymus permits the accumulation of toxic matter in the system. These studies of hypothymization were made on thymectomized animals.

Experiments to determine the result of excessive thymus function (hyperthymization) are peculiarly contradictory, unsatisfactory, and inconclusive. In these experiments thymus glands have been implanted, thymus juice has been injected, and thymus feeding has been practised.

Mayo and McGrath (Ibid.) conclude "that the thymic function is concerned in the general process of nutrition, particularly with the ossification of bone"; further, it is probable "that the thymus and the chromaffin portion of the adrenals act antagonistically on the sympathetic."

Although the thymus should have practically disappeared between the twelfth and twentieth year, there are cases in which it fails to disappear at all. There may never have been any symptoms to suggest persistence and the gland may be discovered at a necropsy.

The thymus gland may enlarge. The enlargement is most usual in childhood, but if the thymus is persistent, may occur much later in life. The condition is hypertrophy or hyperplasia. Enlargement of the gland may be met with in exophthalmic goiter, leukemia, Hodgkin's disease, and status lymphaticus. It may produce no symptoms whatever. It may be responsible for asthma and for sudden death (thymic asthma and thymic death). Enlargement is recognized by the *x*-rays.

Infectious and wasting diseases in children may cause atrophy of the thymus. The gland may be the seat of hemorrhages, inflammation, tuberculosis, calculi formation, and necrosis (in diphtheria). In congenital syphilis the thymus may be the seat of gummata or fibrosis.

Dubois's abscesses occur in syphilis of the thymus. Many spaces form and the spaces are full of a fluid resembling pus or actually being pus.

Enlargement by tumors may occur. Innocent tumors (fibroma, myxoma, and adenoma) are rare. Malignant tumors (carcinoma and sarcoma) are more common. The most common growths are small-celled sarcomata.

Tumors of the thymus cause the symptoms of mediastinal tumor (cyanosis, dyspnea, exophthalmus, cough, alterations in voice, circulatory disturbance).

**Status Lymphaticus** (*Status Thymicus; Lymphatism*).—This condition is associated with enlargement of the thymus. (See page 225.)

**Operations for Thymus Enlargement or Tumor.**—(See Oliver, in "Archiv. gén. d. Chir.," vi, 138, 1912.)

To perform *exothymopexy* expose the thymus by an incision above the sternum, catch the gland and capsule with forceps, draw them up as far as possible, and suture to the fascia. This operation is without much value. A decompression operation can be done by resecting the manubrium. It does not cure, but may give relief.

Thymectomy has been performed a number of times. It may be extracapsular or intracapsular, partial or complete. In 42 reported cases there were 15 deaths.

## XXXIV. DISEASES AND INJURIES OF THE LYMPHATICS

**Wounds, Ruptures, and Occlusions of the Left Thoracic Duct.**—It was long believed that wounds of any part of the thoracic duct were almost certainly fatal. It is now known that wounds of the duct at the root of the neck are rarely very dangerous unless the duct is divided close to the vein. A wound of the duct is rarely seen as the result of an accident because the adjacent vital structures are apt to be injured at the same time and death rapidly ensues. Wounds of the duct or of its large branches occasionally, but very rarely, are inflicted during surgical operations. Benetau speaks of 12 cases thus inflicted; in 8 cases the operation was for tuberculous glands, in 3 for malignant glands, and in 1 for ligation of the subclavian artery. One alleged danger of wound of the duct is entrance of air into the adjacent vein. This is said to have happened in 1 case. As a rule, the short end of the cut duct does not bleed, the duct valves preventing hemorrhage. In Fullerton's case, when a grooved director was passed along the stump of the duct by way of a terminal into the vein, blood at once appeared. In most cases the injury is not recognized at the time, but later, when white fluid escapes from the wound. The discharge may continue or may cease spontaneously. If it continues, there is rapid loss of flesh and strength. I assisted Dr. Keen in the case in which he did recognize the wound at the time it was inflicted. A thin fluid was observed flowing rhythmically from a tear in the duct. It is to be remembered that the course of the cervical part of the duct is very variable and sometimes the duct lies very high above the clavicle. There was 1 death in 17 recorded cases (Dudley P. Allen and C. E. Briggs, in "Amer. Med.," Sept. 21, 1901).

The discharge from a cut duct may continue to leak—perhaps a pint or more flowing out during twenty-four hours. If leakage continues, constitutional effects will sooner or later become evident. In Schoff's case ("Wien. klin Woch.," Nov. 28, 1901) it was not known that the duct had been injured until the stitches were removed from the wound in the neck. The wound was found distended with chyle and Schoff packed it with iodoform gauze. Fifteen days later the patient died from chylothorax and pulmonary compression.

Rupture of the thoracic duct or of the receptaculum chyli may occur from traumatism or be a secondary consequence of tuberculosis or carcinoma.

Rupture leads to death by starvation, or to fatal compression by the exuded fluid (Harvey W. Cushing, in "Annals of Surgery," June, 1898). Occlusion of the main duct may be followed by rupture of the receptaculum chyli. Gradual occlusion by a tuberculous or inflammatory growth may not produce any serious symptoms. Cushing assumes that in such a case the lymph-current is reversed and is taken up by the right thoracic duct. In gradual obstruction masses of dilated lymph-vessels may be found, particularly in the thorax and abdomen. If lymph-vessels rupture, chyle flows out and, according to the situation, there arises "chylous ascites, chylothorax, chyluria, or chylous diarrhea" (Ibid.).

**Treatment of Wounds.**—If the wound in the neck does not completely divide the duct, and if the duct wound is discovered at the time of operation, suture the duct. Allen sutured the duct and had no further leakage. Keen sutured the duct and recovery followed. If the duct is completely divided, follow Cushing's advice: "It would seem advisable to place a provisional ligature about the duct on the proximal side of the wound, and to control the leakage, if possible, by a gauze tampon. This would act as a safety-valve, and allow chyle to escape, if the pressure in the duct became too great and there was difficulty in establishing a collateral lymphatic circulation. The patient meanwhile should be given a meager diet. If the leakage should become uncontrollable and threaten starvation, the provisional ligature should be tied, with the hope of a final readjustment of collateral circulation or trusting in the presence of some anomalous anastomotic branch which might suffice to carry the lymph into the venous circulation" (Ibid.). Fullerton tied both ends of a divided duct and the patient recovered ("Brit. Med. Jour.," June 16, 1906). Deanesley ("Lancet," Dec. 26, 1903) inserted the divided duct into the internal jugular vein and sutured it in place. There was some leakage, but recovery ensued. After ligation the duct on the proximal side of the ligature may distend greatly and may actually rupture. When a wounded duct is leaking the patient should be fed exclusively on proteins. The diet should be scanty and the patient must be kept absolutely quiet in order to keep pressure in the duct at as low a level as possible during the establishment of a collateral lymphatic circulation (Fullerton, Loc. cit.).

**Lymphangitis** is inflammation of lymphatic vessels. *Reticular* or *capillary* lymphangitis (erysipeloid), which is inflammation of lymphatic radicles, is seen in some circumscribed inflammation of the skin. It is apt to attack the hands, causing redness and swelling, fading at the point of initial trouble while it spreads at the periphery; it is caused by micro-organisms derived from decomposing animal matter (see page 198). Erysipelas also causes it (see page 199). *Tubular* lymphangitis, which is due to the entry into the lymphatic ducts of virulent micro-organisms or toxic materials, is seen after the infliction of dissecting wounds, septic wounds, snake-bites, etc. It is announced by edema and by minute, hard, red streaks running from the wound up the extremity. Suppuration may occur.

**Septic** or **infective lymphadenitis,** or inflammation of the glands, may follow lymphangitis or may be due to the deposition of infective material, the lymph-vessels not being inflamed. In this form of lymphadenitis there are pain, tenderness, and swelling; in severe cases there are a chill and a septic fever. Suppuration may arise.

The **treatment** is to drain and asepticize the wound, to apply iodin, blue ointment, or ichthyol over the glands and vessels, and to employ rest, heat, and compression. Internally, milk-punch, quinin, and nourishing diet are required. If the glands do not rapidly diminish in size after disinfection of a wound, and if they are in an accessible region, extirpate them. If suppuration of the glands occurs, incise and drain.

**Acute lymphadenitis,** or acute inflammation of the lymphatic glands, may be due to tubercle, syphilis, glanders, cold, or traumatism. Suppuration may or may not occur. In inflammatory lymphadenitis there are pain, heat, and nodular swelling. In severe cases there is fever.

The *treatment* is to asepticize any area of infection, place the glands at rest, apply heat and ichthyol ointment, or inject into the gland every day 5 minims of a 3 per cent. solution of carbolic acid to prevent suppuration. If the glands do not rapidly shrink, extirpate them. If pus forms, evacuate it, drain, and asepticize.

**Chronic lymphadenitis** is almost invariably syphilitic or tuberculous. It requires constitutional treatment and the local use of ichthyol, iodin, or blue ointment. If these remedies are not rapidly successful, tuberculous glands should be removed, but syphilitic glands rarely require such radical treatment.

**Lymphangiectasis** (*varicose lymphatics*), or dilatation of the lymphatic vessels, is due to obstruction. It may be congenital (macroglossia; lymphatic nevus, see page 368). It may be acquired. Many external causes may produce obstruction; for instance, the removal or suppurative annihilation of a considerable group of lymphatics; pressure of a scar or of a new growth upon lymph-vessels; tuberculosis or neoplasm of a group of glands. In many cases of external pressure upon lymphatics there is no lymphangiectasis because the lymph finds other channels. In fact, it has been proved that ligation of a large lymphatic trunk is not of necessity followed by lymphangiectasis. Even when the condition arises from external pressure, it is usually temporary, although, particularly if glandular tumors exist, it may be permanent.

The persistent cases are usually due to obstruction within the ducts, for instance, endothelial proliferation as a result of chronic lymphangitis, or recurrent attacks of acute capillary lymphangitis (erysipelas) or ordinary acute lymphangitis; or tuberculosis and other chronic infections. There may be such a condition as primary intralymphatic endothelial proliferation ("Med. Record," Sept. 6, 1902). Blocking with *filarial worms* may occur, and if it does, the lymphangiectasis is usually situated in the pubic, the inguinal, or the scrotal region, or on the inner side of the thigh. There are two forms of lymphangiectasis: the *varicose*, in which the vessels have a tortuous outline, like varicose veins, but are covered only with surface epithelium; and *lymphatic warts* (*lymphangioma circumscriptum*), in which wart-like masses spring up, these masses being covered with epithelium and filled with lymph. In most cases of lymphangiectasis there is considerable hard edema (*lymphedema*). Lymphangiectasis sometimes develops in an upper extremity after removing the axillary glands, and in the lower extremity after removing the inguinal glands. Periodic attacks of pain and redness occur in the area of lymphangiectasis, and usually at such times fever develops. Rupture of the dilated vessels causes a flow of lymph (*lymphorrhea*). Infection and erysipelas are apt to occur; it may be over and over again. It is uncertain whether these repeated attacks of erysipelas cause and maintain or are predisposed to by lymphangiectasis.

**Treatment.**—If the entire area can be removed, it should be extirpated. Maitland ("Brit. Med. Jour.," Jan. 25, 1902) shows that many varices are local and can be removed. If the varices are only partially removed, lymphorrhea will probably develop.

**Lymphangioma** is an advanced stage of lymphangiectasis (see page 368).

The **treatment** in mild cases is to pierce each vesicle with the negative pole of a galvanic battery and pass a current. In severe cases destroy the mass with the Paquelin cautery or excise it with a knife or with scissors.

**Elephantiasis.**—*True* elephantiasis (elephantiasis Arabum) is chronic hypertrophy of the skin and subcutaneous tissues following upon a lymphangi-

ectasis produced by a nematode worm (the Filaria sanguinis hominis). The disease is only encountered in the tropics. Elephantiasis of the scrotum is called *lymph-scrotum*. Elephantiasis of the leg is called *Barbadoes leg*.

*Spurious* or *pseudo*-elephantiasis (Fig. 815) is hypertrophy of the skin and subcutaneous tissue due to chronic inflammation (for instance, in a leg which possesses an ancient ulcer, or in the scrotum of a man with urinary fistula).

The **treatment** of true elephantiasis is massage and bandaging, sometimes ligation of the artery of supply, extirpation, or amputation.

**Tuberculous Glands.**—(See page 250.)

**Lymphadenoma** (*Malignant Lymphoma; Hodgkin's Disease; Pseudo-leukemia*).—The term "lymphoma" is used loosely to designate any persistent swelling of a lymphatic gland or glands. Lymphadenoma means a swelling of lymph-glands or lymphadenoid tissue, which swelling is progressive in character, involves group after group of glands, is associated with anemia, and often accompanied by secondary growths in the abdominal viscera.

This disease is most common in those under forty, and affects males far more frequently than females. In many cases the disease arises slowly in apparently healthy glands, and exists for some time before it takes on signs of malignancy and invades distant glands. In some cases the disease has a tendency to generalization from the start; in others it appears to remain localized for many months. A gland enlarged from irritation or from tuberculous disease may become lymphadenomatous, and tubercle bacilli can sometimes be found in lymphadenomatous glands. Lazarus asserts that the disease is lymphosarcoma and the tuberculosis accidental. Musser, Sternberg, and others believe that tuberculosis is the disease. Some few believe that lymphadenoma is really tuberculosis, but this view seems to have been definitely disproved.

Fig. 815.—Spurious elephantiasis. No filariæ found. Born and lived in Philadelphia.

That the disease is at least similar to sarcoma seems certain. That it is a variety of sarcoma is highly probable. In Hodgkin's disease Coley's fluid (the mixed toxins of erysipelas and Bacillus prodigiosus) causes reaction as in sarcoma. There is a form of tuberculosis strongly resembling Hodgkin's disease, but I do not believe that the two processes are identical. The glandular and splenic enlargements are neoplastic and not hyperplastic. The new tissue formed is called lymphadenoid tissue and, according to Banti, it is often atypical, tends to invade glandular trabeculæ and capsules, sometimes adjacent tissue, and gives origin to metastases.

Leukemia and pseudoleukemia are closely related, and both, according to Banti, are sarcoma. In leukemia the influence that stimulates proliferation falls chiefly upon the bone-marrow; in Hodgkin's disease, upon the lymph-

nodes (Neumann, quoted by Coley, in a forceful article maintaining that Hodgkin's disease is a type of sarcoma, "Trans. Am. Surg. Assoc.," 1908).

**Symptoms.**—The glands in the neck are usually involved first, but the disease may begin in the axillary glands, the thoracic glands, or the intra-abdominal glands.

Two or more regions are sometimes involved simultaneously or almost simultaneously.

When the disease begins in the neck it affects at first one side, and after many weeks or months the other side becomes involved. The glands are at first hard, separated from each other, movable, and the skin moves freely over them. Later the large glands weld together and form great masses upon both sides of the neck and in the axillæ, which may obstruct respiration.

After a time a very large mass may break through its capsule and infiltrate adjacent structures, and in very rare cases the skin becomes adherent and finally breaks. Intrathoracic symptoms point to involvement of the thoracic glands. It may be possible to palpate enlarged abdominal glands.

The leukocytes may be increased to 20,000 or more, but in many cases the count is normal and the relative proportion of the varieties remains normal. In a certain percentage of cases, without any increase in the number of leukocytes, there is great relative lymphocytosis. Early in the case the number of red cells may be unaffected, but after a time anemia develops. A fall in hemoglobin is noted early and this fall is somewhat more rapid than the decrease of erythrocytes ("Clinical Hematology," by J. C. DaCosta, Jr.).

The spleen is enlarged; the thyroid may be enlarged. When anemia becomes marked there are the ordinary symptoms which go with it, viz., palpitation, breathlessness, indigestion, vertigo, headache, pallor, and sometimes epistaxis. Occasionally, without obvious reason, the glands suddenly increase in size or rapidly undergo a notable but temporary diminution.

Slight fever exists at times in many cases, and ague-like paroxysms may occur. During the existence of fever the glands usually increase rapidly in size.

**Diagnosis.**—In a widespread case the diagnosis is easy; in a localized case it is difficult. True tuberculous glands are most apt to first appear in the submaxillary triangle; lymphadenomatous glands, in the root of the neck or in the occipital triangle. Tuberculous adenitis is most common in children. As a rule, tuberculous glands caseate, but they may remain localized for years if caseation does not occur. The tuberculous glands usually soon become adherent and immovable. Lymphadenoma is most common after twenty, rarely remains localized for more than a few months, rarely softens unless very large, and the glands are separated and movable until a huge mass forms. Early softening, prolonged limitation to one region, and absence of pronounced anemia in a person under twenty point to tubercle. In doubtful cases a gland should be removed for microscopical and bacteriological study.

In widespread tuberculous lymphatic involvement, simulating Hodgkin's disease, fever is far more likely to be present than in Hodgkin's disease. LaRoy ("Archives Internat. de Chir.," 1907, vol. iii) says that tuberculous glands are but little improved by the *x*-rays (a statement I do not altogether endorse), whereas, enlargements in Hodgkin's disease may be greatly benefited, and that in tuberculous conditions there is no particular tendency to hemorrhage and there often is in Hodgkin's disease. Coley ("Trans. Am. Surg. Assoc.," 1908) shows that the patients with Hodgkin's disease react strongly to the toxins of erysipelas.

**Prognosis.**—The disease is almost always, if not invariably, fatal. Most cases die within three years, some die within six months, some few live four or five years or more.

**Treatment.**—If the glands are localized to one side of the neck, or even

to both sides of the neck, remove them. Early removal before dissemination has occurred may possibly save the patient. If early or radical removal is not possible, do not operate, but treat the patient with nutritious food, tonics, course of arsenic, the mixed toxins of erysipelas and the Bacillus prodigiosus, and applications of the *x*-rays. Coley treated 2 cases by the mixed toxins. In both cases the lymphatic, hepatic, and splenic enlargements entirely disappeared ("Surg., Gynec., and Obst.," August, 1911). Efforts are now being made to obtain a curative serum. Beck makes nucleoprotein serum from the glands of cases of Hodgkin's disease.

## XXXV. BANDAGES

A bandage is a fibrous material which is rolled up and is then employed to retain dressings, applications, or appliances to a part, to make pressure, or to correct deformity. It may be composed of flannel, of calico, of unbleached muslin, of plain gauze, of gauze infiltrated with plaster of Paris or soaked in silicate of sodium, or of gauze wet with corrosive sublimate solution. Unbleached muslin, which is the best material for general use, is washed to remove the sizing, is torn into strips, and the edges are stripped of selvage. One end is folded to the extent of 6 inches, this is folded upon itself again and again until a firm center is formed, and over this center the bandage is rolled. In a well-rolled bandage the center cannot be pushed out of the roll. A roller bandage is divided into the initial end, which is within the roll, the body or rolled part, and the terminal end, which is free. In applying a bandage the outer surface of the terminal end is first laid upon the part.

A *cylindrical* part of the body may be covered by a *circular* bandage, each turn exactly covering the previous turns. A *conical* part may be covered by a *spiral* bandage, each turn ascending a little higher than the previous turn. As each turn of a spiral bandage is tight at its upper and loose at its lower edge, the *reverse* was devised to correct this inequality; hence a conical part should be covered by a *spiral reversed* bandage. To make a reverse, hold the roller in the right hand, start the bandage obliquely upward (do not have more than 6 inches of slack), place the thumb across the fresh turn, fold the bandage down without traction, and do not make traction until the turn has been carried well around the limb. A projecting point is covered with *figure-of-8* turns. The groin, shoulder, breast, or axilla can be covered by figure-of-8 turns, each succeeding turn ascending and covering two-thirds of the previous turn and forming a figure like "the leaves of an ear of corn." Such a figure is called a "spica." In bandaging an extremity the peripheral turns should be tighter than the turns nearer the body. Never apply a tight bandage to the leg or the arm without including the foot or the hand. In firm dressings of the forearm and arm it is well to leave the ends of the fingers exposed, and use them as an index of the condition of the circulation in the part. In firm dressings of the leg and thigh leave the toes exposed.

**Spiral Reversed Bandage of the Upper Extremity.**—To apply this form of bandage use a roller 2½ inches wide and 8 yards long. Take a circular turn about the wrist, and a second turn to hold the first; pass obliquely across the back of the hand to the extremities of the fingers; ascend the hand to the root of the thumb by several spiral turns; cover the wrist by ascending figure-of-8 turns; ascend the forearm by spiral reversed turns; cover the elbow by a figure-of-8, and the arm by

Fig. 816.—Spiral reversed bandage of the upper extremity.

spiral reversed turns; end the bandage by two circular turns, and pin them together (Fig. 816).

**Spiral Bandage of All the Fingers** (*Gauntlet*).—The gauntlet bandage requires a roller 1 inch wide and 3 yards long. Take two circular turns around the wrist, pass obliquely across the wrist to the root of the thumb, and descend to its tip by spiral turns; cover in the thumb by ascending spiral turns, and return to the wrist. Cover successively each finger in the same manner, and terminate by two circular turns around the wrist (Fig. 817).

Fig. 817.—Gauntlet bandage.

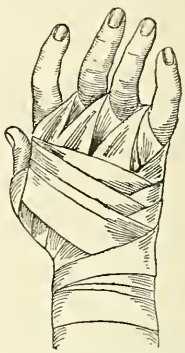


Fig. 818.—Demigauntlet bandage.

**Spiral Bandage of the Palm or Dorsum of the Hand** (*Demigauntlet*).—The demigauntlet requires a roller 1 inch wide and 3 yards long. This bandage has only a limited value; it must not be applied tightly, as it makes much pressure at the finger-roots, but leaves the fingers free. If it is desired to cover the palm, supinate the hand; if to cover the dorsum, pronate the hand. Take two circular turns around the wrist, sweep around the root of the thumb, and return to the point of origin. Treat each finger in the same way. End by circular turns around the wrist (Fig. 818).

**Spica of the Thumb.**—For this bandage use a roller 1 inch wide and 3 yards long. Start at the wrist, and reach the tip of the thumb as in applying

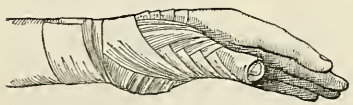


Fig. 819.—Spica of the thumb.

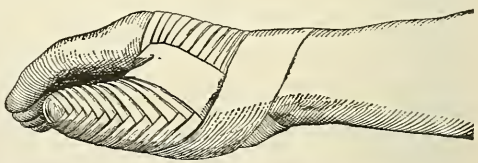


Fig. 820.—Selva's thumb bandage applied.

a spiral bandage of a finger. Make a series of ascending figure-of-8 turns between thumb and wrist, each ascending turn overlying two-thirds of the previous turn; terminate with a circular of the wrist (Fig. 819).

**Selva's Thumb Bandage** (Fig. 820).—Lay the terminal end of the bandage on the outer side of the second phalanx of the thumb, near the base of the phalanx. Carry it over the palmar side of the pulp of the last phalanx to the inner side of the second phalanx. The surgeon holds this turn in place with his left thumb and index-finger. The roller is returned in a recurrent manner to its place of origin, overlaps the preceding turn, and is placed as much as possible on the dorsum. The roller is carried over the dorsum of the terminal phalanx and is turned around the tip, the loop crossing over the center of the nail.

Figure-of-8 turns are now made over the dorsum of the hand, over the palm, and returning to the terminal phalanx, and an ascending spica is made.[1]

**Spiral Reversed Bandage of the Lower Extremity.**—Take a roller 2½ inches wide and 7 yards long, and make two circular turns just above the malleoli, and an oblique turn across the dorsum of the foot to the metatarso-phalangeal articulation: make a circular turn, and cover the foot with ascending spiral reversed turns; return to the ankle by a figure-of-8: ascend the leg by spiral reverses: cover the knee by a figure-of-8, and the thigh by spiral reverses; terminate by two circular turns (Fig. 821).

Fig. 821.—Spiral reversed bandage of the lower extremity.

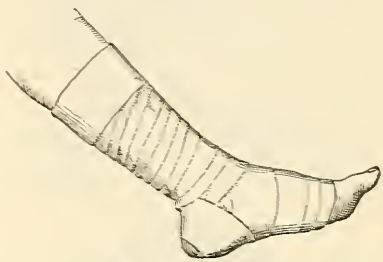


Fig. 822.—Method of covering the heel.

**Bandage of the Foot Covering the Heel** (*American Bandage of the Foot*).—Take a roller 2½ inches wide and 7 yards long. The bandage is begun as a spiral reversed bandage of the lower extremity. After the foot is well covered by ascending spiral reversed turns, carry the bandage directly around the point of the heel and return to the instep: from this point carry it around the back of the ankle, down the side of the heel, under the heel, up to the instep, around the ankle in the opposite direction, down the opposite side of the heel, and under the heel and up to the instep; take the roller to above the malleoli, and end by a circular turn (Fig. 822).

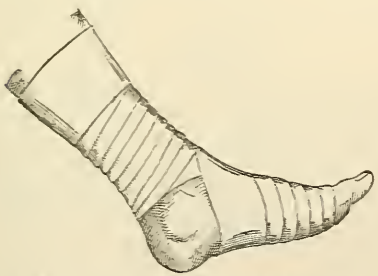


Fig. 823.—Figure-of-8 bandage of the ankle.

Fig. 824.—Spica of the instep.

**Bandage of the Foot Not Covering the Heel** (*French Method*).—Take a roller 2½ inches wide and 6 yards long. Make a spiral reversed bandage of the foot and a figure-of-8 of the ankle-joint (Fig. 823).

**Spiral Bandage of the Foot Covering the Heel** (*Ribbail's Bandage; Spica of the Instep*).—Take a roller 2½ inches wide and 6 yards long. Apply as a spiral reversed bandage of the lower extremity until the metatarsus is well covered. Carry the bandage, parallel with the margin of the foot (the inner

[1] "Medical News," Sept. 28, 1895.

or outer margin, according as to whether it is the left foot or the right), around the posterior aspect of the heel, along the opposite margin of the foot to cross the original turn at the median line of the dorsum. Make a number of these ascending turns, each turn covering in three-fourths of the previous turn; terminate by circular turns above the ankle (Fig. 824).

**Crossed Bandage of Both Eyes** (*Figure-of-8 of Both Eyes*).—Take a roller 2 inches wide and 6 yards long. Make a circular turn around the forehead from right to left, a second turn to hold the first, a turn downward over the left eye, under the left ear, around the back of the neck, and upward under the right ear and over the right eye; repeat these turns, and terminate by a circular turn of the forehead (Fig. 825).

**Borsch's eye-bandage** is convenient and useful (Fig. 827). A narrow bandage is laid along the head and permitted to hang down the face in front of the sound eye. A circular bandage is applied around both eyes, and over the narrow bandage (A). The narrow strip is lifted and pinned, and the sound eye is thus uncovered. Of course, the posterior end of A should first be pinned to the circular turn.

**Barton's Bandage** (*Figure-of-8 of the Jaw and Occiput*).—Take a roller 2 inches wide and 5 yards long. Place the initial extremity of the bandage behind the inion; pass over the right parietal bone, across the vertex, down the left

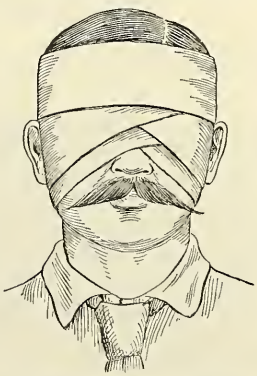


Fig. 825.—Crossed figure-of-8 bandage of both eyes.

Fig. 826.—Barton's bandage or figure-of-8 of the jaw.

side in front of the ear, under the chin, up the right side in front of the ear, across the vertex, and across the left parietal bone to the point of origin. A turn is now taken forward along the right side of the jaw to the chin, and backward along the left side of the jaw from the chin to the nape of the neck; repeat these turns, and pin the points of junction (Fig. 826). In Barton's bandage the ear lies in an uncovered triangle. The bandage may be finished by circular turns around the forehead. Barton's bandage is used for fracture of the lower jaw.

**Gibson's Bandage.**—Take a roller 2 inches wide and 6 yards long. Make three vertical turns around the head and the jaw in front of the ear; reverse the bandage above the level of the ear, and carry it horizontally around the forehead and head three times; drop the bandage to the nape of the neck, and take three turns around the neck and jaw; terminate by taking from the nape of the neck a half-turn upward, carrying the bandage forward to the forehead, and pinning it over the neck and over the forehead. Pin each point of junction (Fig. 828). Gibson's bandage is used for fracture of the lower jaw.

**Crossed Bandage of the Angle of the Jaw** (*Oblique Bandage of the Jaw*).—Take a roller 2 inches wide and 6 yards long. Make a circular turn

around the forehead toward the affected side; and a second turn to hold the first; take the turn to the back of the neck; carry it forward on the sound side, under the ear and chin; now make a series of turns around the head and jaw, in front of the ear on the injured side, but back of the ear on the sound side: these turns successively *advance* on the injured side only; terminate by going backward under the ear of the sound side to the nape of the neck, and then by

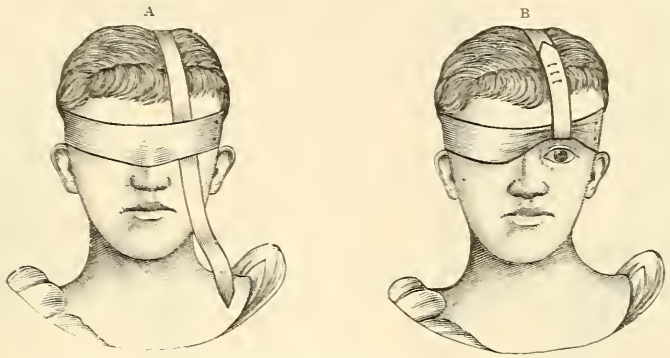



Fig. 827.—Borsch's eye-bandage: A, First step; B, second step.

taking two circular turns around the forehead (Fig. 829). This bandage is used for fractures of the ramus of the jaw and for holding dressings upon the face and the cranium.

**Spica of the Groin** (*Figure-of-8 of the Thigh and Pelvis*).—For one groin the roller is 3 inches wide and 7 yards long; for both groins, 3 inches wide and 10 yards long. Take two circular turns, from right to left, around the waist, then down over the front of the right groin, around the back of the thigh, up over the front of the right groin, around the waist, down over the front of the left groin, round the back of the thigh, up over the left groin, and around the

Fig. 828.—Gibson's bandage.

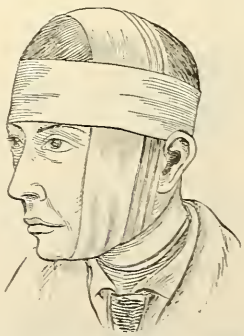


Fig. 829.—Oblique or crossed bandage of the angle of the jaw.

waist. The map being thus laid out, the turns are continued and ascended, each turn overlying one-third of the previous turn, and the bandage is completed by a circular turn around the waist (Fig. 830). Pin the crossed pieces.

**Spica of the Shoulder.**—Take a roller 2½ inches wide and 7 yards long. Make a circular turn and several spiral reversed turns around the upper arm; then, coming from behind forward, carry the bandage over the shoulder, across the front of the chest, through the opposite arm-pit, and return across the back to the shoulder. Make successive and advancing turns (Fig. 831).

Figure-of-8 bandages of the elbow, both shoulders (posterior figure-of-8), the neck and axilla are shown in Figs. 832, 833, and 834.

A figure-of-8 bandage of the breast is shown in Fig. 839.

**Velpeau's Bandage.**—Take a roller $2\frac{1}{2}$ inches wide and 10 yards long. Place the palm of the hand of the injured side upon the shoulder of the sound side, interposing cotton between the arm and the side. Start the bandage at the axilla of the sound side posteriorly, carry it across the back to the shoulder

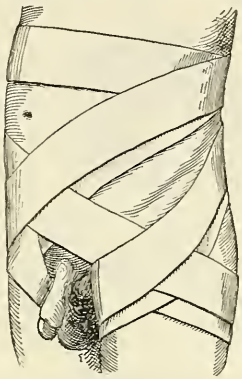


Fig. 830.—Spica of the groin.

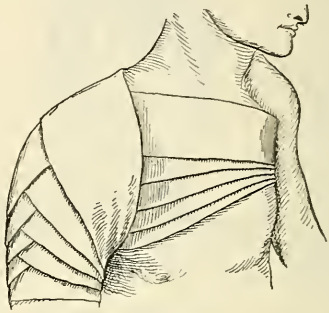


Fig. 831.—Spica of the shoulder.

of the injured side, down the front of the arm and under the arm just above the elbow, returning to the point of origin; repeat this turn, but, on reaching the axilla the second time, cross the back and pass around the chest, including the arm; keep on with these turns, each alternate turn going over the injured clavicle, each alternate turn encircling the arm and the body, the first turns advancing and the second turns ascending (Fig. 835). Pin the crossed pieces. This bandage is used for fracture of the clavicle.

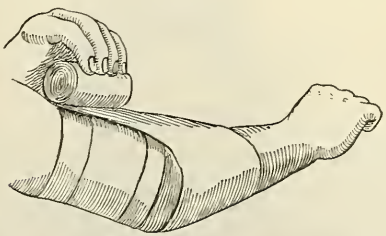


Fig. 832.—Figure-of-8 bandage of the elbow.

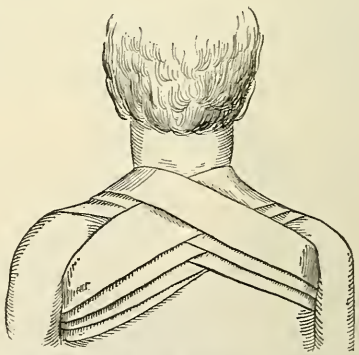


Fig. 833.—Posterior figure-of-8 of both shoulders.

**Desault's Apparatus.**—This apparatus consists of three rollers, a pad, and a sling. Each roller is $2\frac{1}{2}$ inches wide and 7 yards long. The pad, which is wedge shaped, is inserted into the axilla with the base up. The *first roller* is used to hold the pad (Fig. 836). The *second roller* binds the arm to the side over the pad. This pad is a fulcrum, the shoulder is the weight, the arm is the lever, and the second roller of Desault corrects the inward deformity of a fractured clavicle (Fig. 837). The *third roller* corrects the downward and forward displacement. It starts in the axilla of the sound side anteriorly, crosses the chest to the shoulder of the injured side, runs down the back of the arm, around the elbow, and crosses the chest to the point of origin, forming the anterior triangle; it is now carried through the axilla of the sound side to the

back, crosses the back to the shoulder of the injured side, runs down the front of the arm, around the elbow, and across the back to the axilla of the sound

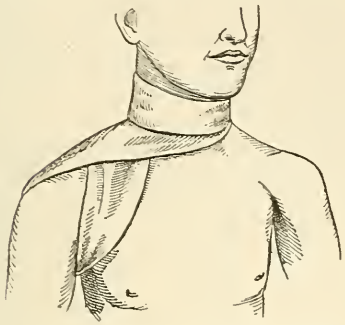


Fig. 834.—Figure-of-8 of neck and axilla.

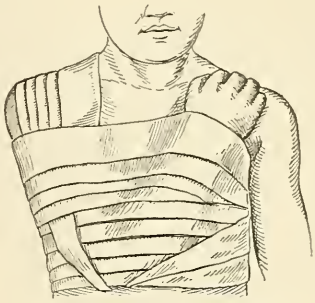


Fig. 835.—Velpeau's bandage.

side, forming the posterior triangle (Fig. 838). The formula for the Desault bandage is: start in the axilla of the sound side anteriorly, run from the axilla

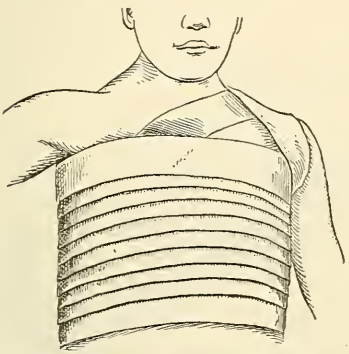


Fig. 836.—Desault's bandage, first roller.

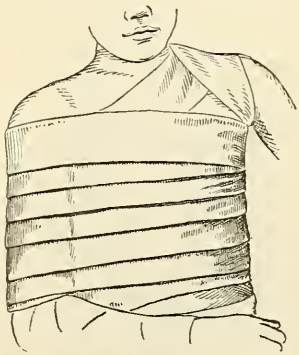


Fig. 837.—Desault's bandage, second roller.

to the shoulder, from the shoulder to the elbow, from the elbow to the axilla, and pass to the back; from the axilla to the shoulder, from the shoulder to the

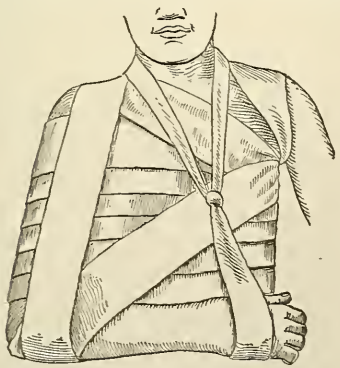


Fig. 838.—Desault's bandage, third roller.

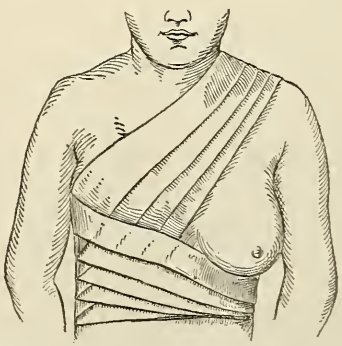


Fig. 839.—Figure-of-8 bandage of the breast.

elbow, from the elbow to the axilla, and pass to the front. Pin the crossed pieces and hang the hand in a sling (Fig. 838).

**Recurrent Bandage of the Head.**—Take a roller 2 inches wide and 6 yards long. Make two circular turns horizontally around the forehead and head; when the middle of the forehead is reached, catch the bandage, take a half-turn, carry the bandage to the occiput, let an assistant catch it, take a half-turn, bring the roller forward to the forehead, covering a portion of the preceding turn; continue this process until the scalp is well covered; terminate with two circular turns around the forehead and head (Fig. 840). It is often advisable to take a turn around the head and chin. Pin the crossed pieces.

**Recurrent Bandage of a Stump.**—Take a roller 2 inches wide and 6 yards long. Make two light circular turns around the root of the stump; make recurrent turns covering the stump as is done in covering the head; take a circular turn around the root of the stump, oblique turns to the top of the stump, circular turns around the tip, and apply an ascending spiral reversed bandage (Fig. 841)

**T-Bandage of the Perineum.**—Pass the transverse part around the body above the iliac crests, and pin it in front; bring one of the tails over the dressing and up between the thigh and the genitals of one side, and the other tail over the dressing and up between the thigh and the genitals of the opposite side; secure these tails to the horizontal band.

Fig. 840.—Recurrent bandage of the head.

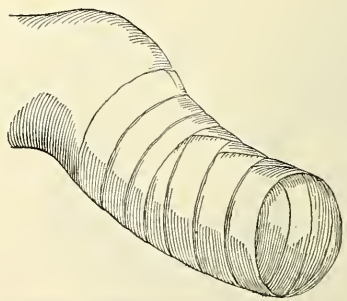


Fig 841.—Recurrent bandage of a stump.

**Handkerchief Bandages.**—Take unbleached muslin 1 yard square. The muslin folded once makes an *oblong* bandage; bringing its diagonal angles together makes a *triangle* bandage; a *cravat* is formed by folding a triangle bandage from summit to base; a *cord* is a twisted cravat. The triangle makes an admirable sling.

**Fixed Dressings.—Plaster-of-Paris Bandage.**—Cover the extremity with a cotton or flannel bandage or with a woolen stocking. Take a gauze roller infiltrated with plaster and place it endwise in a basin of tepid water, the water covering the plaster. When bubbles cease to arise, squeeze the bandage and apply it *without much tension*, smoothing out each turn with a moistened hand. As each bandage is taken from the basin drop a fresh one into the water. Apply four thicknesses of bandage, and finish the dressing by sprinkling dry plaster over the bandage and smoothing it with wet hands. The ordinary plaster will set in from fifteen to thirty minutes. If it is desired to have it set more rapidly, put a tablespoonful of salt in each pint of water used; if to have it set more slowly, pour stale beer into the water. The plaster bandage is removed by sawing it down the front or by moistening with dilute hydrochloric acid and then cutting through the moistened line with a strong knife. Gigli has devised a mode of application which enables us to remove the dressing with ease. A layer of cotton is placed

around the limb. A piece of parchment paper which has been wet and shaken out is placed over the cotton. A cord greased with vaselin is laid upon the paper in a position corresponding to the line we will wish to saw through the plaster. Apply the plaster bandage and see that the ends of the cord project beyond the bandage. When desiring to remove the bandage take a steel wire, make nicks on one side of it by means of a file, and attach the string to the wire. Pull the wire under the bandage. Attach each end of the wire to a wooden handle and saw through the plaster.[1]

**Silicate of Sodium Dressing.**—Protect the part as is done for a plaster bandage. Bandage the limb *loosely* with an ordinary gauze bandage, paint this bandage with silicate of sodium, apply another bandage and paint it, and so on until six layers are applied. Gauze bandages are better than ordinary bandages to take up silicate of sodium. Silicate dressings require from twelve to eighteen hours to dry, and they are removed by softening with warm water and then cutting.

## XXXVI. PLASTIC SURGERY

Plastic surgery includes operations for the repair of deficiencies, for the replacement of lost parts, for the restoration of function in parts tied down by scars, and for the correction of disfiguring projections. Many reparative operations have been devised. Among them are: cheiloplasty, or the construction of a new lip; the closure of a cleft in the palate, the lip, or the penis; the making of a new nose; skin-grafting; grafting of muscle or tendon; nerve-grafting; the introduction of celluloid or metal into the tissues to act as a support; the injection of paraffin into the tissues to amend a depression; the diminution in the size of a lip or a nose; the amendment of protuberant ears; the correction of distortion due to cicatrices; excision of scars; closure of congenital sinuses and of fistulæ; and removal of disfiguring growths.

The subject of plastic surgery is very extensive, and a treatise upon it should be consulted if one wishes to obtain detailed and comprehensive information.

A plastic operation can be successful after lupus only when the disease has been cured. It is useless to do a plastic operation during active syphilis, and a plastic operation for a syphilitic loss of substance is to be performed only after the patient has been thoroughly treated and the disease has been apparently cured. The first step of a plastic operation consists in making the surfaces which are to be brought together raw; the second step is the complete arrest of bleeding; the third step is the approximation of the surfaces without tension; the fourth step is to close any gap from which tissue may have been transplanted; and the final step is the application of the dressings.[2] The following are the methods used:[3]

*Displacement* is the method of stretching or of sliding: (1) Approximation after freshening the edges (as in harelip); (2) sliding into position after transferring tension to other localities (linear incisions to allow of stretching of the skin over large wounds). *Interpolation* is the method of borrowing material from an adjacent or a distant region or from another person: (1) *Transferring a flap with a pedicle*, which flap is put in place at once or is gradually gotten into place by a series of partial operations (as in rhinoplasty, when a flap is taken from the forehead); (2) *transplanting without a pedicle*, which is performed by placing in position and by fixing there portions of tissue recently removed from the part, from another part of the same individual, or from a lower animal (as

[1] "La Semaine Méd.," Nov. 3, 1895. [2] "American Text-Book of Surgery."
[3] Ibid.

replacement of the button of bone after trephining, transplanting a piece of bone from a lower animal to remedy a bone defect in a human being, or the grafting of a piece of nerve from a lower animal or from an amputated human limb to remedy a loss of nerve in a human being). *Retrenchment* is the removal of redundant material and the production of cicatricial contraction.

**Skin-grafting.**—As long ago as 1847 Dr. Frank Hamilton partly covered an ulcer with a pediculated flap, and trusted that the uncovered portion would be healed by new skin from the flap. We may graft small pieces of epithelium taken from the patient, another person, or one of the lower animals, or may graft large pieces of epithelium. The grafts should, if possible, come from the person to be grafted. The epidermic scales may be scraped off the sound skin and grafted. Lusk has blistered the skin with cantharides and grafted portions of the epidermis. The shavings of a corn and fragments of hair roots have been used. The best plan is to cut off and transplant small bits of epidermis.

Fig. 842.—Injury caused by crush and burn. Healed by granulation in eight months. Showing a condition after removal of scar of the palm, which has been repaired by stitching in an autoplastic graft (free flap) from the thigh (George S. Brown).

Grafts may come from another person or from a lower animal, but such grafts are not so apt to grow as those obtained from the individual, and even when they do grow, fail to furnish a secure cicatrix. Frog-skin furnishes unsatisfactory grafts. Some surgeons have used bits of sponge; others, the skin of rabbits, guinea-pigs, or pups. Arnot has employed the lining membrane of a hen's egg, cut in strips and applied upon the wound with the shell-surface uppermost. Small bits of epidermis taken from a recently amputated foreskin or leg may be used.

**Reverdin's Method.**—This operation was devised by Reverdin in 1869. Small bits of epithelium are used, and they are taken, preferably, from the person himself. The surface to be grafted should possess healthy granulations level with the skin. Cleanse the skin from which the grafts are to come, the ulcer, and the skin about it, and, if corrosive sublimate is used, wash it away with a stream of warm normal salt solution. Thrust a sewing-needle under the epidermis to raise it, cut off the graft by a pair of scissors, and place the raw surface of the graft upon the ulcer. After applying a number of grafts, place thin pieces of gutta-percha tissue over them and extending on each side of the ulcer, and so placed as to have distinct in-

tervals between them, the gaps permitting drainage. Rubber tissue must be aseptic and moist with warm normal salt solution. Dress with a pad of aseptic gauze moistened with salt solution; place over this gauze a rubber-dam, and over the latter absorbent cotton and a bandage. In the case of a child apply a light silicate bandage. If the grafted area is very extensive or if it is in the lower extremity, put the patient in bed. In forty-eight hours remove all the dressings except the gutta-percha tissue, irrigate with normal salt solution, and reapply the dressings. All signs of the grafts will often have disappeared. In a day or two more, at the site of grafting, bluish-white spots should appear, which are islands of epidermis. Each graft is capable of forming about $\frac{1}{2}$ inch of cicatrix. Grafting also stimulates the edges of the ulcer to cicatrize and contract. At the end of seven days the special dressings can be dispensed with. The spot from which the grafts were taken is dressed antiseptically. Reverdin's method does not limit cicatricial contraction to any great degree, and the new skin is apt to break down.

Fig. 843.—Claw-hand from burn. A flap with a pedicle was taken from the chest. The pedicle was cut on ninth day.

**The Ollier-Thiersch Method.**—Ollier, of Lyons, in 1872 succeeded in transferring large pieces of epidermis. In 1886 Thiersch, of Leipzig, set forth the technic practically as it is employed to-day. The Ollier-Thiersch method is performed as follows: Thoroughly asepticize the ulcer, the surrounding skin, and the site from which the graft is to come (the inner side of the arm or the thigh), and wash away the mercurial preparation with normal salt solution. Apply dressings wet with salt solution. On bringing the patient into the operating room remove the dressings from the ulcer, scrape the ulcer and its edges, irrigate with salt solution, and compress to arrest hemorrhage. Grafts are then obtained by putting the prepared skin upon the stretch and cutting strips with a razor. While the razor is being used the part is constantly irrigated with salt solution. Mixter's apparatus enables one to perform this operation with great neatness and speed. This apparatus consists of a knife and an open square with sharp points on the under surface. The square is forced down upon the front of the thigh, the epidermis mounts up in the opening to above the level of the metal sides, and the grafts may be cut with ease. The graft contains the epidermis, the rete, and part of the true skin. In Halsted's clinic the skin of the thigh is made tense by pressing and drawing upon it with a piece of asepticized wood, the wood is drawn slowly along, and is followed closely by the sharp catlin, by which the surgeon cuts long grafts. The grafts are pressed into place upon the raw sur-

face, and each graft overlaps a little the edges of the wound and the adjacent grafts. The skin wound is dressed antiseptically, and the grafted area may be dressed as in Reverdin's method. If a ring of aseptic gauze be made to encircle the limb below the grafted area, and another ring above the grafted area, and if on these pads little strips of wood wrapped in aseptic gauze be laid, a cage is made, and around this cage the dressings may be applied (moist chamber plan) (Fig. 844).

**Wolfe's Method.**—It was pointed out by Wolfe that a piece of skin, comprising the entire thickness of that structure, can be successfully transplanted without a pedicle. The ulcer is extirpated and asepticized and bleeding is arrested. The flap is cut one-sixth larger than the surface to be covered. Fat is kept out of the graft. The bit of tissue is laid upon the wound, the edges of the graft being brought against the edges of the raw area. It is not necessary to employ sutures. The part is dressed in a moist chamber. If the graft perishes, remove it.

**Subcutaneous Injection of Paraffin for Prosthetic Purposes.**—The principle of injecting solidifying oils into tissues to mechanically obtain effects was first laid down by J. Leonard Corning in 1891. The use of paraffin was introduced by Gersuny to amend the deformity of a saddle-nose. It has been used to limit incontinence of feces, incontinence of urine in women, to prevent reunion of nerves after division, as a counterfeit testicle, to obliterate small-pox marks, to narrow a hernial ring, to correct sinking of the cheek after removal of the upper jaw, and for other purposes (Moszkowicz, in "Wien. klin.

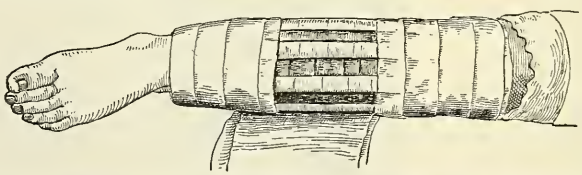


Fig. 844.—Mayer's dressing for Thiersch's method of skin-grafting ("Amer. Text-Book of Surg.").

Woch.," June 20, 1901). Paraffin is not toxic. Its injection may produce some swelling and redness, but applications of cold usually control inflammation. In two or three months the paraffin becomes hard like cartilage and encapsuled. It is questionable whether or not it is subsequently destroyed and replaced by granulation tissue. Sometimes sloughing takes place in the skin above it.

Prepare the paraffin as follows: In Gersuny's clinic solid paraffin is mixed with liquid paraffin. The melting-point of the mixture should be about 104° F. It is rendered sterile by boiling, is injected by a warm syringe, and as a semisolid, the skin having been first warmed by a hot sponge. After injection it is molded into proper shape. It sets in half a minute. It is not wise to use a mixture with a much higher melting-point, because it would possibly cause thrombosis in veins. There are difficulties and even dangers in the use of paraffin for saddle-nose. It should only be used when the skin is loose and elastic. It should never be used if there is great deformity, because then the amount required would surely cause dangerous tension. It is difficult to prevent the injected material from passing down the sides of the nose and up into the forehead. Cases of embolism causing blindness have been reported. The skin may slough if the injection is too hot or if it produces much tension.

**Paraffinoma.**—This term, suggested by Delangre, means an inflammatory new formation which may arise in the submucous or subcutaneous tissue about a depot of injected paraffin. It may or may not ulcerate. It is particularly apt to form in a tuberculous person. The swelling is marked and the

disfigurement great. The possibility of the formation of a paraffinoma is particularly great if paraffin is used in the subcutaneous tissue.

The only treatment is excision.

**Correction of Saddle-nose By the Insertion of a Plate.**—Saddle-nose is a condition in which the bones and cartilages have been destroyed by ulceration or collapsed by injury. It is useless to attempt correction by skin-flaps alone. Paraffin injections (see page 1264) may be used in the less severe cases. In a bad case we must transplant bone-flaps or insert a plate for support. The bone-flap operation is seldom satisfactory and, of necessity, creates a hideous scar. The insertion of a plate may give an excellent result, although the future is always uncertain. In 2 cases I have seen sloughing occur over the plate months after the operation. In 1 of these cases the sloughing was due to a blow from a cow's tail.

The plate used may be of silver, gold, or celluloid. An incision is made to permit the insertion of the plate. I agree with Leonard Freeman ("Annals of Surgery," August, 1907) that the incision along the bridge and the incision

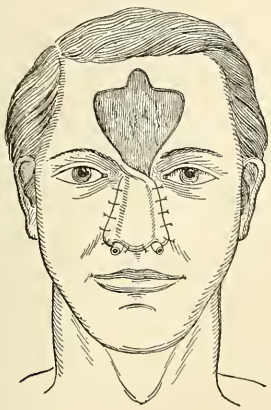


Fig. 845.—Indian method of rhinoplasty.

Fig. 846.—Italian method of rhinoplasty.

in the septum below the tip are both objectionable. The first leaves an unsightly scar and does not allow for stretching of the skin. The incision at the tip gives unsatisfactory access to the tissues requiring separation and is liable to infection from the nostril.

The best incision is Freeman's. This is a short incision across the root of the nose between the eyes. The skin is undermined along the bridge to the tip and along the sides. The undermined skin can be stretched if necessary.

**Rhinoplasty.**—The complete operation may be performed by transferring a flap from the forehead. This is known as the Indian operation. It was employed for centuries in India, and interest in it was awakened in England about 1820 by Mr. Carpue. The edges of the defect are made raw. A model of the desired nose, made out of gutta-percha, has its outlines marked upon the forehead, and the cut is made $\frac{1}{4}$ inch outside of the outline, so as to allow room for retraction. The flap is turned down and sutured in place (Fig. 845), care being taken not to cut off the blood-supply in the pedicle. Plugs of gauze or tubes are inserted to support the flap.

The complete operation can be performed by the Italian method (Tagliacotian method). This method was first described in Tagliacozzi's book, which was published in 1597. In this operation the flap is marked out on

the arm, is made twice the size of the desired nose, and is left attached by a broad pedicle. The nasal surface is rendered raw at proper regions, and the flap is sutured in place, the hand being held upon the head by a special apparatus (Fig. 846). The raw surface upon the arm is dressed. In about three weeks the flap is cut loose from the arm, and is pared and corrected as may be necessary.

The operations for harelip and cleft palate, and plastic operations on muscles, nerves, tendons, and bones are considered in other portions of this work.

## XXXVII. DISEASES AND INJURIES OF THE GENITO-URINARY ORGANS

**Hematuria.**—By this term is meant the voiding of bloody urine or of pure blood, the blood arising from any portion of the urinary apparatus, and the condition being a symptom and not a disease. In hematuria the urine contains more than blood coloring-matter, it also contains blood-corpuscles. The condition in which there is coloring-matter only is called *hemoglobinuria.* Hemoglobinuria may arise after burns, etherization, and taking urotropin, and during various fevers, especially in malaria (see page 1267). Hematuria may be a symptom of disease or of injury of some part of the urinary system (calculus, acute nephritis, pyelitis, renal tuberculosis, prostatic enlargement, morbid growth, wounds or contusions of the genito-urinary tract), of blood disorganizations (purpura, scurvy, variola, leukemia, or anemia), or of metallic poisoning (mercury, lead, or arsenic). It may arise during typhoid fever, in the beginning of an acute fever (especially variola), in hemophilia, in nephralgia, in malarial fever, and in kidney infarction resulting from endocarditis. It may be caused by parasites (Filaria sanguinis-hominis and Bilharzia hæmatobia). Some drugs are renal irritants and may cause hematuria (cantharides, oil of turpentine). Oxaluria is an occasional cause. The most usual cause of renal hematuria is stone. The color of the urine in hematuria may be anything between a light pink and a decided black, but these colors may be produced by agents other than blood. (See Sollmann's "Text-Book of Pharmacology.") Carbolic and salicylic acids make urine brown or greenish black; beet-root and sorrel, the color of blood; methylene-blue, green or blue. In melanosis and splenic fever the urine becomes brown. Senna and rhubarb make an acid urine yellowish brown and an alkaline urine purple. In jaundice the urine is yellow or green. Coal-tar products may make it blackish brown. Picric acid makes it yellow. Santonin makes an acid urine yellow and an alkaline urine pink. Logwood imparts no color to acid urine, but colors alkaline urine violet or reddish. Trional, sulfonal, tar, tannic acid, and gallic acid make urine brown.

**Tests for Blood.**—**Spectroscopal Test.**—Bloody urine, if fresh and diluted with water, shows the two absorption bands of oxyhemoglobin. The addition of ammonium sulphid causes the two bands to give place to the band of reduced hemoglobin. If bloody urine stands for some time the four bands of methemoglobin are discovered (von Jaksch).

**Heller's Test.**—Add potassium hydrate to the urine and boil; a red precipitate of earthy phosphates and hematin forms. Throw the precipitate upon a filter and treat it with acetic acid; a red solution is produced, which soon fades.

**Rosenthal's Test.**—Take the precipitate from caustic potash, dry it, and test it for hematin; put some of the dry sediment on a slide, add a crystal of common salt, apply a cover-glass, and cause a few drops of glacial acetic acid to flow under the glass; warm, but do not boil. Teichmann's crystals will appear on cooling.

**Struve's Test.**—Test the urine with hydrate of potassium, and add acetic acid in excess; a dark precipitate forms, which will yield crystals of hematin when treated with sal ammoniac and glacial acetic acid.

**Almen's Test (Guaiac Test).**—Take 10 c.c. of urine and pour upon its surface a mixture of equal parts of tincture of guaiac and old oil of turpentine; at the point of junction of this fluid with the urine there forms a white ring which turns to a striking blue. If a man is taking iodid of potash his urine shows blue in the guaiac test.

**The Benzidin Test.**—This is very delicate. If the reaction cannot be obtained the urine is certainly free of blood. Add 1 c.c. of glacial acetic acid to 10 c.c. of urine. Add to the mixture one-third of its volume of ether containing a few drops of alcohol, shake, and allow to stand. The ether rises to the top, is taken off by a pipet, and is put in a test-tube in which is the benzidin mixture (0.5 c.c. of a solution of a little benzidin in 2 c.c. of glacial acetic acid and 2 c.c. of hydrogen dioxid). Blood turns the reagent green or blue within two minutes. Later the color changes to purple. (See Holland's "Medical Chemistry and Toxicology.")

**Microscopical Test.**—The microscope shows numerous corpuscles except in a very alkaline urine, when but few corpuscles may be found.

In hemoglobinuria—a condition sometimes occurring after burns, as a result of large doses of urotropin, and during malaria, acute febrile maladies, metallic poisoning, acute alcoholism, poisoning by mushrooms, chlorate of potash, coal-tar products, pyrogallic acid, and naphthol—there is present blood coloring-matter, which is shown by Heller's test and by Almen's test. The spectroscope shows methemoglobin. The microscope shows no corpuscles or only a few, but discloses masses of pigment. Hemoglobinuria does not occur in diseases limited to the genito-urinary tract.

**Determination of the Source of the Blood.**—In a woman, be sure that the bloody urine is not due to a mixture with menstrual blood. If menstruation does exist, obtain the urine for examination by a catheter. The three-glass test may be of service. Blood may be thoroughly mixed with urine. Renal blood is sure to be mixed. Bladder blood may or may not be. Blood from the urethra comes out with the first urine. The source of blood may be determined certainly only by the urethroscope, cystoscope, or urethral catheter.

**Bleeding from the Kidney-substance.**—Bleeding from the *pelvis* of the kidney and from the *ureter* may be due to inflammation, congestion, contusion, stone, vicarious menstruation, hemorrhagic diathesis, powerful diuretics, fevers, purpura, tumors, catheterization of the ureter, etc. Blood is thoroughly mixed with the urine and no sediment forms (smoky urine). The corpuscles are profoundly altered, are devoid of coloring-matter, and show pale-yellow rings. The severity of the hemorrhage is measured by the number of the corpuscles. Von Jaksch states that the diagnosis between renal and ureteral hemorrhage rests on the nature of the casts and epithelium present. From the pelvis of the kidney and from the ureter comes small epithelium, the cells from the superficial layers being polygonal or elliptical, those from the deeper layers being oval or irregular. In hemorrhage from the ureter the cells are few; in hemorrhage from the pelvis they are plentiful and rest upon one another like "tiles on a roof" (von Jaksch). Cells from the tubules of the kidney are small, granular, and polyhedral, have large nuclei, and are often so arranged as to form cylinders (epithelial casts). The urine during and immediately after a renal hemorrhage is apt to be acid unless alkalis have been administered, unless the bleeding has been severe, or unless pus is present in the urine. A very large renal hemorrhage may cause the passage of almost pure blood. In *renal* hematuria there are aching

in the loin, numbness of the corresponding leg, and often renal colic. The use of the cystoscope enables the surgeon to determine if the hemorrhage is vesical or renal, and if it comes from one or both kidneys. If the bladder fluid is kept clear, the blood can be seen flowing out of the ureter of the damaged organ, or if both ureters are catheterized a sample of urine can be obtained from each kidney. Even when skilfully used the ureteral catheter is apt to cause slight hemorrhage. Hence, after catheterization, microscopical hemorrhage does not count in diagnosis.

**Spontaneous Hemorrhage Into the Kidney.**—This may or may not cause hematuria. It may arise during nephritis or may depend on arterial disease. No lesion may be discoverable. If accompanied by hematuria it is classed as essential hematuria (see below). The bleeding may take place beneath the capsule or through a tear in the capsule into the perirenal tissue. In some cases bleeding begins in the perirenal tissue. A possible cause is aneurysm of the renal artery. There are 26 cases of this condition on record ("Lancet," Jan. 27, 1912). Spontaneous hemorrhage comes on suddenly with shock and pain in the loin. There may or may not be hematuria. A mass can be palpated. In some cases the skin becomes discolored. Always operate. If hemorrhage comes from perirenal tissue, excise the bleeding tissue. If blood comes from the kidney it is usually necessary to perform nephrectomy. In spontaneous hemorrhage blood may tear its way into the peritoneal cavity. (See article by Russell S. Fowler, in "Annals of Surgery," Dec., 1911.)

**Essential Hematuria.**—In this condition the ureteral catheter reveals blood from one kidney only, there being no demonstrable lesion to account for the condition. Randall, in a very comprehensive article, draws the following conclusions regarding this condition ("Jour. Am. Med. Assoc.," Jan. 4, 1913).

He divides essential hematurias into three groups: (1) Nephritis with congestion plays the leading rôle. (2) Varicosities of vessels of renal pelvis resulting from some extrinsic condition affecting the kidney circulation. (3) Hemorrhage due to rupture of capillaries or to diapedesis of red blood-corpuscles. In either of these conditions congestion probably exists.

I believe that some cases are due to unrecognized papilloma. Essential hematuria is sometimes called *unilateral hematuria*, an unfortunate name, as many cases of hematuria which do not belong to this group are unilateral.

**Ureteral Catheterism.**—Catheterization of the ureters may give information of the greatest value. It enables the surgeon to obtain the urine from one kidney unmixed with urine from the other kidney and uncontaminated by material from the bladder or urethra. By this method we can determine if pus, blood, bacilli, etc., come from the ureter or kidney, and from which ureter or kidney. A stricture of or a calculus in a ureter can be located; hydronephrosis and pyonephrosis can be diagnosticated; the presence of both kidneys, if either kidney is diseased or if both are diseased, and the secretory capacity of each kidney in a given time, can be ascertained. The method is also employed to treat various conditions of the ureter and kidney.

Kelly impressed upon the profession that the ureters in women can be catheterized when the patient by the knee-chest posture permits the atmospherical distention of the bladder, so that the ureteral orifices can be inspected through a speculum. Light is reflected into the speculum, a forehead mirror and an electric light being employed. It may be necessary to dilate the urethra before inserting the speculum. It is rarely necessary to give a general anesthetic. Kelly moistens a bit of cotton wrapped on a metal rod in a 10 per cent. solution of cocain, introduces it just within the external urethral orifice, and holds it there for five minutes before beginning the operation. When the ureteral orifice of one side is found by inspection through the speculum, he introduces a sterile flexible silk catheter lubricated with boroglycerid

and it is pushed up from 4 to 6 inches in the ureter. A similar tube is introduced into the other ureter and the separated urines are collected in test-tubes. (See Kelly's "Operative Gynecology.") The catheterization of the ureters by this method can be performed only by a dextrous and experienced man; but such an individual can do it with ease and celerity; as practised by Kelly himself it seems, until one tries it, the perfection of easy simplicity.

Kelly has catheterized the ureter in man by inserting a straight speculum, placing the patient in the knee-chest position to inflate the bladder with air, and introducing a metallic catheter. (For a discussion of the technic of catheterizing the ureters see Cystoscopy, page 1301.)

**Segregation of urine** is the method of obtaining urine separately from each ureter by segregating each ureter's supply into an artificial trough, from which it is drawn. The method is seldom used in the United States at the present time. It is painful, unreliable, and has been replaced by ureteral catheterization. In cases in which the ureters cannot be found, segregation may be employed. The three most practical segregators are Harris's, Luys's, and Cathelin's. Professor Harris, of Chicago, has devised an instrument (Fig. 847) which in some cases simplifies the problem of obtaining unmixed urine

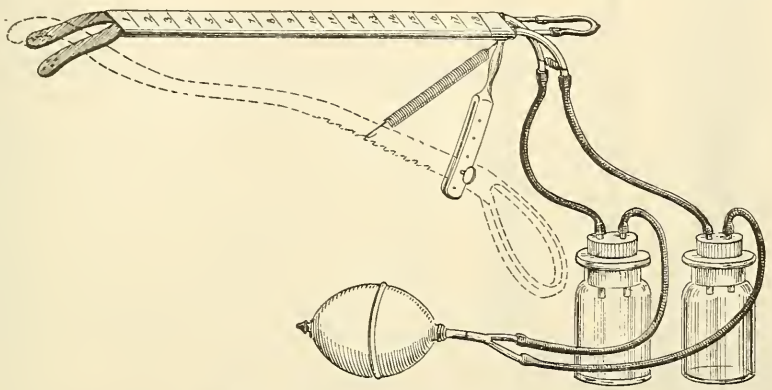


Fig. 847.—Harris's segregator fitted for use.

from each ureter. The double catheter is passed into the bladder. The lever is inserted in the rectum of the male and the vagina of the female. The lever is fastened to the perforated frame from the double catheter. The double catheter is now opened in the bladder, and the blades of the instrument are held in position by a spring. The end of the lever in the vagina or rectum humps up the floor of the bladder between the separated ends of the divided catheter, and forms a longtitudinal septum or watershed between the ureteral orifices. The end of each catheter lies in the bottom of a pocket to the side of the watershed. "By producing a very slight exhaustion of the air in the vials by means of the bulb the urine, as fast as it escapes from the ureters, drops directly into the ends of the catheters and flows at once into the vials, right and left respectively."[1]

In using this instrument place the patient flat on his back upon a table, the thighs and legs being flexed, and the feet, hips, and head being on the same level. Irrigate the bladder thoroughly with sterile water and have 150 c.c. of fluid in the bladder when the blades are opened. Leave the instrument in place for thirty minutes. It is rarely necessary to give an anesthetic. In some cases cocain must be used, and in some cases of painful cystitis ether

[1] "Jour. Cutan. and Gen.-Urin. Dis.," May, 1899.

should be given. Harris says the instrument should not be used if there is a growth of the bladder that bleeds easily, if the bladder is contracted, if there is a very large prostate or a vesical stone.[1]

In catheterization of the ureters there is always some danger of carrying infection upward from the bladder, and sometimes catheterization is impossible. It is impossible if great quantities of blood or pus make the urine opaque, or if inflammation of the bladder wall hides one or both ureteral orifices. The Harris method of segregation produces considerable pain, but is free from this danger. As a matter of fact, however, Harris's method often possesses elements of uncertainty, because the septum may not be perfect and the urine from one side sometimes contaminates the urine from the other. The separator devised by Luys in 1901 finds some warm advocates (Fig. 848). It causes less pain and accomplishes more certain results than the instruments of Harris or Cathelin. Barringer ("Am. Jour. Med. Sciences," March, 1907) points out that "there are certain classes of cases in which the separator cannot be used. They are the following: (A) Those in which the bladder capacity is less than 20 c.c.; (B) those in which the urethra is not penetrable by the instrument; and (C) those in which the base or neck of the bladder is distorted by (*a*) marked prostatic hypertrophy; (*b*) extreme anteversion or anteflexion of the uterus; (*c*) certain uterine tumors; and (*d*) marked cystocele." Catheterization of the ureters is not so safe as separation, is far more difficult, but gives more certain results.

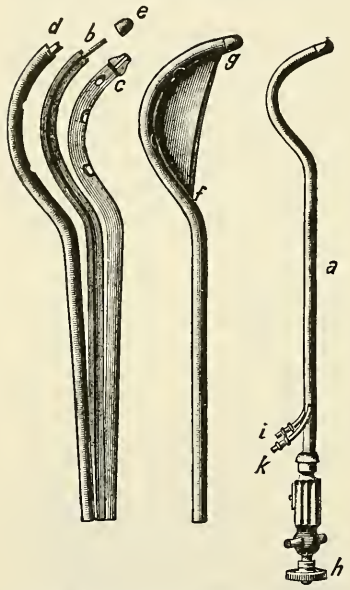



Fig. 848.—Luys's separator: *a*, The composite instrument ready for introduction; *i* and *k*, discharge tubes; *h*, screw to regulate the tension of the membrane; *b*, flat middle piece; *c* and *d*, grooved lateral portions; *e*, tip uniting the parts; *g–f*, rubber membrane, tense. The chain is not visible in the figure (Sahli).

**Urethral Hemorrhage.**—In urethral bleeding blood appears independently of micturition, or blood comes out first and is followed by clear urine. Urethral hemorrhage may arise from acute urethritis, from an inflamed stricture, from the passage of an instrument, or from some other traumatism. It may be due to a polypus, to a stone in the prostatic urethra, to violent or prolonged sexual effort.

The source of urethral hemorrhage can be ascertained by the use of the endoscope or, better, by means of the cysto-urethroscope.

**Vesical hemorrhage, including hemorrhage from the prostate,** may follow the relief or retention of urine (hence do not draw off all the urine at once when a bladder is distended), may be due to stone, inflammation, tumors, etc., or may arise from traumatisms, instrumental or otherwise. The color of the urine is usually bright red, but if long retained in the bladder it becomes black and often tarry. The reaction is alkaline. The clots, when floated out, are large and without definite shape. In micturition the urine is clear or only a little colored at the beginning, but becomes darker and darker as micturition continues, and as it ends the flow may consist of almost pure blood. In very small vesical hemorrhages the urine may be smoky. Crystals of triple phosphate indicate bladder disorder. The microscope shows

[1] M. I. Harris, in "Medicine," April, 1898.

colorless and swollen corpuscles and many polygonal cells. Symptoms of bladder mischief usually exist, but cystoscopical examination or exploratory suprapubic cystotomy may be required for the diagnosis.

**Pain in Genito-urinary Diseases.**—Pain as a symptom of genito-urinary disease may be found at some point distant from the seat of lesion. A stone in the bladder causes pain in the head of the penis just back of the meatus; stone in the kidney induces pain in the loin, the groin, the thigh, and the testicle; inflammation of the testicle causes pain in the line of the cord in the groin. In other cases of genito-urinary disease pain is felt at the seat of lesion, as in urethritis and prostatitis. Pain felt before micturition, and being relieved by the act, is found in cystitis and in retention of urine. Pain is felt during micturition in inflammation of the bladder, prostate and urethra, and in the passage of gravel or stone. Pain which is acute at the end of micturition is noted in stone in the bladder, in trigonitis or urethrocystitis (inflammation of the neck of the bladder), and in inflammation of the prostate gland. The pain caused by stone in the bladder, it may be observed, is ameliorated by rest and is aggravated by exercise unless the stone is encysted. The pain caused by acute prostatitis is intensified by defecation and the act is accompanied by the appearance at the meatus of a few drops of starch-like mucus.

**Frequency of Micturition.**—Frequent micturition arises from irritation of the sensory nerves, from phimosis, contracted meatus, inflammations, very acid urine, calculi, urethral stricture, and hyperesthesia of the urethra, Frequency of micturition may be due to spinal irritability from concussion or from sexual excess, from contraction of the bladder rendering the viscus unable to hold much, from worry, anxiety, fear, or from excessive urinary secretion, as in diabetes or in the first stage of contracted kidney. Frequent micturition exists in obstruction by enlarged prostate and in atony of the bladder walls. Hypersecretion of urine plus bladder intolerance is known as "nervousness of the bladder," and is found in hysteria. Frequency of micturition increased by *movement* is observed in stone and tumor of the bladder. Nocturnal frequency of micturition is present in cases of enlarged prostate and atony of the muscular walls of the bladder. Frequency of micturition with diminution of the diameter of the stream suggests a constriction of the urethral canal; frequency of micturition with diminished projectile force suggests a posterior stricture, enlarged prostate, or bladder atony. Slowness of micturition hints at enlarged prostate, atony, or urethral stricture.

*Sir Henry Thompson's diagnostic questions* are as follows:

"1. Have you any, and, if so, what, frequency in passing water? Is frequency more manifest during the night or the day? Is frequency more manifest during motion or rest? Does any other circumstance affect it?

"2. Is there pain on passing urine, and, if so, is it before, during, or after the act? What is its character—acute, smarting, dull, transitory, or continuous? What is its seat? Is it felt at other times, and is it produced or intensified by sudden movements?

"3. What is the character of the stream? Is it small or large; twisted or irregular; strong or weak; continuous, remitting, or intermitting? Does it come by the meatus, or partly or entirely through fistulæ?

"4. Is the character of the urine altered? What is its appearance, color, odor, reaction, and specific gravity? Is it clear or turbid, and, if turbid, is it so at the time of passing? Does it vary in quantity? Are the normal constituents increased or diminished? Does it contain abnormal elements, as albumin or sugar? What inorganic deposits are found? What organic materials are met with?

"5. Has the urine ever contained blood? If so, was the color brown or

bright red; were the blood and urine thoroughly mixed; was the blood passed at the end or at the beginning of micturition, or did it come only with the last drops of urine; or was it passed independently of micturition?

"6. Inquire as to pain in the back, loins, and hips, permanent or transitory, and for the occurrence of severe paroxysms of pain in these regions."

**The Determination of the Excretory Capacity of the Kidneys in Health and in Disease.—The Phloridzin Test.**—This test is made with comparative ease and may aid the surgeon in determining whether he is justified in performing some operation of convenience. It enables him to estimate with a fair amount of accuracy the capacity for elimination possessed by the kidneys. The test depends on the fact that the healthy epithelium of the glomeruli and tubes, when stimulated to activity by phloridzin, forms sugar from that drug and thus produces temporary glycosuria. When the epithelium is diseased, little or no glycosuria occurs. The test is applied as follows: The dose is about 5 to 10 mg. of phloridzin, according to the body-weight of the patient. It is administered hypodermatically, the bladder having been emptied beforehand. If the eliminating powers of the kidney are at a healthy level, sugar should appear in the urine within half an hour of the injection. If at the end of this time only a small amount of sugar can be detected, one may assume that the kidneys are affected; and if no sugar can be found, a serious renal disease may be assumed to exist.

The actual standard that is to be considered as the normal amount of sugar which should be eliminated after the administration of phloridzin is a matter of some uncertainty. It is usually estimated at 0.3 per cent., a less amount of sugar than this being taken as an evidence of renal difficulty (Watson and Bailey, in "Report of Boston City Hospital for 1902"). The sugar is separated from the phloridzin in the epithelium of the glomeruli and tubules of the cortex of the kidney. The drug seems to be entirely harmless.

It is because phloridzin is acted upon by the kidney epithelium that this test is better than the methylene-blue test. The latter does not really measure the excretory power of the kidney epithelium; it merely shows to what degree the kidney is permeable in the mechanical sense. Personally, I should not be disposed to set aside older and more thorough methods of urinary analysis for the phloridzin test, although I believe that it has a range of distinct usefulness.

**The Methylene-blue Test** (*The Method of Achard and Castaign*).—When methylene-blue is injected hypodermatically it normally is changed into a chromogen, appears in the urine within half an hour, and disappears in from thirty-six to forty-eight hours. If the blue color is not manifest in the urine for an hour or more, there is impairment of renal permeability. Only 50 per cent. of the amount ingested is removed by the kidneys. Accuracy in the test is not possible unless the amount of the methylene-blue actually passing into the urine in a given time is determined. The dose given hypodermatically is 0.05 gm. in 1 c.c. of sterile water. The test is unreliable and the blue color may appear in the urine in half an hour in some cases of marked kidney disease. At its best the test only indicates the freedom of mechanical renal permeability.

**The Indigo-carmin Test.**—This drug should be largely excreted in a few minutes. It colors the urine. It is an unreliable method. It will show functional failure, but there may be no functional failure even in organic disease. In many cases the color appears as quickly in urine from a diseased kidney as in urine from the sound organ.

This method enables us to recognize functional incapacity. By it we may forecast uremia. If the urine is collected from each ureter separately we can tell the capacity of each kidney. In this test, if color does not appear until

after twenty-five minutes, and if during the first hour less than 30 per cent. of the amount introduced is excreted, a contemplated surgical operation should be postponed or abandoned.

**The Phenolsulphonephthalein Test.**—This is the most reliable one. The drug is not toxic. It is given in an alkaline solution. A hypodermatic injection of 6 mg. is administered. It appears in the urine when the kidneys are normal in from six to twelve minutes, and from 40 to 60 per cent. of the amount given is excreted during the first hour. It colors urine red and a quantitative estimation can be made by means of a colorimeter. When the color first appears, and how much of the drug is excreted in the first hour, must be noted.

**Cryoscopy** (*Korânyi's Method*).—Cryoscopy is the determination of the freezing-point of a liquid and the comparison of this with the freezing-point of distilled water. It is applied particularly to blood and urine. This method is complex and difficult of application, requires a considerable amount of fluid, and is not regarded as very valuable. The freezing-point of a fluid depends upon the number of molecules it contains. The freezing-point goes hand in hand with molecular concentration—great concentration gives a low freezing-point; little concentration, a high freezing-point. Cryoscopy of the blood and urine is used to determine the adequacy of renal activity. Normal blood freezes at about —0.56° or —0.57° C. Healthy urine freezes between —0.9° and —2° C. In renal inadequacy the freezing-point of the blood is lower than normal and the freezing-point of the urine is higher. It is held that surgical operation is contra-indicated if there is such a degree of renal inactivity that the freezing-point of the blood is at or below —0.6° C. and if the freezing-point of the urine is at or above 1° C. The urine is obtained from each kidney separately and is compared with the blood's molecular composition.

## Diseases and Injuries of the Kidney and Ureter

**Tumors of the Kidney.**—Tumors, innocent or malignant, may arise in the kidney. Among the innocent tumors are fibroma, lipoma, angioma, and adenoma. Hypernephroma of the kidney arises from fragments of adrenal tissue included in the kidney. Hypernephromata were thought to be renal lipomata until 1883, when Grawitz showed they contained adrenal elements ("Virchow's Archiv.," xciii). The name "hypernephroma" was suggested by Birch-Hirschfeld in 1896. A hypernephroma may arise directly from the suprarenal gland or it may arise from an adrenal "rest" or aberrant gland. Such rests may be met with in the substance of the kidney, under the renal capsule, in the perirenal tissue, in the testicle, the ovary, the liver, the inguinal canal, the mesentery, among the spermatic vessels, in the broad ligament, in the renal plexus, or in the solar plexus (W. W. Keen). The term hypernephroma is applied to any growth which arises from adrenal cells "whether the growth be adenoma, carcinoma, or sarcoma in type" (Duffield, in "N. Y. Med. Jour.," May 1, 1909). The tissue of a hypernephroma is identical with the adrenal gland, and it contains fat and glycogen. The exact nature of such a tumor is unsettled. It is probably an adenoma, but some consider it to be a sarcoma and others a carcinoma. Some tumors give no evidence of malignancy; some are very malignant. A malignant hypernephroma grows rather rapidly, eventually attains a large size, and is sometimes painful. A patient in the Philadelphia Hospital from whom I removed a hypernephroma complained of tenderness in the left side and occasional attacks like renal colic during which he passed bloody urine. The tumor could be easily palpated in the left loin. The kidney was removed and resembled a huge kidney of nearly normal shape, but nodular in outline. Dr. Coplin found it to be hypernephroma. In this case there was no increase of arterial tension. The patient died. Another case

was a woman of forty-five who was brought to the Jefferson Hospital. She had suffered from pain in the loin for months. It was paroxysmal, but lacked the radiation of renal colic. Hematuria appeared long after the pain had begun. It was persistent, but small in amount. Palpation detected a tumor and the *x*-rays showed enlarged kidney. There was no increase of arterial tension. Recovery followed nephrectomy. In a man, aged thirty-four, in the Philadelphia Hospital there were attacks of severe pain referred to the groin, testicle, and loin. The bleeding was profuse. A mass was palpable in the right upper abdomen and loin. Examination of blood showed hemolysis, but there was no hypertension. The growth was exposed in the loin. Hemorrhage was so violent that it became necessary to open the abdomen and ligate the renal pedicle. The kidney was removed and the patient recovered.

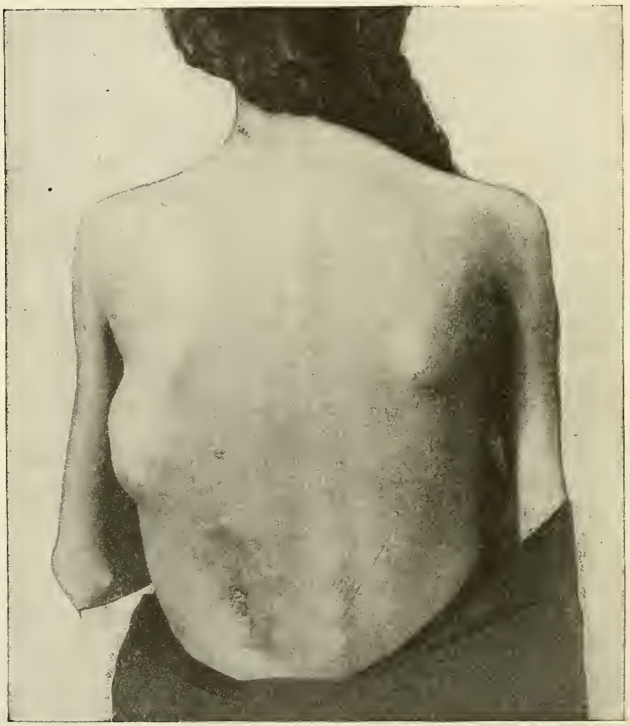


Fig. 849.—Sarcoma of kidney with metastasis (Horwitz).

Very malignant cases have proved fatal within six weeks after symptoms were observed. Some patients have lived three years. A hypernephroma of malignant nature involves adjacent structures, and gives rise, after a time, to metastases, particularly by way of the blood. The bones are most liable to metastatic deposit, but such deposits may occur in the lungs, liver, and other regions. In a case upon which I operated for a supposed adenomatous goiter the condition was really metastatic hypernephroma. Hypernephromata are infinitely more common in the kidney than anywhere else. They tend particularly to occur in middle life. Sarcoma or carcinoma may arise in the kidney. Sarcoma is most common in the young, and may reach an enormous size (Fig. 849). A malignant tumor of the kidney produces hematuria, the urine often containing blood-casts of the ureter, kidney, and pelvis, and sometimes, though rarely, characteristic cells. Pain is often present in the loin and thigh,

and there may be colic-like attacks when clots are passing through the ureter. Emaciation is rapid and pronounced. A tumor can usually be palpated. Pyelography with a 10 per cent. solution of collargol may aid in the diagnosis (see page 1309). The only possible treatment for a malignant growth is early nephrectomy. In some few cases an innocent tumor can be removed by a partial nephrectomy. A malignant tumor requires a complete nephrectomy. In making a diagnosis of renal tumor use the cystoscope. If blood is coming from above the bladder, note if it is from one or from both ureters. Blood from both would contra-indicate nephrectomy. Before removing a kidney it is necessary to be sure that the patient is possessed of two kidneys. Note if urine flows from each ureter, or, if uncertain, catheterize the ureters.

**Nephroptosis, Prolapse of the Kidney, or Mobile Kidney.**—There are two forms of this condition: (1) *Movable kidney*, which is an organ freely moving back of the peritoneum, either within the cavity of its fibrofatty capsule or entirely without its capsule (this condition is acquired); and (2) *floating* or *wandering kidney*, an organ having a mesonephron and lying within the peritoneal cavity (this rare condition is always congenital). Keen states that there may be drawn a clear theoretical distinction between movable and floating kidney, but practically there is no rigid line of demarcation, as a movable kidney may have as large a range of movement as a floating kidney. The kidney is normally somewhat mobile, and nephroptosis is considered to exist only when the range of movement exceeds distinctly what is normal. Normally, on inspiration the kidney descends about ½ inch. It is seldom that a normal kidney can be palpated in men, but in most women the right kidney can be palpated, and in some women the left organ can also be felt. Harris ("Jour. Amer. Med. Assoc.," June 1, 1901) describes three degrees of movable kidney. In cases of the first degree, one-half of the organ can be distinctly grasped and the kidney can be made to recede. In cases of the second degree both hands can be brought together above the kidney. In cases of the third degree the kidney has descended as low as the pelvic brim or has moved to or beyond the umbilicus. The organ may drop below the brim of the pelvis, may cross the vertebral column, or may reach the anterior abdominal wall. When a movable kidney becomes fixed in an abnormal situation, the organ is spoken of as *dislocated*.

Women suffer from movable kidney more often than do men. Küster estimates that 4.41 per cent. of women examined in general surgical practice have movable kidney. Edebohls finds it in 20 per cent., and Harris in 56 per cent., of cases in gynecological practice. In about one-half of the cases it gives rise to little or no trouble. A movable kidney is found in the great majority of cases upon the right side. In many cases it is bilateral, the right kidney being usually the most mobile. Splanchnoptosis may be associated with acquired nephroptosis. Floating kidney is always congenital. Movability of the kidney is occasionally, but rarely, found in children, though congenital cases occasionally occur. In a congenital case there is not splanchnoptosis. Tuffier has reported 3 cases in children six, nine, and ten years of age respectively, and J. Cromby reported 18 cases of floating kidney in children, the youngest patient being three months of age (quoted by Harris, Ibid.). Among the assigned causes of the movable condition are to be named traumatism; strains; abdominal-wall laxity from pregnancy, removal of a tumor, or tapping for ascites; absorption of peritoneal fat from wasting disease (Edebohls); tight lacing; uterine displacements; and enteroptosis leading to traction on the transverse mesocolon. The condition is certainly often associated with ptosis of the other abdominal viscera (enteroptosis, gastroptosis, etc.).

Traumatism is rarely the immediate and essential cause of a true movable

kidney. In some cases people assert that pain began immediately after a blow, an attack of coughing, violent vomiting, lifting, straining at stool, a fall, or in parturition. In such cases the kidney may have been mobile before the accident. Again, pain is not proof of the inauguration of movability. It is probable, however, that traumatism may loosen the kidney and that mobility may subsequently develop. Gutterbock says that a kidney in normal relations cannot be rendered mobile by a simple fall or a trivial force. Loosening can be induced only by rupturing surrounding tissues; and if this happens, symptoms of a distinct nature will indicate the seat of injury. Becher and Lennhoff claimed that there is a connection between movability of the kidney and the length and breadth of the body. They have laid down a formula, viz.: Measure the distance from the suprasternal notch to the crest of the pubes. Divide this by the smallest circumference of the abdomen. Multiply the product by 100. The result is the abdominal index. If the index is greater than 75 there is a tendency to movable kidney. If it is less than 75 there is no such tendency. Harris makes out a strong case for the view that the condition is due to the relation existing between the location of the kidney and the body form. He divides the body into three zones: The upper zone contains the lungs and heart. The middle contains the liver, stomach, spleen, pancreas, and the greater part of each kidney. The lower contains the intestinal canal and the lesser part of each kidney. When there is a naturally small or a diminished capacity of the middle zone, the kidney is displaced downward. The right kidney is pressed upon by the heavy liver, which drives it down; the left kidney is pressed upon by the comparatively small spleen. Hence movable kidney is more common on the right side than on the left. The upper pole of the kidney is first pushed forward and then the entire organ descends (M. L. Harris, in "Jour. Amer. Med. Assoc.," June 1, 1901). Harris maintains that the amount of mobility depends upon the degree of contraction of the middle zone and upon internal traumatisms (lifting, straining, coughing, etc.).

**Symptoms of Both Forms.**—There may be no discomfort whatever, or the patient may be a confirmed invalid. The usual symptoms are epigastric pain (just to the left of the middle line), which disappears when the kidney is replaced, dragging pain in the loin, and paroxysms like nephritic colic. Sudden attacks of violent pain in the kidney or stomach may occur—attacks which are accompanied by nausea, vomiting, great weakness or collapse, vertigo, chills, and subsequently elevated temperature (*Dietl's crises*). Dietl's crises are due to kinking or twisting of the ureter or renal vessels or to inflammation of the kidney. They may be caused by physical exertion or indiscretion in diet and may be followed by hydronephrosis or strangulation of the renal vessels. A few years ago I operated upon a man suffering from a violent and prolonged crisis and found a twist of the vessels and ureter. In a Dietl's crisis there is congestion or strangulation or both. An incomplete or temporary twist of the renal pedicle may induce simply pain in the abdomen and loin, hematuria, albuminuria, and cylindruria.

The question as to whether or not abdominal pain is due to movable kidney is sometimes in doubt. The localization of the pain may lead us to suspect appendicitis. Some surgeons think that catarrhal appendicitis is often associated with movable kidney, but I do not think the association is common. "Dr. Kelly has shown us how to solve this doubtful question between appendicular pain and the pain of movable kidney. He catheterizes each ureter separately, and introduces into each catheter as much fluid as the renal pelvis will hold without causing pain. He then measures this fluid from each side, and determines whether it is in excess of an estimated average. If it is in excess, he is sure that dilatation has begun. He then injects the

kidney again, with the deliberate purpose of producing pain; and if the patient recognizes this pain due to the distention as of the same character and in the same position as that which he has previously felt, Dr. Kelly assumes that the pain has been due to the kidney, and not to the appendix, and recommends an operation to fix the kidney" (the author, in "New York Med. Jour.," August 4, 1906). Usually in a case of movable kidney there is a sense of a moving body in the abdomen, and the patient has aggravated indigestion, often accompanied by vomiting. Constipation is the rule, and violent attacks of cardiac palpitation are common. Most subjects of kidney mobility are extremely nervous—many of them hysterical or hypochondriacal. Persistent vasomotor paresis causes cold hands and feet and often albuminuria. Temporary jaundice is not uncommon. There is frequently irritability of the bladder. Vertigo and insomnia are present in many cases. The patient cannot sleep when lying on the sound side (Goelet). In women the sexual organs are almost invariably deranged, and menstruation aggravates the pain and discomfort. All the symptoms are intensified by exertion and are modified by rest. The urine is normal except after violent exercise, when it may contain blood. Splanchnoptosis may also exist, and if it does, the pulsations of the abdominal aorta are strongly noticeable because that structure is bared by gastroptosis. The proof of the existence of movable kidney is the finding of a mass, movable on respiration, change of position, and palpation, shaped like that organ, pressure upon which occasions no sensation or causes pain or a sickening feeling. A "lumbar recess" (Morris) may sometimes be found, and percussion over the loin gives resonance. In some cases a movable kidney can be readily detected when the patient stands up, but is difficult to find when he is recumbent. Franks's method of examination is very satisfactory in most cases (Fig. 850). The patient is placed recumbent. If dealing with a right kidney, the surgeon stands to the right side and pushes four fingers of his left hand in the loin below the twelfth rib, and rests the thumb lightly in front just below the ribs. The patient takes a full breath and holds it a moment, and just before he empties his lungs the surgeon presses his thumb up deeply below the ribs. During expiration the thumb follows the liver, and the fingers press toward the front. If with the right hand the kidney can be felt entirely below the left hand, the case is one of movable kidney. If such a condition is detected, press hard with the right hand, and gradually loosen the grasp of the left hand, and the kidney will slip between the fingers and ascend. A normally mobile kidney descends so that its lower end can be felt, but it moves back during expiration.[1] Goelet uses Kendal Franks's method of palpation, but has the patient stand, with the weight resting on the leg of the sound side and with the leg of the impaired side slightly flexed and resting on the toes. The body leans a little forward. A movable kidney must not be mistaken for a distended gall-blad-

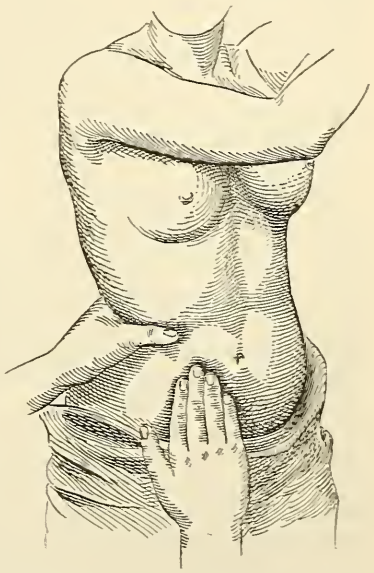


Fig. 850.—A. H. Goelet's method of palpation for the detection of a prolapsed kidney.

[1] "Brit. Med. Jour.," Oct. 12, 1895.

der, a tumor of the mesentery, stomach, or omentum, a phantom tumor, an ovarian tumor, or a cancer of the pancreas. A distended gall-bladder can be pushed upward, but not backward, and not downward unless the liver is movable; it is extremely tender, and cannot be pushed out of reach. A kidney can be pushed upward and backward—in fact, in all directions. An enlarged gall-bladder can always be palpated. A movable kidney which is not enlarged can be felt at times and not at others (Henry Morris). A movable kidney may pass between the examiner's fingers, and if pushed into the loin, it tends to remain; but if a distended gall-bladder is pushed into the loin, it springs out as soon as pressure is relaxed (Henry Morris). The *x*-ray study of the kidney pelvis after injection with collargol is known as *pyelography* (see page 1310). The *x*-ray picture of a kidney so injected is a pyelogram. This method is of the highest diagnostic value. It will always show a displaced kidney, and it indicates hydronephrosis. One picture should be taken with the patient standing, another with the patient recumbent. It is important to remember that in about one-half of the cases of movable right kidney the left kidney is also movable, but to a less degree. Appendicitis is thought by some to be more frequent in individuals with movable right kidney than in those free from renal mobility. Sometimes a movable kidney endangers life, rupture of the kidney, twisting or rupture of the ureter, or strangulation of the renal vessels occurring, the ultimate cause of death being albuminuria, uremia, or hydronephrosis.

**Treatment.**—Mobile kidney is treated as follows: If the kidney is but slightly mobile and there are no local symptoms, the treatment should be non-operative: (1) The *rest treatment of S. Weir Mitchell* may be tried; it often markedly mitigates the symptoms, but does not seem to cure. (2) *Mechanical support* should always be tried. The most satisfactory mode of applying it is by the corset recommended by Gallant ("Am. Jour. Obstet.," July, 1901). This corset is long and straight in front, and when applied, fits firmly over the hips and lower abdomen, less firmly at the waist, and least firmly above.

The patient lies down, a pillow being under the buttocks and the knees being drawn up. While in this attitude the corset is put on and it is laced from below up. If the attempt to apply the corset develops tenderness, keep the patient at rest in bed until it can be applied without pain. In some cases conservative treatment is not indicated; in others it fails.

In every case of very movable kidney and in some cases in which movability is not great operation is indicated.

"In a case in which the kidney exhibits trivial movability, but in which the range of mobility is found to be gradually and certainly increasing, or in any case of kidney movability in which there are distinct local symptoms, operation is indicated. The distinct local symptoms mean the beginning of actual harm to the kidney, and the progressive increase of movability means the ultimate attainment of a wide range of movement. A kidney which is widely movable may at any time twist upon the ureter and the renal vessels; and it is certain to suffer from partial or slight twists, probably many times repeated in the twenty-four hours, even if a severe twist does not occur. A deduction from the foregoing statements is that a patient suffering with nephroptosis, even when the mobility is slight, should be examined at regular intervals, to note whether the area of movement is extending, or whether local symptoms have arisen. Three local symptoms that should be regarded as indications for operation are severe pain in the renal region, distinct tenderness of the kidney, and enlargement of the kidney" (the author, in "New York Med. Jour.," August 4, 1906). Billington ("Brit. Med. Jour.," May 1, 1909) formulates the following indications for operation:

1. When renal pain is so severe or persistent as to cause serious inconvenience. The ordinary dragging pain in the loin is not an indication. Billington refers to severe pain due to perirenal inflammation, ureteral obstruction, or impeded venous return.

2. When there are harassing and depressing gastric and colonic troubles (gross lesions being absent).

3. Cases of spinal and cerebral neurasthenia.

4. Cases of lunacy. Personally I do not operate on groups 3 and 4 unless there are signs of grave renal disaster.

The usual operation chosen will be nephropexy, very seldom nephrectomy. (1) *Nephropexy* is the operation employed in most instances (see page 1296). It is the author's experience that if the patient has had marked nervous symptoms for a long time, nephropexy will rarely cause them to pass away permanently, even though the kidney remains firmly anchored. (2) *Nephrectomy* is necessary only in very rare cases; it may be done for dislocated kidney, when grave kidney disease exists, or when nephropexy has failed in a case of great severity.

In many cases of this trouble no operation should be performed, the use of Gallant's corset securing, perhaps, decided or complete relief. I do not operate if the kidney is only slightly movable and if there are no local symptoms or if there are merely the general symptoms of hysteria. If the mobility is slight and the hysterical and neurotic condition is pronounced, anchoring the kidney will not cure the nervous condition. In these nervous cases, associated with prolapse of the kidney, there is usually also prolapse of the other abdominal viscera; and both kidneys are, as a rule, movable, the right, however, in most cases being decidedly more movable than the left.

If there is but slight mobility of the kidney, but the range of movement is, week by week and month by month, increasing, or if we find a case of movable kidney in which there are distinct symptoms, an operation should be performed. The existence of definite local symptoms means beginning harm to the kidney; and if we find the area of movement gradually increasing, we know that eventually it will become extensive. Any widely movable kidney may twist the ureter and the renal vessels, producing serious trouble or even disaster, and consequently should be fixed by operation. Even if a severe twist does not take place, the kidney is bound to suffer from partial or slight twists. Such kidneys will eventually become hydronephrotic. The meaning of the term "slight mobility" is indicated on a previous page (see page 1275).

One is not unusually in doubt in cases of movable kidney whether a pain indicates local trouble with the kidney or catarrhal appendicitis, because the pain may be located in the appendix region. Kelly, of Johns Hopkins Hospital, has shown how to solve this problem (see page 1276).

There are many operations for movable kidney. In all of them the kidney is exposed in the loin. Some make a vertical and some an oblique incision. Edebohls makes a vertical incision, forces the kidney out of the wound, incises the fibrous capsule longitudinally, turns a cuff down on each side, and applies sutures. These sutures traverse the kidney substance and the fold of capsule on each side. The upper suture catches the periosteum of the last rib; the other sutures catch the lumbar fascia. Drainage is not required, and the suture material employed is kangaroo-tendon or chromicized catgut.

Some surgeons simply pass sutures through the uncut capsule and the kidney substance and thus fasten the kidney to the lumbar fascia. Others split the capsule and pass sutures through the edge of the capsule and the wound edges, but not through the kidney substance.

To promise success, an operation ought to restore the kidney nearly to its normal position and fix it permanently in place. It is undesirable to inflict

damage on the kidney itself, and I do not believe in any operation that seeks to obtain fixation by passing sutures through the kidney substance. In cases in which decapsulation is performed the kidney will grow fast without any special method of suturing.

Most of the operations suggested do not place the kidney sufficiently high up to get it into a fair position. Kelly's operation gets it higher than most of them, and Goelet's operation gets it well into place. In many of the suture operations the sutures are placed in the convex surface of the kidney or the kidney capsule, and on fixing the kidney by tying the sutures there is a permanent quarter twist of the ureter—a condition that may be responsible for great pain. This may be obviated entirely by the ingenious method of Goelet ("Annals of Surgery," Dec., 1903). I believe, however, that the suture operations which do lift the kidney well up toward its proper place and in which the sutures are applied on the posterior surface and not the convexity, tilt the upper pole forward into a permanent and perhaps disastrous position. Such operations lift the kidney from below its midline and thus fix the lower half of the organ, but leave the upper half unfixed. I believe, too, that in many cases in which kidneys have been sutured they get loose again and that the best operation, after all, is that by the use of slings of iodoform gauze (see page 1296).

**Injuries of the Kidney.**—**Laceration** or **rupture** is caused by falls and by blows upon the back or the belly.

*Symptoms.*—In some cases the parenchymatous structure is torn, but the capsule is not torn, and in consequence urine and blood are not extravasated into the perirenal connective tissue or into the peritoneal cavity. In other cases the parenchyma and capsule are both torn and urine and blood are extravasated into the perirenal tissues, the peritoneal cavity, or both of these regions. The laceration may be trivial, may be considerable, or may tear the kidney apart. The symptoms depend on the gravity of the injury. A slight tear without involvement of the capsule may produce practically no symptoms at all. A more severe injury produces shock, and, if profuse bleeding occurs, the general symptoms of hemorrhage. In intraperitoneal rupture there is profuse and usually fatal hemorrhage. In laceration of the kidney there are severe pain in the loin, which shoots into the testicle, and lumbar tenderness. If there is considerable perirenal bleeding the loin will be full and dull on percussion, and if the hemorrhage is large, a palpable mass will form after a time and after some days the skin will become discolored. There is usually frequent and painful micturition, and in some cases suppression of urine. Hematuria occurs in renal laceration unless the rupture was intraperitoneal or the ureter was torn. If the rupture was intraperitoneal there are evidences of profuse internal hemorrhage, abdominal rigidity, etc. (Daniel N. Eisendrath, "Jour. Amer. Med. Assoc.," Oct. 25, 1902). It is important to remember that hematuria can arise from simple renal contusion, and that even severe kidney damage does not of necessity cause bloody urine. If there is hematuria, the use of the cystoscope, catheterization of the ureters, or the employment of Harris's segregator will demonstrate from which kidney the blood comes. A kidney laceration may be followed by secondary hemorrhage, perirenal suppuration, hydronephrosis, or pyonephrosis. The force of the injury may have caused kidney displacement.

*Treatment.*—In an intraperitoneal rupture laparotomy should be performed. As a rule, nephrectomy is necessary, but it may be possible to arrest hemorrhage by packing. If the shock is pronounced and if there is increasing fulness in the loin, whether hematuria exists or not, or if blood comes profusely from the ureter, whether or not there is much shock or lumbar fulness, make an exploratory lumbar incision and stop the bleeding by

packing or by a purse-string suture (Figs. 851, 852), or, if necessary, perform partial, or even complete, nephrectomy. Ordinarily, after a kidney injury, when there is not great shock, increasing lumbar swelling, or severe hematuria, treat by rest in bed and by feeding with liquid food or by nutritive enemata to prevent vomiting. Opium, tannic acid, or gallic acid may be used. Apply ice-bags to the loin and the side of the abdomen, and after bleeding ceases strap the loin and apply a binder. If large blood-clots in the bladder cause pain or retention of urine, introduce a catheter and inject the bladder with boric acid, or use the tube and evacuator of a Bigelow apparatus. If this procedure fails, open the bladder by a suprapubic incision and drain.

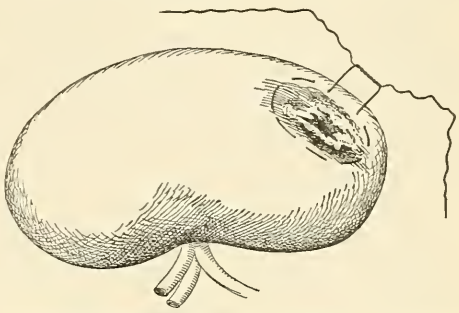


Fig. 851.—Purse-string suture applied to a perforation (after Schacher).

*Results of Operation.*—Up to 1894 there had never been a case of intraperitoneal rupture operated upon. During the following seven years 6 were operated upon and all recovered (Daniel N. Eisendrath, "Jour. Amer. Med. Assoc.," Oct. 25, 1902). Küster collected 47 cases of nephrectomy, and 83 per cent. recovered. Keen estimates the mortality of primary nephrectomy for rupture at 20 per cent., and of secondary nephrectomy at 38.5 per cent. Without operation intraperitoneal rupture is inevitably fatal. Of extraperitoneal ruptures, 70 per cent. recover without operation (Eisendrath). Francis S. Watson ("Boston Med. and Surg. Jour.," July 16, 1903) has collected 660 cases of subparietal injury of the kidney. The following statistics are of interest: Treated expectantly: 273 cases with 81 deaths, a mortality of 29.6 per cent. Treated by operations other than nephrectomy: 99 cases with 7 deaths, a mortality of 7.7 per cent. Treated by nephrectomy: 115 cases with 25 deaths, a mortality of 21.7 per cent.

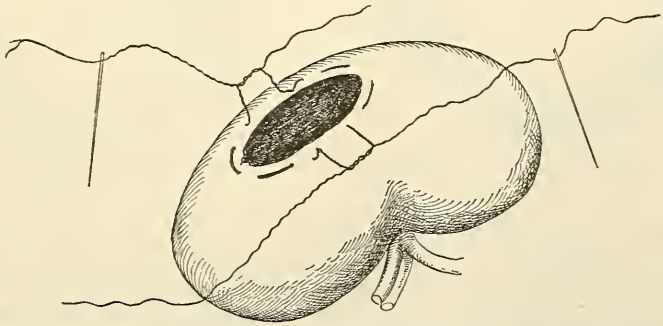


Fig. 852.—Showing the application of a double purse-string suture for the arrest of hemorrhage in large wound (after Schacher).

**Perforating wounds of the kidney,** if purely posterior, do not involve the peritoneum; if anterior, they do. The *symptoms* are escape of blood and urine by the wound; hematuria is usual, but not invariable; pain as in rupture; the patient may be unable to micturate; and nausea, vomiting, and constitutional signs of hemorrhage exist. Traumatic peritonitis, perinephric abscess, or general sepsis may ensue. Confirm the diagnosis by exploration

with the finger after operative exposure. Extraperitoneal injuries give a good, and intraperitoneal a bad, prognosis.

*Treatment.*—If the wound of the kidney is extraperitoneal, enlarge the lumbar wound to permit of drainage, and arrest hemorrhage by packing and hot water or by a purse-string suture (Figs. 851, 852).

*Suture of the Kidney.*—The tendency of any suture material to cut through the kidney structure is great. The following simple procedure greatly lessens this danger: Cut the ordinary catgut roll into three or four parts, as shown in Fig. 853. Slip beneath the exposed loops of the purse-string suture, as shown in Fig. 854, a section of the catgut roll consisting of a series of short stiff strands. These permit fairly firm tying of the suture-ligature without cutting of the kidney structure by the suture. In nephrotomy the same procedure can be used to hold the kidney wound together.

Asepticize the wound, insert a drainage-tube down to the kidney, dress with bichlorid gauze and make frequent changes of dressings, keep the patient in bed and on a low diet, and give gallic acid and opium. In some cases nephrectomy, partal or complete, will be required. In intraperitoneal wounds perform an abdominal section and, as a rule, remove the damaged organ (see Nephrectomy).

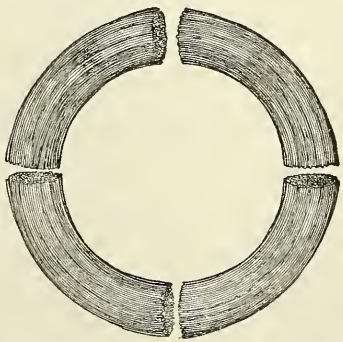


Fig. 853.—Catgut ring cut into quarters to be inserted under the suture as shown in Fig. 854.

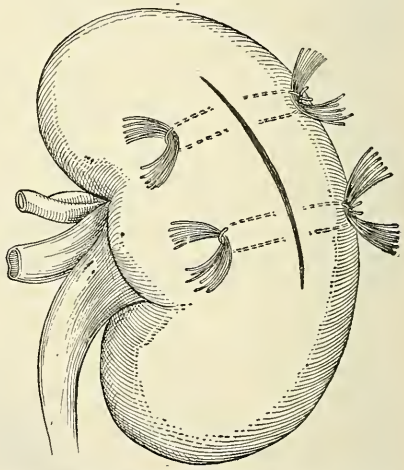


Fig. 854.—Stellwagen's suture of kidney to prevent cutting after tying.

**Wounds of the Ureters.**—Rupture from external violence is an extremely rare accident. Eisendrath ("Jour. Amer. Med. Assoc.," Oct. 25, 1902) found only 3 undoubted cases on record. A rupture or wound from accidental violence is almost invariably associated with other serious injuries. The ureter may be wounded accidentally by the surgeon during the performance of an abdominal operation, or it may be wounded intentionally, as in Morris's case, in which a malignant growth was incorporated with the ureter. There is particular danger of injuring the ureter in operations upon intraligamentary growths, because the ureter is displaced and often resembles an adhesion. The rule of surgery is that when working about the ureter the surgeon neither clamps nor cuts any structure without a careful preliminary examination. Rupture causes severe shock and extravasation of urine around the kidney or into the peritoneal cavity. In extraperitoneal rupture a palpable mass forms in the loin. When the ureter is divided in an operation, a flow of urine is seen.

**Treatment.**—The upper three-fourths of the ureter can be reached by an extraperitoneal incision, which is a prolongation of the incision for lumbar nephrectomy, running from the twelfth rib downward, and forward to 1 inch

anterior to the anterior superior spine of the ilium, and then parallel to Poupart's ligament until a point is reached above its middle (Fenger). Israel's incision begins at the anterior edge of the erector spinæ mass, one finger's length below the twelfth rib, is taken forward parallel with the rib until it reaches the line of the rib's tip, and is then carried toward the middle of Poupart's ligament until the line for ligation of the common iliac artery is reached, and is then taken toward the middle line as far as the outer border of the rectus muscle. The lower one-fourth of the ureter can be reached by abdominal section, by sacral resection, or by an incision like that for extraperitoneal ligation of the iliac vessels. The best operation to reach the lower ureter is Gibson's (see page 1299). If it seems probable that the ureter is wounded or ruptured, explore, and if this is found to be the case, endeavor to restore the continuity of the tube. A longitudinal cut can be sutured with fine catgut. If the ureter is cut across near the bladder, implant the proximal end into the bladder and ligate the distal end (Van Hook, Penrose, Kelly). If it is cut above the bladder portion, perform lateral implantation by Van Hook's method (see page 1300).

A longitudinal wound of the ureter inflicted during an abdominal operation should be sutured, but if the duct cannot be readily reached, simply make a posterior incision and drain with rubber tissue, as the longitudinal wound will heal by granulation if no sutures are inserted (Van Hook). In a case of transverse division perform uretero-ureterostomy or vesical implantation; or, if neither of these methods is feasible, make a urinary fistula in the loin or perform nephrectomy.

**Renal Calculus.**—A stone in the kidney is formed by the precipitation of urinary salts into the renal epithelial cells and the gluing together of these salts and cells by material from mucus or blood-clot, this mass serving as a nucleus on which accretion takes place. Most calculi escape when small, as *gravel*. The **cause** is a highly acid urine, which induces catarrh of the renal tubes. Such high concentration of urine is favored by a sedentary life, by the ingestion of much alcohol or nitrogenous food, by constipation, by an inactive skin, and by a torpid liver. The slaves of poverty are particularly liable to calculi because of the use of unsuitable foods and the formation of great amounts of nitrogenous waste. Males suffer more often than do females; certain locations favor the development of the malady, and a family tendency sometimes exists.

The **symptoms** of stone in the kidney may not appear for years after the stone forms, but generally they are manifested early. There may be no pain. There had been none in 13 cases out of 23 which came to autopsy and were reported by Clark. Usually there is pain; the severity of the pain depending upon the roughness and movability rather than upon the size of the stone. A fixed stone in the kidney and a smooth stone in the pelvis may cause little or no pain. A rough stone in the pelvis causes severe pain. The patient usually complains of pain in the loin, and sometimes of pain in the iliac region. Deep percussion over the kidney causes pain in the loin, even when pressure is painless (Jordan Lloyd's symptom). Pain is aggravated by exercise and pressure, and the kidney is usually enlarged. The urine is often somewhat albuminous, and may from time to time contain blood. Frequency of micturition is noted during the day, but seldom when at rest at night. The urine may be purulent. *Nephritic colic* is due to the washing of a calculus into the orifice of the ureter, which it blocks, tears, or distends. The pain is either sudden or gradual in onset, is fearful in intensity, and runs from the lumbar region down the corresponding thigh and spermatic cord (the testicle being retracted), and into the abdomen and back. There are nausea, vomiting, collapse, sometimes unconsciousness

or convulsions. Frequent attempts at micturition are productive of pain, but of little urine. Rectal tenesmus is common. The urine is often, but by no means always, smoky from blood. Blood may be found by the microscope when it cannot be detected by the naked eye. In rare cases fatal hemorrhage occurs. Blood is present in about one-half the cases. After a time the pain vanishes, the stone having passed into the bladder or having fallen back into the pelvis of the kidney. Slight attacks of colic occur from the passage of small stones or plugs of mucus. A calculus retained in the kidney eventually excites pyelitis, pus appears in the urine, and soreness or pain in the loin exists. Kelly says: Even if pus is found, we are not always sure from which kidney it came. Pain or swelling may point to one side, but we are not sure that the outer

Fig. 855.—Stone in kidney.

organ is not also affected. The cystoscope must be used. Bloody or purulent urine may be seen coming from one ureter. If able to pass the renal catheter into one ureter, attach a syringe, and by making suction draw out any pus which may be present. In renal calculi cases this fluid is apt to contain fragments of uric acid. By using a renal bougie coated with dental wax it may be possible to make scratches on the instrument when it comes in contact with a concretion.[1] When a stone is impacted in the renal pelvis the point of greatest tenderness on pressure is below the last rib, by the edge of the erector spinæ muscle. In septic cases there may be chills and irregular fever, and often there is leukocytosis. In most cases a stone in the kidney or ureter can be

[1] Howard Kelly, in "Med. News," Nov. 30, 1895.

skiagraphed. Nephrolithiasis may cause death by exhaustion, by sepsis, by rupture of a hydronephrosis, or by amyloid degeneration.

**Treatment.**—For the gravel of the uric-acid diathesis use alkalis, especially the liquor potassii citratis, and reduce the amount of nitrogen in the diet to a minimum, at the same time washing out the organs by copious drafts of water. Citrate of lithia, given in the water, is supposed to add to the therapeutic effect. Some surgeons prescribe natural lithia water. Piperazin, in doses of 5 to 8 gr. three times a day, is highly commended by some. Exercise is to be insisted on. When gravel is phosphatic, order strychnin, the mineral acids, and rest at the seaside. When oxalate of lime is found, restrict the diet, use the mineral acids, recommend travel or rest amid new surroundings, and give an occasional course of sodii phosphas, ½ dram three times a day, taken in a natural lithia water. Nephritic colic is relieved by hypodermatic injection of morphin and atropin, a hot bath, diluent drinks, and possibly the inhalation of ether. After an attack watch all the urine passed to see if a stone appears. If one does not soon appear, use the cystoscope, and if a stone is found in the bladder, wash out that viscus with an evacuator. This is very important, as the vesical stone may fail to pass, and if it remains in the bladder it will progressively enlarge. Further, finding it proves the diagnosis of renal colic. If a stone impacts in the ureter, perform the operation of *ureterolithotomy*. The diagnosis of this impaction is in many cases aided by the *x*-rays, but is sometimes possible only after exploratory laparotomy. If the symptoms point to stone in the kidney, always take a skiagraph. If this shows a stone, if medical treatment fails, or has failed, and if the other kidney is not organically diseased, operate. If in doubt in spite of the skiagraph, make an exploratory lumbar incision; feel the surface of the kidney with the finger, sound the inside of the organ with a needle, or open the organ for exploration, and if a stone is detected, incise the kidney and remove the stone. Keen is of the opinion that operation should not be performed if the urea is below 1 per cent. If, after nephrolithotomy, suppression of urine occurs, cut into the other kidney, as in one-half of all cases a stone will be found lodged there. I agree with Brewer ("Med. Record," March 20, 1909) that "a kidney containing one or more calculi, and also the seat of an advanced septic process, should be removed if the opposite organ is healthy. To leave such a kidney is to invite subsequent trouble from recurrence of stone, pyonephrosis, or long-continued sepsis." I agree with him when he says: "It is also often safer to remove a kidney with multiple calculi embedded in its substance than to inflict the trauma necessary to remove them, as alarming primary or secondary hemorrhage is apt to occur." In a case of my own a most persistent postoperative hemorrhage forced me to perform nephrectomy to save life.

**Calculus Impacted in the Ureter.**—A ureteral calculus comes from the kidney, sometimes dropping, but more often being forced, into the tube. A stone may be arrested at any one of the points of constriction. There are three points of constriction in the ureter: one point is about 2 inches below the renal pelvis, another is at the pelvic brim, another is about ½ inch from the bladder orifice of the ureter. The highest point has a diameter of about $\frac{1}{7}$ inch, the middle point a diameter of about ¼ inch, the lower point a diameter of about $\frac{1}{10}$ inch. A small stone may completely block the ureter. A large stone may fail to completely block it because the ureter dilates above, the stone acts as a ball-valve, and urine trickles by.

**Symptoms.**—Attacks of violent pain of the nature of renal colic occur, and not unusually there is a rigor with the attack and fever after it. Such an attack may be followed by hematuria. The urine should be examined microscopically during several days after a colic to see if it contains blood-cells. Tenderness

can be developed at the point of impaction, the point of greatest tenderness being in the loin *below* the level of the kidney or in the iliac region (Perkins). In stone in the ureter pain is not developed by pressure in the loin at the level of the kidney. If a stone partly obstructs the ureter, the urine is pale, of low specific gravity, and free from albumin. Impaction near the bladder causes symptoms similar to stone in the bladder (Jordan Lloyd). These symptoms are frequent micturition, pain at the seat of impaction, pain in the head of the penis, and bloody urine. If a stone is impacted in the lower end of the ureter a finger in the vagina or rectum will find tenderness and perhaps will feel the stone. In a woman, a stone lodged in front of the broad ligament may be felt by a finger in the vagina. Back of this region and up to the pelvic brim a stone may be felt by a finger in the rectum. A cystoscopic examination, in unusual cases, may show a portion of stone projecting from a ureter (Kelly). Impaction near the kidney is accompanied by hematuria and pyuria, lumbar pain, pain radiating into the groin, thigh, or testicle, and retraction of the testicle. These symptoms are identical in character with the symptoms caused by stone in the renal pelvis. Complete obstruction of the ureter causes hydronephrosis. Pyonephrosis results from infection of a hydronephrosis. In some cases a stone acts as a ball-valve, plugs the ureter for a time, during which a lumbar mass develops, and then allows the urine to flow. A copious flow of urine is accompanied by disappearance of the lumbar mass. Complete urinary suppression may follow blocking of a ureter by a calculus. If a ureteral catheter tipped with wax is introduced, a calculus will make distinct scratches upon it (Kelly). The Cunningham catheter (see Fig. 866, *e*) may assist in detecting a stone. It has not been successful in our hands when the stone was impacted more than 6 c.c. above the ureteral outlet. This catheter may also be of assistance in dislodging the stone.

The *x*-rays are very valuable in diagnosis. A pyelographic study of the ureter and pelvis may give material assistance.

**Treatment.**—During a painful paroxysm give morphin and use hot packs. Belladonna is useful by inducing relaxation of spasm. The ozonized oil of turpentine, given in capsules in 10-min. doses, is often valuable. It was employed by the elder Gross. For the pain Bransford Lewis ("Jour. Am. Med. Assoc.," Jan. 29, 1910) catheterizes the ureter and injects into the renal pelvis 20 min. of a 1 per cent. solution of alypin (monohydrochlorid of benzoyl). Alypin is a powder. It is soluble in water and is to be sterilized by boiling for not over five minutes. The attack may terminate and not return, because the calculus passes. If such an attack does pass away, the urine should be examined after every act of micturition to see if the stone is voided from the bladder. After a day or two, if the stone does not appear, use the cystoscope, perhaps catheterize the ureter, and thus discover if the stone is in the bladder or if it is impacted. If the stone is in the bladder, use the Bigelow evacuator to effect removal, or crush the stone by the small forceps of Bransford Lewis or by the forceps of Leo Buerger. The stone must never be allowed to remain, as it will surely enlarge and cause subsequent trouble. If a stone is found impacted in the ureter have the patient drink water freely. Sterile olive oil may be injected into the ureter through a ureteral catheter, or the ureteral orifice may be dilated by a suitable bougie. If the impacted stone is very near the bladder the ureteral orifice may be slit. Simple catheterization of the ureter may be followed by expulsion of the stone. If in spite of these procedures the stone remains impacted in the ureter, the question of operation presents itself. An impacted stone is certainly a peril, but how immediate the danger it is often impossible to say. In some cases stones have remained impacted for many years without doing obvious harm. In other cases the kidney is rapidly destroyed. The stone may pass after having been retained

for a long time, and drinking freely of water favors its expulsion. One of my cases had long had a retained stone, came to the hospital for operation, and passed the stone. Sooner or later a retained stone will lead to disaster and it ought to be removed by operation. It will cause, if retained, thickening or ulceration of the ureter, dilatation of the ureter above it, and kidney trouble. A lodged stone increases gradually in size and other stones may form above it. The extraperitoneal operation is to be chosen in most cases. Even when the stone is impacted below the pelvic brim, it is usually better to do the extraperitoneal operation. (See Ureterolithotomy.)

**Abscess of the kidney** may be caused by traumatism, by calculus, by stricture of the ureter, by disease of the bladder, by the union of miliary abscesses (tuberculosis), by pyemia, and by certain parasites.

The **symptoms** are pus in the urine (this is usual, but not invariable), hematuria in traumatic cases, and pain running into the groin. The urine in most cases is alkaline. Constitutional symptoms of suppuration exist, the fever being far higher than that generally met with in renal tuberculosis. The bladder should be examined with a cystoscope to determine that the turbid urine flows from the ureter and to identify the diseased side. It is well, if possible, to catheterize the ureters.

The **treatment** in the early stage is rest, morphin, purgation, anodynes, and hot fomentations. In some cases repeated lavage through the ureter may cure. When the diagnosis is clear, and lavage fails or is not thought desirable, incise the loin, open the kidney and stitch it to the abdominal wall, or, if the organ be badly damaged, remove it.

**Pyelitis** and **pyelonephritis,** which usually affect only one gland, are caused by urethral stricture, by stopping of the ureter by blood-clot, by vesical paralysis, by stone in the bladder or in the kidney, by enlargement of the prostate gland, by ascending tuberculous or gonococcic infection, by growths in the calices, and by certain drugs. It is said to occasionally arise from large doses of oil of sandalwood or urotropin. Such drugs could not furnish pus cells and, at most, could only irritate and thus predispose by lowering resistance. Rare cases are due to hematogenous infection by tubercle bacilli. The colon bacillus is the organism most often responsible.

**Symptoms.**—A patient who has, or who has had, retention of urine develops high fever, often preceded by a chill, and headache, stupor, and dry tongue are noted. Unlike acute Bright's disease, there is neither edema nor dry skin, convulsions do not occur, the urine is plentiful and contains pus, but rarely blood. The **prognosis** is very bad.

The **treatment** is to remove the obstruction if possible. If the urine be acid, give liquor potassii citratis; if alklaine, give benzoic acid. Gallic acid, eucalyptol, and small doses of copaiba or cubebs are recommended. Venice turpentine, camphor, and opium may be given in pill form. Quinin is used to stimulate the patient. The bladder is to be washed out every day with boric acid solution (3 gr. to 1 oz. of water). Cups, dry or moist, and hot sand-bags or bran-bags are to be applied to the loin. Alcohol may be sparingly administered. Urotropin is a useful drug. Lavage of the pelvis by means of the hydrostatic apparatus shown in Fig. 874 may be practised and is often most useful. For lavage, use a return flow catheter; first wash the pelvis gently with normal salt solution or sterile distilled water. Silver nitrate in solution, of a strength of $\frac{1}{10000}$, may be used.

S. B. Dudgeon and A. Ross, of London ("Annals of Surgery," March, 1910), recommend in colon infections the use of autogenous vaccine, and report a series of cures. A sterile urine was exceptional in chronic cases. Small doses of 100,000,000 to 200,000,000, administered every five days, were best. Relapses are apt to occur.

**Perinephritis** is an inflammation of the perinephric fatty tissue produced by cold, febrile disease, slight traumatism, or the spread of inflammation from another part.

The **symptoms** of this condition are rigidity of the spine, the inclination being toward the affected side, flexion of the thigh, pain in the loin and iliac region, and often pain in the knee. The symptoms resemble those of hip-joint disease in the second stage. Suppuration may or may not take place. In some suppurative cases the condition strongly suggests woody phlegmon (see page 136).

The **treatment** is wet cups to the loin, heat to the loin, rest, purgation by salines, morphin for pain, and, after the acute stage, potassium iodid internally. In a lingering case an incision should be made. It will frequently be followed by cure. Decapsulation has advocates. If suppuration occurs it is necessary to incise and drain.

**Perinephric or Perirenal Abscesses.**—An abscess in the perinephric fat is known as a perinephric or perirenal abscess. *Primary abscess* may follow a chill, may develop during or after an acute febrile disturbance, or may be caused by pus flowing from some other part, as the spine. Slight traumatisms, by producing hemorrhage, make the perinephric region a point of least resistance and lead to abscess. The causative injury may be produced by digging, stamping, coughing, falling, carrying a burden, lifting a weight, or riding on a horse or on a jolting wagon. *Consecutive abscess* is secondary to kidney inflammation, suppuration, calculus, tuberculosis, or cyst. In the consecutive form the symptoms may be masked by the malady to which perinephric abscess is secondary. As a rule, in a case of perinephric abscess there are found the constitutional symptoms of suppuration. There is high leukocytosis. The local symptoms are a deep aching and paroxysmal pain in the loin, intensified by lumbar pressure. There may be pain in the iliac region and pain in the knee. Edema of the corresponding foot and lameness are not unusual. The thigh is often drawn up. The spine is rigid and inclined toward the diseased side. Edema of the skin is usual, but fluctuation is not. An exploratory incision will settle a doubtful diagnosis.

The **treatment** is to lay open the abscess, wash it out, and drain.

**Stricture of the Ureter.**—This is usually at or near the termination of the ureter. It is due to gonococcic inflammation, pyogenic inflammation, or tuberculosis. The symptoms, as Howard Kelly says, are at first those of a vesical or renal inflammation. The diagnosis is made by the ureteral catheter. We may be unable to introduce it; we may introduce it with difficulty and find that the pelvis of the kidney is distended and that the urine obtained is slightly acid or even alkaline, much lower in urea than the urine from the other kidney, and perhaps contains pus. Stricture of the ureter causes hydronephrosis or pyonephrosis. Great care must be exercised in the examination of the ureter for stricture, and we know of cases diagnosticated as stricture that were simply spastic contractions or deviations in the course of the tube resembling stricture. Some urologists have regarded the condition as quite common, but we do not believe it is so; in several of the cases so diagnosticated a pyelographic study of the ureter has revealed tortuosities due to a descended kidney. These cases will often have greatly enlarged pelves and many of them suffer from mild attacks of kidney colic. Before arriving at a diagnosis the Blasucci catheter should be used and a pyelographic study be made.

**Treatment.**—Dilatation with bougies, resection of the diseased portion and anastomosis, resection of the diseased portion and implantation of the sound end into the bladder, or division of the stricture and suture. In tuberculosis the diseased kidney and ureter may be removed.

**Hydronephrosis** is a condition of the kidney resulting from an impediment to the outflow of urine by obstruction in the ureter, the bladder, or the urethra, the calices of the kidney becoming overdistended with urine and the glandular tissue being absorbed by pressure. It has been asserted by Albarran that secretion of urine ceases in a kidney whose ureter is completely blocked, distention being due purely to congestion. Hydronephrosis may be congenital, due usually to twisting of the ureter or to valve-formation obstructing the ureter at its point of junction with the pelvis of the kidney, the valve being produced because the ureter passes into the kidney pelvis at an unnatural angle. Occasionally imperforate urinary meatus produces hydronephrosis of both kidneys.

The **causes** of the acquired form are the pressure of pelvic growths or pregnancy, inflammation or tumor of the bladder, stone in the bladder, kidney, or ureter, twisting or kinking of the ureter of a movable kidney, enlargement of the prostate gland, and stricture of the urethra. Acquired hydronephrosis may involve both kidneys, all of one kidney, or only a part of a single gland.

**Symptoms.**—Hydronephrosis is most frequent in females. When a lumbar tumor is absent there may be no symptoms, or there may be pain in the back and abdomen, frequent micturition, a persistent or intermittent diminution in urine, or even occasional anuria. A mass may be found in the loin, which growth is dull on percussion and may come and go, a large urinary flow occasionally occurring as it disappears. Hydronephrosis may last a long while if only one kidney be involved, but death is not far distant if both glands suffer. Death occurs from uremia, from pressure on adjacent organs, or from rupture into the peritoneal cavity. The diagnosis is aided by the use of the cystoscope, by catheterizing the ureters, and by pyelographic study.

**Treatment** by aspiration may possibly cure, but the operation will have to be done repeatedly. Tapping on the left side is performed just below the last intercostal space; on the right side the tap is made midway between the last rib and the crest of the ilium. Some few cases have been cured by catheterizing the ureter (Pawlik). The proper operation in most cases is nephrotomy, stitching the edges of the cut kidney to the surface. After the kidney has been opened, explore the ureter by means of a uterine sound or an elastic bougie. A healthy ureter will permit the passage of an instrument of the size of from No. 9 to No. 12 of the French scale (Fenger). If the opening of the ureter into the pelvis cannot be found, open the pelvis or open the ureter. A valve should be slit longitudinally and sutured transversely (Fenger). If a permanent suppurating fistula ensues or if the organ is found extensively damaged, nephrectomy is to be performed, provided the other kidney is in reasonably good condition.

**Pyonephrosis** or **surgical kidney** is a condition in which the pelvis and the calices of the kidney are distended with pus or with pus and urine. The whole kidney may be destroyed. This condition has the same causes as hydronephrosis, for it is, in reality, usually an infected hydronephrosis. In some cases the inaugural malady is pyelitis, which causes blocking of a ureter. Watson, of Boston, has reported 2 cases associated with obliteration of the ureter by a mass of fibrous tissue (stricture of the ureter).

The **symptoms** are those due to the obstructing cause plus pyelitis. Pus may appear in the urine in incomplete obstruction, or it may intermittently come and go. Bacilluria and especially colon bacillus infection of the urine has a strong tendency, unless speedily controlled, to cause pyonephrosis. Constitutional symptoms of suppuration are soon manifest. A mass like the tumor of hydronephrosis may appear in the loin. If only one kidney is involved, and if the disease is due to blocking of a ureter,

recovery is to be expected. The diagnosis is rendered more certain by the use of the cystoscope and by catheterizing the ureters.

The **treatment** in the early stages comprises removal, if possible, of the cause of obstruction, and the employment of measures directed to the cure of the pyelitis. If obstruction is not complete, palliative measures may be employed for the tumor. If fever continues; if there is great visceral derangement; if pain is severe and constant, and if the mass continually enlarges, perform nephrotomy, stitching the organ to the surface if possible, or removing it if it is hopelessly disorganized and the other kidney is in a good or a fairly good condition.

**Chronic Tuberculosis of the Kidney.**—This condition may begin in one kidney, no other area of infection existing in the body. In such cases the bacteria were deposited from the blood. Even when the bacteria are deposited from the blood there is, in most cases, an initial focus of tuberculosis somewhere else in the body. Primary renal tuberculosis remains for a long time a local disease, but, unfortunately, the other kidney, sooner or later, is very apt to become involved, the process in the first kidney affecting the bladder and secondarily the other kidney. The important point is that tuberculosis of the kidney arising in this manner is at first a unilateral disease.

Tuberculosis of the kidney is seldom a primary disease and usually arises secondarily to tuberculosis of the prostate, bladder, or epididymis. In such a condition the kidney disease is always bilateral. Renal tuberculosis is particularly common in the third and fourth decades of life, and is more frequent in males than in females.

**Symptoms.**—Renal tuberculosis of arterial origin may exhibit no symptoms until the disease is far advanced. Renal tuberculosis secondary to disease of the bladder or prostate always presents symptoms.[1] A very common symptom of renal tuberculosis is the sudden onset of polyuria and frequent micturition. The patient is annoyed day and night, and in some cases micturition is distinctly painful. Paroxysms of renal pain are not unusual. The urine is acid and may contain pus or blood. Tubercle bacilli may be found in the urine or in the sediment of centrifuged urine, but they may be absent. Repeated examination should be made before it can be stated certainly that bacilli are absent. The presence of bacilli proves the diagnosis, but their absence does not negative it (Willy Meyer). If bacilli are not found, inject some of the urinary sediment into a guinea-pig, and note if tuberculosis arises in the animal. Czerny has shown that in cases of tuberculous kidney in which bacilli are not found in the urine the administration of tuberculin will cause great numbers to appear. This agent will also cause a marked febrile reaction if tuberculosis exists. The urine may or may not be albuminous.

In many cases the kidney is obviously enlarged, and the renal area is frequently tender and occasionally painful The patient loses flesh, and there is nocturnal fever followed by sweating. The use of the cystoscope furnishes important information. It shows from which ureter turbid urine is coming. Catheterization of the ureters should be practised by some one who is expert. Always examine carefully to determine if one or both kidneys are involved, if the bladder is diseased, and if the prostate gland or seminal vesicles are tuberculous.

**Treatment.**—Lumbar nephrectomy is not justifiable in the very beginning of a case, because such a patient may be cured by a combination of medical and hygienic treatment, and the weakening effect of the operation of nephrectomy may cause the other kidney to develop tuberculosis rapidly. Tell such a patient to lead an outdoor life. Brown recommends camp-life in the Adirondacks during the summer, and sends such patients south during

[1] F. Tilden Brown, "New York Med. Jour.," April 10, 1897.

the winter. If a patient cannot go to another climate, urge upon him the necessity of practically living out-of-doors (see page 230). Insist upon the taking of plenty of nutritious food. Full antituberculous treatment is indicated (see page 230).

If the kidney is markedly enlarged; if there is profuse hematuria; if the fever is high and persistent; if only one kidney is involved, and if the bladder and prostate are free from disease, perform nephrectomy. In cases with involvement of the other kidney or of the genito-urinary tract lower down, nephrectomy is not justifiable, although nephrotomy for drainage may, for a time, greatly benefit the patient.

**Operations On the Kidney and Ureter.**—**Operation for Chronic Nephritis.**—In 1897 Mr. Reginald Harrison advocated puncture of the kidney to relieve tension in cases of albuminuria, and in 1901 advocated incision of the true capsule of the kidney and puncture of the gland to accomplish the same purpose ("Brit. Med. Jour.," Oct. 19, 1901). Alexander Hugh Ferguson in March, 1899, reported 2 cases of interstitial nephritis cured symptomatically by decapsulation and multiple punctures ("Jour. Am. Med. Assoc.," March 11, 1899). Dr. Geo. M. Edebohls observed, between 1892 and 1897, that in certain cases of movable kidney with albuminuria the albumin and casts disappeared after nephropexy. Rose, Wolff, and Ferguson have observed the same fact. Harrison believes that renipuncture removes the symptoms by abating tension, but Edebohls concludes that nephropexy relieves the condition and possibly cures it by establishing vascular adhesions which carry on additional supply of blood. He proposed to operate for Bright's disease in 1899 ("Med. News," April 22, 1899). Edebohls deliberately operated for chronic nephritis and claimed 8 complete recoveries from chronic Bright's disease ("Med. Record," Dec. 21, 1901). There can be no doubt whatever that operation is sometimes followed by polyuria, disappearance of edema and other symptoms, and apparent cure. But in some cases the disappearance of symptoms has been too rapid to permit of the assumption that new vessels have caused it. In such cases it seems much more probable that relief of tension is the real curative factor. The capsule of the kidney is only slightly elastic, and tension may be brought about by an increase in the blood-supply, by edema, and by cell proliferation. Increased tension causes pain and perhaps hematuria, and tension is relieved by Harrison's plan of incising the capsule. Simple incision is easier, safer, and probably just as useful as stripping the capsule off of the kidney. Edebohls advocates decapsulation and says that the polyuria begins about the tenth day after operation; that improvement begins in one month and is gradual; that the cure is due to vascular adhesions; that the adhesions contain more arteries than veins; that the free blood-supply absorbs exudate and products of inflammation, frees the tubes and glomeruli from pressure and constriction, causes the reëstablishment of a normal circulation and the regeneration of epithelium (Ibid.).

The exact status of the operation is not as yet determined. It does, however, seem to be proved that operation is in some cases followed by apparent cure or great amelioration of the condition. Whether permanent cure is ever thus obtained is doubtful, and the part played by rest in bed and drugs in effecting an improvement must not be lost sight of. I have seen no case of genuine cure. Albumin has always continued present in the urine. It is certain that the operation is unjustifiable unless medical treatment has been tried and failed, unless the symptoms are growing worse, and unless they indicate danger. James Tyson ("Med. Record," July 8, 1911) sets forth dangerous symptoms as follows: Persistent dropsy, uremia (violent headache, convulsions, or coma), excessive amount of albumin causing anemia and weakness, anuria. The best results are obtained in chronic parenchy-

matous nephritis associated with marked anasarca. Pain and bloody urine are often much improved by incising the capsule. Postoperative suppression and the anuria of acute infectious diseases may be favorably influenced by the operation. In perinephritis it may prove curative. An important fact which Rovsing maintains and Edebohls proves is that chronic nephritis may be for some time a unilateral disease. (Read the views of Schmidt, in "Med. Record," Sept. 13, 1902; of Rovsing, of Copenhagen, in "Mittheilungen aus den Grenzegebieten der Medicin und Chirurgie," vol. x, 1902, and editorial in "Jour. Am. Med. Assoc.," Jan. 11, 1902; James Tyson, in "Trans. of Amer. Physicians," 1911, and "Med. Record," July 8, 1911.) Personally, I would not operate in the presence of grave and advanced cardiovascular disease. Tyson believes that albuminuric retinitis, very irregular heart, and valvular disease forbid operation, and that in patients over fifty the operation is of slight value.

The operation as practised by Edebohls may be done on both kidneys either at one sitting or in two séances. In some cases only one kidney is subjected to operation. Edebohls takes a very radical view and would operate on any case free from incurable complications, if an anesthetic can be given and if the life-expectancy without operation is not less than one month ("Med. Record," Dec. 21, 1901). Ether is given or nitrous oxid and oxygen. Lay the patient prone with an air-cushion under the belly and expose the kidney by a vertical incision at the edge of the erector spinæ mass, which cut does not open the sheath of the muscle. Remove the fatty capsule from the true capsule, continuing the dissection around each pole until the pelvis of the kidney is reached. The kidney is extruded from the wound, the true capsule is incised along the convex border and around each pole, is separated from the kidney, and is cut away close to its junction with the kidney pelvis. The kidney is then returned to its bed of fat and the wound is closed (Ibid.). Edebohls does not drain unless there is considerable edema. He reported 18 operations without a death. In 9 of the cases the operation was done more than one year ago, and 8 of them are said to be cured. Personally, I do not believe that the operation can really cure Bright's disease. It cannot restore altered connective tissue and epithelial cells. The new blood-supply must be through scar tissue, and we have yet to learn that such a blood-supply can be efficient. The operation should be restricted to acute nephritis, to acute exacerbations in chronic cases, to conditions with severe renal pain, hematuria, anasarca, very high albumin percentage, or persistent and notable diminution in the amount of urine voided (Ertzbischoff, in "Archiv. générales de Chirurgie," April, 1908).

**Nephrotomy** means incision of a kidney, but the term is sometimes, though wrongly, applied to the exploratory exposure of the kidney without incision of the organ. When the kidney wound is left open, as it almost invariably is, the operation should be called *nephrostomy*. The operation is employed to evacuate infectious material, relieve tension, permit of the removal of a calculus or exploration of the ureter, and for diagnosis of renal disease. The patient lies upon the sound side, a sand-pillow or a cylindrical air-bag being placed under the flank. The *incision* is made ½ inch below the last rib and close to the outer border of the erector spinæ mass, and runs obliquely downward and forward toward the iliac crest for 3 inches, the incision being enlarged later if required. Divide the skin, the superficial fascia, the fat, the external oblique, the posterior border of the internal oblique, and the outer edge of the latissimus dorsi. This incision exposes the lumbar fascia. Push aside the last dorsal nerve and incise the lumbar fascia, when the perirenal fat will bulge into the wound. Two distinct layers of fat exist. Tear through this fat with dissecting forceps to expose the kidney, which can now be opened while it is forced into the wound

by the hand of an assistant making abdominal pressure. In some cases the kidney can be brought out of the wound for exploration and operation. In others it cannot. When it cannot be drawn out it is brought into the wound and supported by means of a pad of gauze under each pole.

Kocher's incision for nephrotomy is begun in the angle between the sacrolumbalis muscle and the twelfth rib, and is carried downward, forward, and outward to the axillary line (see Fig. 248). This incision divides the skin, subcutaneous tissues, lumbar fascia, the latissimus dorsi, and the serratus posticus inferior muscles. If possible, the kidney is brought out of the wound.

Edebohls's method enables the surgeon to explore the kidney thoroughly because this organ is brought outside of the body. It is not suited to cases in which the organ is much enlarged or when it has a short pedicle. Its best field is in operations for movable kidney. The patient lies prone, with a large cylindrical inflated rubber pad beneath his abdomen. A vertical incision is made close to the border of the erector spinæ muscle, from just below the last rib to just above the iliac crest. The sheath of the muscle is not opened. The fibers of the latissimus dorsi are separated by blunt dissection. The iliohypogastric nerve is found and retracted. The transversalis fascia is incised and the fatty capsule reached. The two layers of the fatty capsule are torn through and the kidney exposed. The fatty capsule is *well separated* from the kidney front and back. The patient is pulled by the legs toward the foot of the table, the pad remaining stationary. This change of position brings the pad beneath the chest, abdominal respiration takes place, the kidney is forced into the wound, and can be easily withdrawn and thoroughly examined. In many cases the lumbar incision does not expose the kidney pedicle completely enough to make the surgeon feel that he can readily clamp it should nephrectomy become necessary, and in any operation upon the kidney nephrectomy may at any minute become necessary. Cutting the twelfth rib adds to the exposure, but in cutting it the pleura may be opened. Wm. J. Mayo ("Annals of Surgery," Jan., 1912) in 203 lumbar operations divided the twelfth rib 51 times, and in 13 of the operations accidentally opened the pleural cavity. In not a case did the lung collapse (probably because the patients lay upon the abdomen with the hips somewhat elevated, the chest being fixed by the position). In each case the torn pleura was sutured, the stitches including tissue of the diaphragm.

Mayo discovered that after division of the quadratus lumborum muscle and the lateral arcuate ligament (which binds the twelfth rib to the transverse process of the first lumbar vertebra) the rib can be lifted out of the way and does not need to be divided. Mayo now operates as follows: Beginning over the eleventh rib $2\frac{1}{2}$ inches external to the spines of the dorsal vertebræ make a longitudinal incision 3 inches in length. From this point carry the incision downward and forward along the anterior margin of the quadratus lumborum to 1 inch above the crest of the ilium; it is then carried forward parallel to the crest. The twelfth rib is cleared nearly to its articulation; the pleura is pushed up out of the way. The erector spinæ mass is retracted toward the spine. The costal margin is raised by retraction. A wide area for operation is exposed, the kidney can be brought out of the wound, and the pedicle can be seen and reached.

A common method of opening the kidney is to make a longitudinal incision through its convexity sufficiently large to admit the finger into the renal pelvis. This incision may be enlarged if necessary until the kidney is split in half. When the operation is completed close the kidney wound completely by means of a round needle and catgut, unless drainage is necessary or packing is required. Another popular incision is placed longitudinally a little posterior to the convex border. This goes through the bloodless zone of Hyrtl, and causes less hemorrhage than the incision along the convexity. Marwedel's incision is a

transverse cut at the middle of the convex border of the kidney into the renal pelvis. It is doubtful if this incision is accompanied by less bleeding than longitudinal incision of the convexity. After the completion of our work on the kidney the lumbar wound is closed completely or is partially closed to permit of drainage.

**Operation for Stone in the Kidney By Pyelotomy or Nephrotomy (Pyelolithotomy, Nephrolithotomy).**—It used to be held that incision of the pelvis of the kidney is far more apt to be followed by fistula than incision of the substance of the kidney. The Mayos, Bevan, and others have proved that this conviction is untrue. In many cases of stone pyelotomy is preferred to nephrotomy. It is used for single stones of moderate size in the pelvis and unaccompanied by suppuration. When we have a large branched stone, coral-shaped stones, multiple stones, and stones accompanied by distinct and gross evidences of infection, then nephrotomy should be preferred to pyelotomy (Bevan and Smith, in paper read before the American Surg. Assoc. in 1908). In both operations the patient is placed on the sound side with a sand-pillow or cylindrical air-bag under the flank. In both cases the incision recommended for nephrotomy is used. In both cases the kidney is lifted into the wound or brought out of the wound so that it may be satisfactorily palpated. In both cases bleeding is controlled by having an assistant, if possible, grasp the pedicle with his fingers or with a pair of forceps, each blade of which is covered with a bit of rubber tube, while the surgeon opens the pelvis or the kidney tissue, removes the stone, and explores with the finger.

*Pyelolithotomy.*—Remove the fat from the posterior surface of the pelvis of the kidney. We may then be sure that no aberrant vessel is in our way. The normal vessels are all in front of the pelvis. The posterior wall of the pelvis is now opened, the stone is extracted, the pelvis explored, the cut in the pelvis is sutured with fine catgut sutures, the kidney is restored to place, a cigarette drain is introduced, and the wound in the loin is closed.

*Nephrolithotomy.*—The methods of incision, exposure, and opening the kidney are described on page 1292, under Nephrotomy. When the kidney has been opened, loosen the calculus with the nail, and remove it with the finger, with a scoop, or with forceps. After removing the stone, suture the incision with catgut, and release the pressure on the pedicle. Hemorrhage will usually be controlled, but sometimes violent bleeding occurs. If in spite of this plan bleeding occurs, take out the stitches and apply pressure and hot water, compress the bleeding points by suture-ligatures, and close again by Stellwagen's stitches (see Figs. 853, 854). In some cases plug with iodoform gauze, suturing the gauze in place within the kidney by fine catgut and leaving it until it loosens. When hemorrhage ceases, put a large drainage-tube down to the kidney. Close the wound in the muscles and integument and dress antiseptically. The dressings must be changed frequently and the tube should be shortened daily. In some cases nephrectomy is necessary (see page 1285). Formerly in these cases I always drained for a time and removed the kidney secondarily, believing that the patient would gain strength in the interval and stand the severe operation of nephrectomy better. I am satisfied that in most cases this view is wrong, because removal of a kidney bound down by adhesions is one of the most perilous and difficult operations of surgery.

**Nephrectomy** is the removal of a kidney. There are two methods of nephrectomy—the *lumbar* and the *abdominal*. The first nephrectomy (according to Watson) was performed in 1861 by an American, Walcott. The operation was transperitoneal and was for the removal of a cancerous kidney. Simon, in 1869, performed the first lumbar nephrectomy and the first successful nephrectomy. Before performing nephrectomy ascertain the competence of the kidneys. If at least 1 per cent. of urea is not being excreted, it is very un-

safe to operate. Be sure the patient possesses two kidneys. Examination of the bladder by the cystoscope will show the ureteral orifices, a strong indication that both kidneys are present. Nevertheless, when we reflect that a horseshoe kidney has two ureters, the proof is not absolute. Catheterization of the ureters is advisable if it can be performed, but it will probably require a specialist to perform it. Proof absolute of the presence of two kidneys consists in feeling both of them. If in doubt as to the question, and if uncertain as to the ability of the organ which is to be left, feel each kidney during the operation and before removing either, or perform a preliminary exploratory laparotomy.

**Lumbar Nephrectomy.**—The patient is placed on the sound side and a pillow is placed under the loin. Several incisions have been proposed. In many cases the oblique incision is first made to permit of exploration. This incision is begun ½ inch below the last rib and by the edge of the erector spinæ muscle, and is carried downward and forward toward the iliac crest. In some cases a kidney can be removed through this cut. In other cases the cut must be enlarged. It can be enlarged by extending the cut downward. Morris enlarges it by adding to it a vertical incision, which begins 1 inch below the origin of the oblique cut. König's incision for nephrectomy consists of a vertical cut by the edge of the erector spinæ, carried almost to the iliac crest, from which point it is curved forward toward the umbilicus, and is carried to or even through the rectus muscle. After thorough exposure lift the kidney and separate it from the peritoneum, if possible, with the finger; clamp the pedicle; pass an armed aneurysm needle between the vessels of the pedicle; ligate in two places; cut between the threads, and arrest hemorrhage permanently by ligation of each vessel. If the ureter be healthy, ligate it with silk and drop it back; if it be foul and purulent, scrape it with a sharp spoon, wash it with corrosive sublimate, and touch it with pure carbolic acid, and then either ligate it with catgut and drop it back or sew it into the wound. If hemorrhage persists from the wound, plug with gauze. Insert a drainage-tube and close the wound. If the peritoneum be accidentally opened, close it with Lembert sutures. Kocher's method is excellent, and enables the surgeon to feel the opposite kidney before removing the one which is known to be diseased. The incision is begun as described on page 1293, and is carried forward so as to expose the reflection of the peritoneum on to the colon in the posterior axillary line (see Fig. 248).[1] At this point the peritoneum is opened, and the surgeon's hand is inserted into the abdominal cavity and feels the other kidney. If another kidney exists and it is found to be healthy, the diseased organ may be removed. Brewer's personal statistics show 53 cases of nephrectomy with 2 deaths, a mortality of 3.8 per cent. ("Med. Record," March 20, 1909).

**Abdominal nephrectomy** is more dangerous than the lumbar operation. The position is supine. The incision is that of Langenbeck—4 inches in length in the linea semilunaris, its center corresponding to the umbilicus. Open the abdomen, introduce a hand, feel the kidneys, and if both show serious disease, do not perform nephrectomy. If we decide to remove one kidney, keep the small intestine away by pads, push the colon toward the umbilicus, incise the outer layer of the mesocolon, and bare the kidney. Strip off the peritoneum from the kidney and its vessels, and ligate the vessels by passing strong silk through the center of the pedicle with an aneurysm needle. Ligate the ureter if healthy, and divide it. If the ureter is septic, fasten it to an opening made in the loin by cutting on to forceps pushed to the outer edge of the quadratus lumborum. Stop bleeding, irrigate the belly cavity, and dress as usual, employing drainage only when septic matter has passed into the peritoneal cavity or when oozing of blood is persistent.

[1] Kocher's "Text-Book of Operative Surgery."

**Nephrectomy in Children.**—The operation is proper in certain non-malignant troubles. Jepson did a successful nephrectomy for a congenital cystic kidney on a patient four months and fourteen days of age. Rovsing did it successfully for congenital hydronephrosis, the patient being nine months old. Roswell Park did a successful nephrectomy for congenital cystic kidney on a child twenty-three months of age. The value of nephrectomy for sarcoma is certainly doubtful. The operation never really cures, and if an operative recovery is obtained the disease appears after a time in the other kidney. Jessup performed nephrectomy in 11 children and every case died within two and one-half years of the operation. The operation often prolongs life and relieves discomfort, but does not cure.

**Partial Nephrectomy.**—This operation may be performed in some cases for wounds, cysts, and innocent tumors. After removing the damaged or diseased part, bleeding points are ligated with catgut. The wound surfaces are approximated as well as possible by catgut sutures. Drainage is intro-

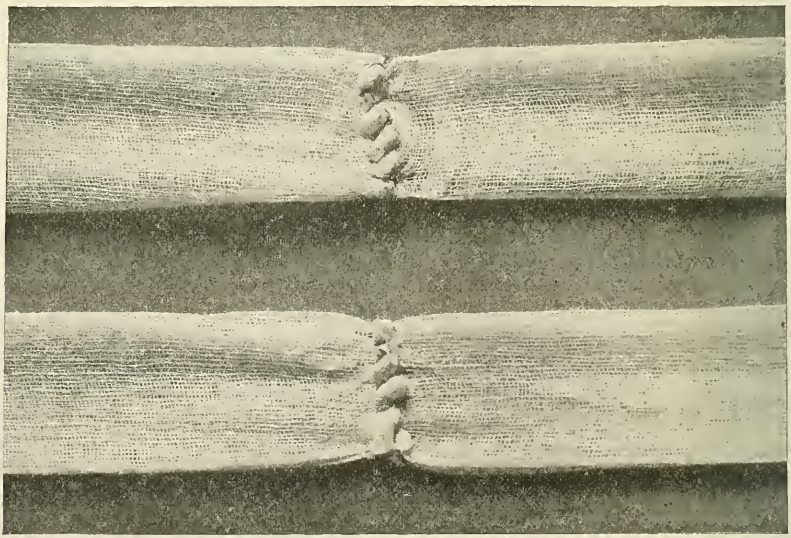


Fig. 856.—Gauze slings, each composed of two pieces sutured together with fine plain catgut.

duced. The value of partial nephrectomy in some cases seems certain, and we should apply it when possible instead of the complete operation,[1] except in cases of malignant disease.

**Renipuncture.**—This is an operation devised by Reginald Harrison for the relief of albuminuria due to elevated tension. The kidney is exposed in the loin, the capsule is incised, and punctures are made in the kidney. Simple incision of the capsule will usually relieve nephralgia. (See Operation for Chronic Nephritis, page 1291).

**Nephropexy** is fixation of a movable kidney. The term "nephrorrhaphy," so long used for the operation, really means suturing a wound in the kidney.

**The Author's Modification of the Elder Senn's Operation.**—Many surgeons feel that it is not desirable to pass sutures through the kidney substance, and I have entirely abandoned the use of them in operations for movable kidney. Urinary fistula has followed suturing. Again, the value of such sutures

[1] See Oscar Bloch, in "Brit. Med. Jour.," Oct. 17, 1896; also reports of Czerny, Bardenheuer, Tuffier, and Kümmell.

is very doubtful. The kidney is a very soft organ, and if it is suspended by sutures, they are certain to cut out. In most suture operations the kidney when restored to place is not placed sufficiently high and has its ureter and vessels looking forward; in other words, there is a one-fourth twist in the ureter. In operations like Goelet's and Kelly's, which raise the kidney much nearer its proper level and which do not twist the ureter and renal vessels, the upper pole is not anchored and tends to tilt forward (see page 1279). The operation herein described fixes the kidney without using sutures.

The patient lies upon his abdomen, Edebohls's bag being placed directly beneath the lower abdomen. A vertical or slightly oblique lumbar incision is made, the perirenal fat is exposed, and its two layers are torn through until the kidney is reached. The fatty capsule is thoroughly stripped from the entire organ. The kidney is brought out of the wound. This is accomplished by pulling the patient toward the foot of the bed, so that the pad gets under the ribs, when traction on the fibrofatty capsule will cause the kidney to emerge from the wound. The posterior fatty capsule is cut away, and also the anterior fatty capsule up to the hilum. The true capsule of

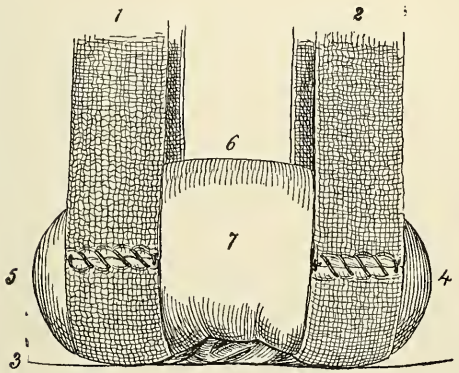



Fig. 857.—Right kidney projecting from wound. Observer standing on right side of patient: 1 and 2, Slings in place, with sutures external; 3, skin of the back; 4, upper renal pole; 5, lower renal pole; 6, convex border of kidney; 7, external surface of kindey. (Slings should be broader than those shown in illustration.)

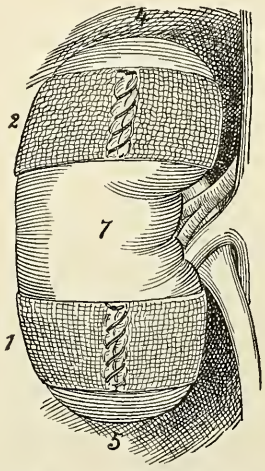



Fig. 858.—Right kidney restored to place, seen from in front: 1 and 2, Slings in place, sutures anterior; 4, upper renal pole; 5, lower renal pole; 7, anterior surface of kidney. (Slings should be broader than those shown in illustration.)

the kidney is scarified. I always have packing prepared by suturing together with the finest plain catgut the ends of two pieces of iodoform gauze. Two such strands are prepared (Fig. 856). One piece of iodoform gauze is placed under the upper end of the kidney, and another piece under the lower end, the sling in each instance being directly under the kidney with the suture line external and not in front, as the kidney protrudes from the wound in the back (Fig. 857). When the kidney is replaced the suture line will lie in front (Fig. 858). The kidney is replaced and will then lie in a sling composed of two pieces of gauze, the ends of which protrude from the wound. Another piece is placed below the lower renal pole to fill up the space which always exists there and to stimulate granulation. This space below the kidney is a frequent cause of subsequent loosening in most suture operations, because the kidney hangs in it unsupported, as a bucket hangs in a well. Harris recognizes this, and in his operation closes the space by sutures. Gauze is packed in over and about the kidney, and over this the two long slings

are tied. Several sutures of silkworm-gut are inserted to close the superficial parts and the lumbar aponeurosis; some are tied and some are left untied. A large gauze pad is placed upon the abdomen over the anterior surface of the kidney, and the lumbar wound is dressed with gauze. The dressing and gauze pad are held in place by a binder. In about eight or ten days the gauze should be soaked with dripping salt solution during half an hour and the packing removed. At this time the catgut is destroyed and the gauze can be easily pulled out. The tied sutures are cut and removed, the sutures left unfastened are tied, and a small piece of gauze is inserted as a drain between the granulating surfaces. If a continuous piece of gauze was used, ether must be given before removal is attempted. Further, in the old operation, a large wound was left to granulate and weeks were often required to obtain healing. In this operation the wound is usually entirely healed in from eighteen to twenty-one days. After the performance of nephropexy the patient remains

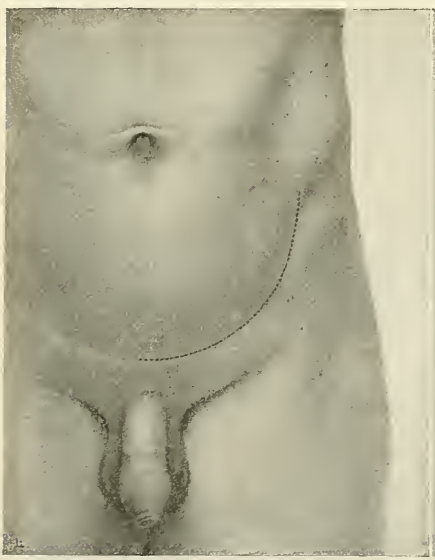


Fig. 859.—Gibson's incision for operations on the lower ureter. The superficial incision (C. L. Gibson).

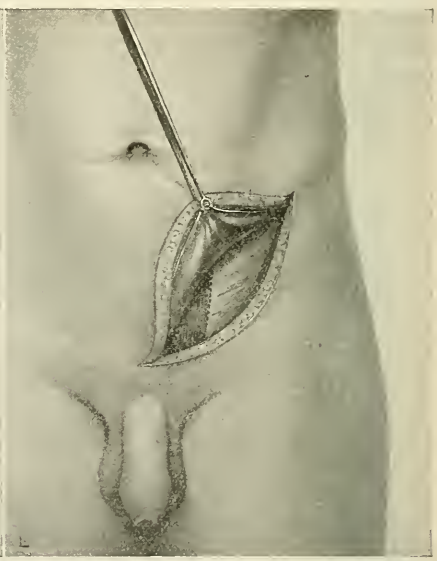


Fig. 860.—Gibson's incision for operations on the lower ureter. The upper flap, consisting of skin, external and internal oblique muscles, is retracted. The dotted line represents the line of incision in the transversalis fascia (C. L. Gibson).

in bed for three weeks. By this operation the kidney is placed in a proper situation, is surrounded with granulations which are converted into scar-tissue, and the organ becomes encased in a box of fibrous tissue. I believe that a kidney so treated will probably remain fixed.

If a kidney has been decapsulated, gauze slings should not be placed around it, because removal of them might lacerate the kidney. After decapsulation the surgeon rests content when he has filled the space below the kidney with a support of gauze.

**Ureterolithotomy.**—If the stone is impacted in the upper two-thirds or three-fourths of the tube it may be reached by an incision from the twelfth rib downward and forward to just anterior to the anterosuperior spine of the ilium and then parallel to Poupart's ligament until above its middle. The peritoneum is stripped up as in extraperitoneal ligation of the iliac vessels. The ureter adheres to the peritoneum. The operation is strictly extraperitoneal.

**Gibson's incision for operations on the lower ureter** ("Amer. Jour. Med. Sciences," Jan., 1910) is the most useful for reaching and amply exposing the lower ureter. Further, it enables us to feel and accurately operate upon the tube, the entire operation being extraperitoneal. Gibson thus describes his operation:

"The skin incision runs from the midline about a finger's breadth above the pubes, horizontally outward nearly parallel to Poupart's ligament at first (Fig. 859), and curves rather sharply upward at its midpoint to end about opposite the anterior superior spine of the ilium. This incision is deepened in the same line through the aponeurosis of the external oblique and the internal oblique muscle—the latter is the only structure which suffers any real damage, and only to a slight degree, for the lower part of the incision runs about parallel to its fibers, only the ascending leg cuts across a small part of these fibers. The incision stops short of the transversalis, which is not dis-

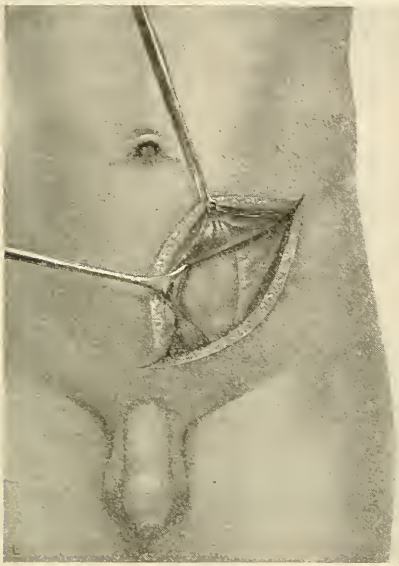


Fig. 861.—Gibson's incision for operations on the lower ureter. The edge of the rectus muscle is strongly retracted inward; between it and the cut edge of the transversalis fascia the peritoneum is exposed (C. L. Gibson).

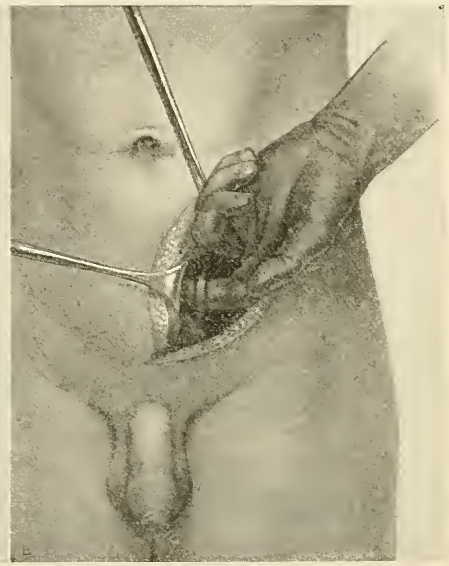


Fig. 862.—Gibson's incision for operations on the lower ureter. The peritoneum has been pushed upward. The ureter is lifted out of the pelvis and brought to the level of the external wound (C. L. Gibson).

turbed at all. With efficient retraction of the upper flap the external border of the rectus muscle is identified (Fig. 860) and the *fascia* of the transversalis is now divided by a vertical incision close to and parallel to the rectus—that is, at *right angles* to the original incision. Two retractors are now inserted, the outer one retracts the cut edge of the transversalis outward, the other (Fig. 861) pulls the rectus muscle well toward the midline. A generous space is thus obtained, situated well toward the midline (the lower part of the ureter is practically in the midline and difficult of access by other extraperitoneal exposures). The floor of this space is occupied by the peritoneum. The patient being in a complete Trendelenburg position, the peritoneum is easily and gently pushed away, and a free access to the pelvis is secured. So ample is the space and view that the whole hand can be introduced under the control of the eye. The ureter is released from its surroundings and easily brought to the level of the wound" (Fig. 862).

When the tube has been exposed it is opened by a longitudinal incision. The stone is removed. The ureter is explored by means of a sound to see if it is free. After removing the stone, close the wound in the ureter with sutures of very fine chromic gut. Deep sutures pass through all the coats and are tied. Over this a layer of superficial sutures are inserted. Close the tissues about the ureter and drain by rubber tissue. Never drain by gauze. To do so will cause a urinary fistula. In a woman a stone near the vesical opening can be reached by a vaginal incision. Stone in the vesical portion of the ureter may perhaps be removed by aid of an operating cystoscope or by forceps after the performance of a suprapubic cystotomy.

My colleague, Prof. John H. Gibbon, advocates a combined intra- and extraperitoneal route for stones anywhere in the lower two-thirds of the ureter. The peritoneal incision permits of exploration and exact localization of the stone, allows the surgeon to push the calculus from an inaccessible into an easily reachable position, and makes the removal vastly easier. The stone is removed by extraperitoneal incision of the ureter, and the peritoneum is closed (Gibbon, in "Annals of Surg., Gynec., and Obstet.," May, 1908).

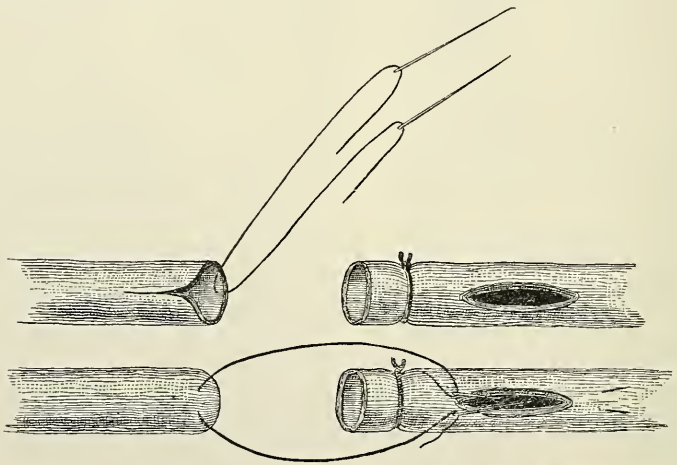


Fig. 863.—Van Hook's method of ureteral anastomosis.

**Uretero-ureterostomy** (*Van Hook's Operation*).—In this operation ligate the lower end of the divided ureter with silk or catgut. About $\frac{1}{4}$ inch below the ligature make an incision in the long axis of the tube. This incision is in length equal to twice the diameter of the tube. Each end of a piece of fine catgut is threaded to a fine needle. This thread is passed through the upper end of the ureter (Fig. 863). The needles are made to enter the lower end of the tube through the door made by the surgeon. They are pushed through the wall of the ureter $\frac{1}{2}$ inch below the door (Fig. 863). Traction upon the strings causes invagination, and the ligature ends are tied. If the operation is intraperitoneal the ureter is wrapped about with peritoneum.

**Intestinal Implantation of the Ureters.**—This operation may be employed in exstrophy of the bladder and in vesical cancer in which it is necessary to remove the bladder. After this operation there is danger of infection of the ureters and consequent ascending ureteritis and pyelonephritis, and the presence of urine in the bowel usually causes inflammation of the rectum and incontinence of urine may take place.

Maydl asserts that a piece of the bas-fond should be removed with the ureter, and implanted with it into the intestine, the flange hanging free in

the lumen of the gut. If this is done, the relations of the ureter to the muscular coat of the bladder are not interfered with, stricture is less likely to occur, ascending infection is antagonized, and suppurative conditions arise at the margin of the flange rather than, as in other methods, directly in the cut ureter. Maydl has collected the records of 14 cases operated upon by this method, with 2 deaths.[1] I performed it twice, with 1 death. In vesical exstrophy Peterson transplants a vesical flap containing both ureteral orifices into the descending colon.

**Cystoscopy** is the employment of the cystoscope for the study of the interior of the bladder, the prostate, the ureteral orifices, and the appearance of the fluid coming from each kidney.

The cystoscope is an instrument of great value in the hands of a skilful and experienced man, but is practically useless when employed by a novice. All of my cystoscopic examinations are made for me by Dr. Stellwagen or some other expert. In using a cystoscope the mucous membrane may be burned with a hot lamp. This causes inflammation, and if an eschar forms, it will be cast off, exposing a granulating surface. Schmidt calls attention to this injury, speaks of the condition as *ulcer cystoscopicum*, says it is in the fundus, has the shape of the instrument, and heals in from fourteen to twenty-one days ("Jour. Amer. Med. Assoc.," July 19, 1902).

Cystoscopic examination of the bladder owes its present position to Messrs. Max Nitze and Joseph Leiter, who were the first to introduce practical instruments: Nitze in 1876 and Leiter in 1879. The great obstacle in former years was the danger of burning the mucous membrane by overheating of the lamp. The invention of the cold lamp by E. C. Preston eliminated that danger, and was a great step forward. Nitze was the first to recognize the futility of examination by reflected light and constructed an instrument with the light on the end which to-day is copied by all of the modern instruments.

Cystoscopes may be divided into several types: (1) The examining instrument; (2) the instrument carrying ureteral catheters or the catheterizing cystoscope; (3) the operating cystoscope. They all have certain mechanical features in common, but differ in their construction and lens arrangement.

The examining instruments are of two fundamental types—the direct and the indirect. In the direct there is a straight telescope with a series of wide angle lens that give the picture of the bladder in front and slightly to the side. In the indirect the lens is so ground and set as to enable the operator to look toward the prostate.

Catheterizing cystoscopes are also of two kinds—the direct catheterizing and the indirect. In the direct catheterizing cystoscope the catheters are passed directly from the carrying tubes into the ureters. In the indirect catheterizing cystoscope the catheter is bent or directed by a lever on the end of the instrument operated by a thumb-screw on the penile end. By raising or lowering the lever the angle of the catheter is altered to facilitate its introduction into the ureteral orifice.

It is obvious that the bladder should be distended in order to permit of cystoscopic examination. For this purpose air and water have been utilized. The modern tendency is in favor of water distention. Some of the operating instruments, notably that of Prof. Lewis, require air.

Water as a dilating medium is superior to air, unless operative work is to be done through the instrument, in which case the inevitable hemorrhage precludes the use of water. The air instruments show but a small field, the distention causes pain and is often followed by mild cystitis.

From the foregoing it is plain that one type of instrument is not suited to all cases, and each has its claim to practical utility. Some cases can be most

[1] Editorial in "Jour. Amer. Med. Assoc.," May 6, 1899.

readily catheterized by the direct, while others are only reached by the indirect, plan. It is my conviction that if the ureteral orifices are normally placed, the direct method is quicker and more readily learned by the beginner. Men who have had training had best use the A. C. M. I. cystoscope (Figs. 864, 865). It is an indirect or oblique cystoscope, but has a corrected image field which enables the operator to work as he sees and not the reverse (as is necessary with the uncorrected image field). These new instruments cause less pain than direct instruments, and give a very wide field. When from any cause the ureteral orifice turns backward (which only happens in a very small percentage of cases) the indirect cystoscope is indispensable, it being practically impossible to reach the ureteral orifice by the direct instrument.

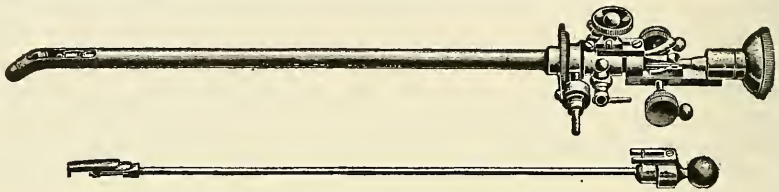


Fig. 864.—The A. C. M. I. double catheterizing cystoscope.

There are at present many different forms of cystoscopes on the market, notable among them are the Nitze, Casper, Tilden Brown, Cabot's modification of the Tilden Brown, Buerger, Albarran, Bransford Lewis, and the so-called Universal. The universal instruments of Bransford Lewis and Tilden Brown possess features of great practical utility.

*Sterilization of Cystoscopes and Ureteral Catheters.*—In order to sterilize the cystoscope before using, place it for five minutes in a solution of mercury oxycyanid (1:1000) or formaldehyd (1:500). The sterilizing power of formaldehyd gas is not to be relied on. Some operators use a 5 per cent. solution of phenol, in which the instrument is immersed for twenty-four hours, after which it is dipped into glycerin, which both neutralizes the phenol and lubricates the

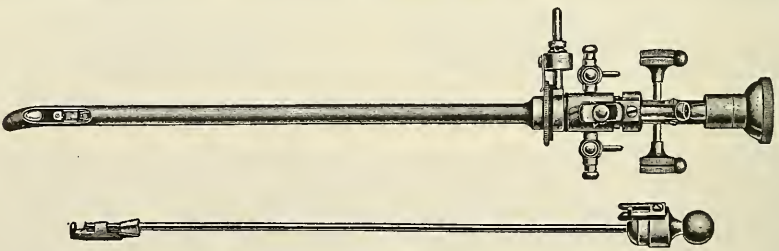


Fig. 865.—The A. C. M. I. operating cystoscope.

tube. The mercury oxycyanid solution is easily used and is thoroughly satisfactory. Should formaldehyd solution or formalin gas be used, always wash the instrument thoroughly with salt solution before using to prevent irritation by the sterilizing medium.

Dr. M. P. Willard, of San Francisco, in "Jour. Amer. Med. Assoc.," October 4, 1913, advocates the following method for sterilizing ureteral catheters: The ureteral catheter is placed in a muslin bag 2 cm. wide by 75 cm. long. The bag is enclosed, with the catheter in it, in paraffin paper, and secured by encircling strips of adhesive plaster. As many may be prepared as is thought necessary. The package is placed for twenty minutes in an autoclave with a pressure of 8 to 10 pounds. When the catheter is to be used the paper is removed and the end of the instrument is exposed by pulling back the linen bag and the

tip of the catheter is engaged in the carrying tube of the cystoscope. As the catheter is fed through the cystoscope the linen bag is pulled back; in this way the fingers do not come in contact with the catheter.

Ureteral catheters can be sterilized by placing them in an open shallow dish containing a saturated solution of ammonium sulphate or sodium chlorid and boiling them for five or ten minutes. The following precautions must be observed: Each catheter should be wrapped in gauze and the bottom of the pan covered with gauze. If the catheters are allowed to touch each other or curl upon themselves the shellac coating will melt and ruin the instruments. They may also be sterilized in the same manner as the cystoscope, care being taken if formalin was used to carefully wash away the germicide, especially from the lumen of the catheter.

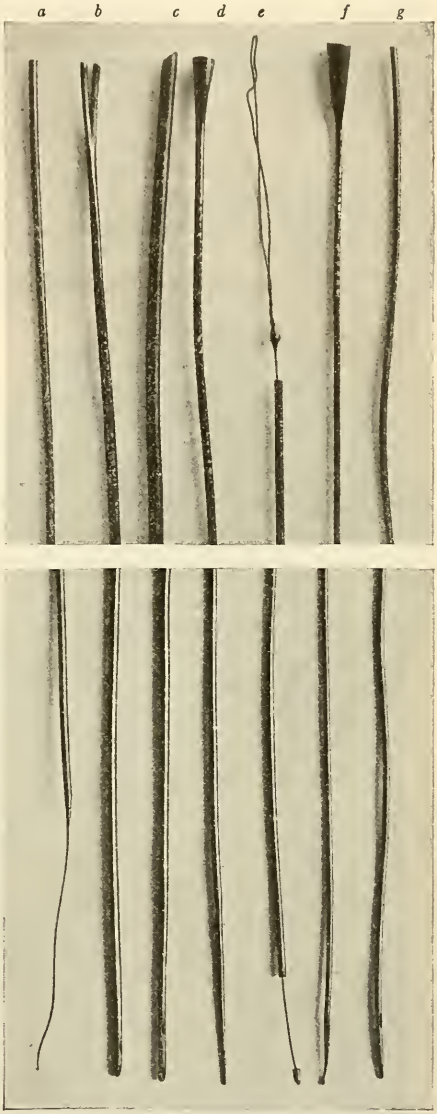



Fig. 866.—Different types of ureteral catheters: *a*, Blasucci; *b*, return flow for irrigation; *c*, Garceau; *d*, another type of Blasucci; *e*, Cunningham; *f*, olivary tipped; *g*, *x*-ray whistle tip.

**Contra-indications to Cystoscopy.**—The bladder must hold at the very least 50 c.c. of fluid. If it holds less cystoscopy is useless. Examination is either impossible or unsatisfactory if the prostate is greatly enlarged. Contracted meatus urinarius and stricture of less caliber than No. 24 of the French scale are correctible contra-indications. Papillomata and other tumors and foreign bodies of the urethra are impediments. In acute Bright's disease instrumentation is dangerous, because it may be followed by suppression of urine. The following are further contra-indications, as suggested by Follen Cabot and Henry G. Spooner, in "Med. Record," July 11, 1903: When it is obvious that operative intervention would be useless; when there is a large tumor; in acute cystitis; in tuberculosis in which the diagnosis is evident without the cystoscope. In women, pelvic adhesions and large exudates may interfere with cystoscopy.

**Technic of Cystoscopy.**—The patient should, if possible, be carefully sounded once a day for several days previous to the cystoscopic examination; this establishes tolerance of the urethra. He should be placed upon a suitable table, such as that devised by Tilden Brown or Bransford Lewis, as shown in Fig. 868.

The urethra is cleaned by irrigation with salt solution. General anesthesia is rarely necessary provided the operator is gentle in his manipulations.

There are, however, some hypersensitive urethras which it is necessary to anesthetize. Cocainization of the urethra will usually be efficient. This is carried out by instillation through the Keyes-Ultzmann syringe or, better, through a soft-rubber catheter into the posterior portion of the canal of

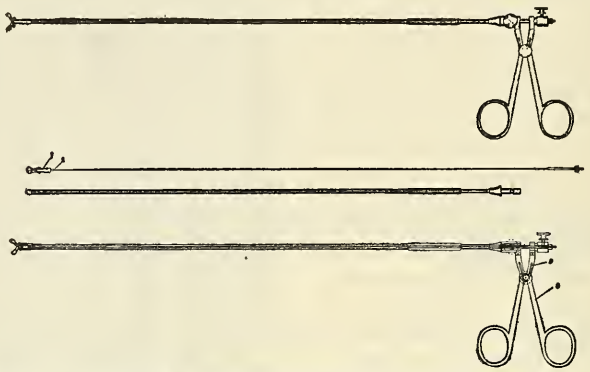


Fig. 867.—Buerger's rongeur forceps with flexible shank for use with Buerger's operating cystoscope.

1 dram of a 4 per cent. solution of cocain. Alypin, placed in the canal at the points of greatest tenderness by means of Dr. Lewis's repositor, answers very well. These tablets are allowed to dissolve in the urethral mucus. The points of greatest tenderness are the fossa navicularis, the bulbo membranous junction, and the prostatic urethra about the verumontanum. In cases complicated by

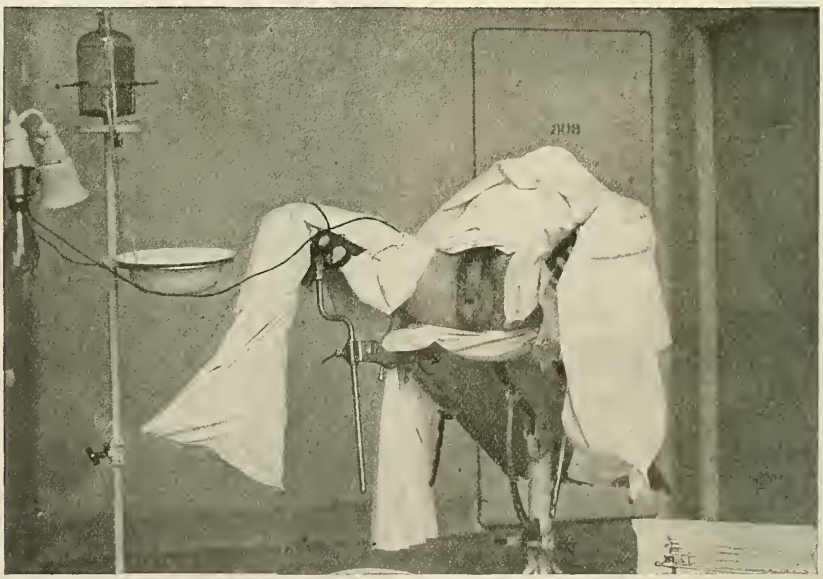


Fig. 868.—Tilden Brown's cystoscopic table with patient in position for examination.

cystitis, preliminary lavage of the bladder should be practised. This may be given through the cystoscope sheath or through a soft-rubber catheter. If the bladder is not diseased, it is merely necessary to draw off the urine. After the urine has been withdrawn, the cystoscope sheath should be firmly but gently held in place. The thumb of the left hand presses downward while the index-

finger presses upward on the under surface of the sheath. This procedure raises the beak of the instrument from the sensitive trigone, where most of the pain and hemorrhage are produced by vesical tenesmus.

In regard to the selection of the distending medium, the operator may use sterile water, normal salt solution, boric acid solution, or mercury oxycyanid solution (1:5000). The bladder should be moderately distended and, if possible, from 8 to 10 ounces of fluid are allowed to remain in the viscus. In contracted or hypersensitive bladders it is well to elevate the hips and so relieve the pressure on the sensitive vesical neck and trigone.

The temperature of the distending fluid is of great importance, particularly if there is difficulty in finding the ureteral outlet. If this is the case, the temperature of the fluid should be about 60° F. This enables the operator to see a so-called swhirl wnen the warm jet of urine mixes with the cold water in the bladder. It is sometimes advisable to give methylene-blue before examining for the same reason. The blue stream from the ureter at once identifies the tube.

After the bladder has been filled the telescope containing the catheters is inserted and the light is turned on. The instrument, then held in the median line, is pushed gently back, well into the bladder, pressure being always made to keep the beak from impinging upon the floor of the viscus. The cystoscope is now slowly withdrawn, a careful watch being maintained through the telescope for the *interureteral bar* (Fig. 869). This structure (there has been much argument as to whether this structure really exists) marks the posterior border of the trigone. The examiner has still another guide to the trigone in the blood-vessels, which in a general way run anteroposteriorly. There is a distinct line of demarcation between the fish-net loop arrangement of the mucous membrane of the sides and base of the bladder and the mucous membrane in the trigone. Fig. 869 gives a general idea of the vascular arrangement. When the interureteral bar or posterior border of the trigone has been located, the instrument is gently and slowly moved laterally, so that the border of the cystoscope is kept in the field until the end of the bar is reached. The instrument is then held stationary for a moment and a sharp lookout is kept for the jet of urine or the swirl. The ureteral orifice is thus indicated. Should the field become blurred by blood on the lens, or should the solution in the bladder become mixed with blood, continuous irrigation must be kept up through the cystoscope.

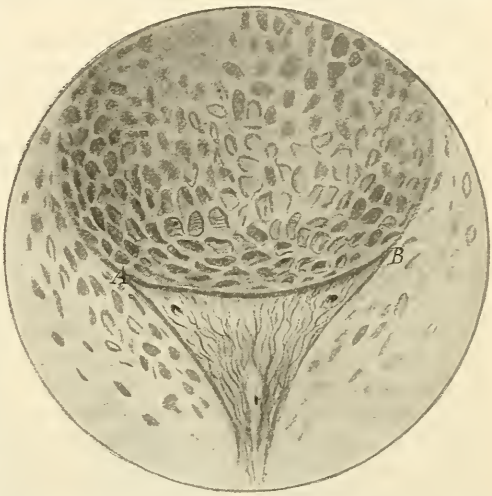



Fig. 869.—The interior of the bladder, showing arrangement of the vessels of the trigone: *A*, *B*, interureteral bar.

*The Ureteral Orifices.*—As before mentioned, the ureteral orifices are usually located at the terminations of the interureteral bar, and occupy the basal angles of a triangle formed by the vesical outlet and the base of the trigone. They are usually about 1 to 1½ cm. from the median line and about 2½ cm. from the vesical end of the urethra. They are recognized as small slits or as tubercles on the surface of the mucous membrane. Their position is not always the same. Some of the abnormal positions more commonly met with are as follows: one

ureter closer to or further removed from the median line, both ureters emptying into the bladder close to the median line, generally about one-quarter or one-half the length of the bar. Malposition toward the vesical outlet and in some very rare instances both emptying on the same side. Sacculation and pocketing of the bladder with swelling of the mucous membrane often make localization of the ureteral orifices difficult. It is then that the administration of methylene-blue or indigo-carmin may enable the investigator to more readily locate the ureters.

After locating a ureteral orifice the vesical end of the cystoscope is carried directly over the opening and the catheter is projected against the opening, where it is allowed to remain for a moment in order to overcome the spasm of the sphincteric muscular fibers. The patient is instructed to hold his breath and the opening is carefully watched until a jet of urine is forced through, during which act the catheter is gently inserted into the ureter. The passing of the urine is accompanied by dilatation of the opening and greatly facilitates the passage of the instrument. After the catheter has passed the sphincter muscle it should be carried up the canal by *very gentle* pressure. The practice of passing the instrument quickly, so commonly resorted to by the novice in order to gain time, is pernicious, and almost invariably causes laceration and hemorrhage. Before inserting the catheter some operators prefer to coat it with paraffin in order to render it absolutely smooth and further to assist in the detection of calculus. This is not necessary. Any instrument with an imperfect eye should be discarded.

The round-pointed catheter should be used wherever possible, as with it there is less danger of injuring the delicate mucous lining of the canal. The sharp-pointed olivary tipped instrument often causes blood to appear. If possible, insert the catheter into the renal pelvis, for then hemorrhage from trauma is less likely to invalidate the examination, and spasm of the ureter, with sucking of the mucous membrane into the eye of the catheter, will not occur. Having finished the catheterization of one side, the catheter is left in place, the instrument handle is swung to the opposite side and a similar procedure is carried out. Always have a distinguishing mark upon the catheters. A very good method is to use different colored instruments, so that there may be no confusion after withdrawal of the cystoscope. In some cases only one ureter can be catheterized. In such a case the bladder should be thoroughly washed, and the other catheter, left on the floor of the bladder, will collect the urine from the uncatheterized side and give a fair idea of the condition of that kidney.

*Removal of the Cystoscope.*—The light should be turned off and a small amount of fluid allowed to escape through the barrel. The lens system carrying the catheter is then loosened and gently rotated from side to side to free the catheters. During this procedure the catheters are drawn gently backward, but not with sufficient force to cause kinking, and the lens system is removed. Next the sheath is withdrawn by carrying it straight up over the abdomen.

**Collection of the Urine.**—The protruding catheters are carefully wiped with sterile gauze and are permitted to drain into separate bottles or test-tubes, marked respectively right and left. The orifices of the receivers should be plugged with sterile absorbent cotton, which will collect and prevent any admixture of urine or water coming from the bladder by capillary drainage. The bottles should also be held in such position as to prevent such capillary drainage reaching them. With regard to the collection of the samples, there are certain precautions that should be observed: (1) The samples should, if possible, be collected in three separate bottles from each side, allowing each set to remain in position a definite time, generally one-half hour. These should be carefully watched and marked first, second, and third half-hours. They should each be

examined separately, and by a summation of the examinations the opinion should be reached. Almost invariably microscopical blood can be found in the urine drawn by catheterization of the ureters. In a study of 15 cases of normal ureters, blood not being present before catheterization after catheterizing with the greatest care, microscopical blood was present in all but 3. The erythrocytes are usually most numerous in the first bottle, gradually diminishing, until in the third there are very few or none. Should the blood increase in amount in the second or third bottle, it is an important clinical fact.

In some instances one or both catheters fail to drain. This may be due to a bubble of air in the instrument, impinging of the mucous membrane upon the eye, plugging by blood-clot, mucus, pus, or particles of gravel. A syringe is then essential. The blunt hollow needle should be inserted into the catheter and suction applied; should this fail, not over 4 c.c. of sterile normal salt solution may be injected through the catheter. This procedure, however, must be carried out very gently, because of the danger of overdilating the renal pelvis and thus producing colic.

The carrying capacity of the pelvis of the kidney is uncertain. Careful observations indicate that there is no definite carrying capacity that may be

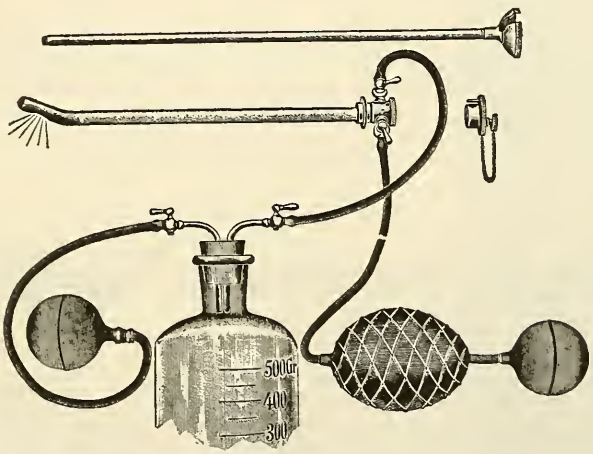



Fig. 870.—Bulbs and bottle for distending bladder and collecting washings.

called normal. The range of difference is great and even differs at certain times in the same kidney. In many kidneys it is about 8 to 10 c.c., but, on the other hand, it may be as high as 20 c.c. and still be normal. It would seem as though the blood-supply of the organ had something to do with the pelvic capacity, for a congested circulation means distended vessels, and they probably impinge upon the pelvis. Again, the question of producing renal colic by overdilatation of the pelvis is a very uncertain guide as to the capacity, for in many instances the ureteral and pelvic musculature is sufficient to force the fluid down the ureter with the catheter *in situ* and yet produce no evidence of colic. This has been seen in many instances in making a pyelographic study by means of a solution of collargol, which is much thicker than urine or water.

A uropyknometer is used for taking the specific gravity of small quantities of urine removed by the ureteral catheter.

**The Operating Cystoscope.**—The most practical operating cystoscopes are Bransford Lewis's and the A. C. M. I. instrument (see Fig. 865). These instruments greatly facilitate the removal of small portions of stone and foreign bodies from the bladder and the ureteral orifices. They are also useful for making ap-

plications to ulcerated areas. It is not wise to remove papillomatous tumors by them, as most villous papillomatous growths sooner or later exhibit the elements of malignancy and should have their pedicles removed with a portion of the vesical wall, which can only be safely done after suprapubic cystotomy. We have succeeded in fulguration of intravesical papillomata of a benign type

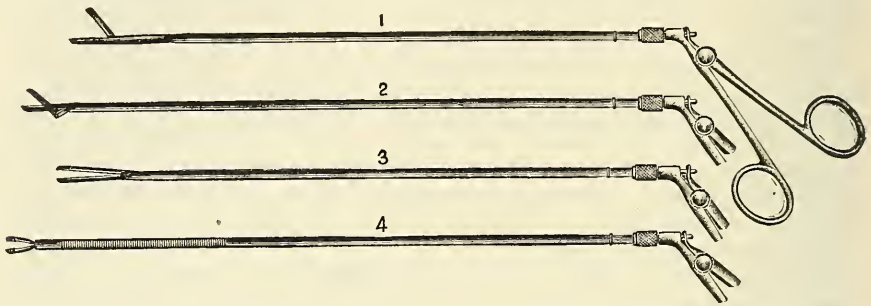



Fig. 871.—1, Scissors; 2, tweezers; 3, forceps; and 4, punch for use with the Lewis's cystoscope.

by means of the high-frequency current, and Prof. Hiram R. Loux has had several such cases that have remained well for over two years.

**Practical Value of Cystoscopy and Ureteral Catheterization.**—The cystoscope has attained a very important position in the surgeon's armamentarium. It undoubtedly affords the best method of establishing an accurate diagnosis

Fig. 872.—"Universal" cystoscope with wire snare.

founded on pathological findings. By it one is enabled to determine the presence and character of cystitis, stone, foreign bodies, and malformations. Without it the presence of ulcerations could merely be guessed at by the clinical history. Its greatest field is in the early diagnosis of the cause of renal and vesical hemorrhages. In the detection of renal neoplasms it may be said to have revolutionized surgery. It makes possible the early removal of such

Fig. 873.—Lewis's repositor.

growths before malignancy has signed the patient's death warrant. Cases of prostatic hypertrophy, in which the lobes project backward, cannot be accurately diagnosticated without the cystoscope; with it the parts can be seen and the clinical diagnosis positively confirmed. It makes possible the removal of small foreign bodies, such as stone, pieces of cotton, or filiform bougie through the urethra, thus saving formidable cutting operations. It is needless to mention the great difficulty found by surgeons in former years in determining the source

of symptomless hemorrhage, a problem which is now comparatively easy of solution.

Ureteral catheterization makes possible the irrigation of the ureter and pelvis of the kidney in cases of ureteritis and pyelitis.

The technic of lavage is as follows: The gravity tubes for instilling fluid, shown in Fig. 874, are carefully sterilized and the metal nozzles arranged so as to fit correctly the lumen of the ureteral catheters. It is always necessary to make certain that the nozzles will fit and that they and the catheters are free. This should never be neglected. It has been found on several occasions that one or the other was blocked, and, again, any particles of catheter or other material that might form a nidus for stone formation should be washed out. We advise the irrigation to be done by means of gravity in preference to a syringe, as the fluid may be delivered more evenly and, so to speak, may be "sneaked" into the pelvis of the kidney. This gentle trickle of warmed fluid does not seem to excite acutely the musculature of the ureter and pelvis; in consequence there is not so much colic, and when colic occurs it does not persist so long. About this point there has been some difference of opinion, but the evidence is in favor of the gravity method both for lavage and pyelography.

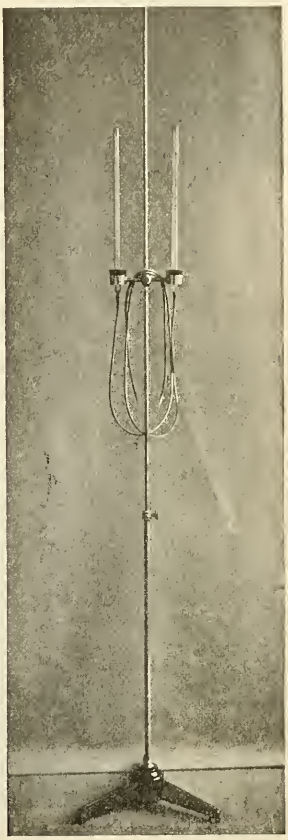


Fig. 874.—Apparatus for filling pelves in pyelography; also for doing lavage of ureters and pelves.

There are several solutions that may be used. We believe that normal salt solution is the best for mere cleansing purposes. Saturated solution of boric acid may be used if a mild antiseptic is needed. Either of these solutions may be followed by the different silver solutions in strengths calculated to suit the case in hand. The nitrate of silver is generally used in a strength of 1:8000, but a much stronger solution may be resorted to. In the clinic of Prof. Loux excellent results have been obtained from a 10 per cent. solution of argyrol.

The catheters are passed into one or both ureters and the fluid is permitted to pass very slowly into the pelvis or ureter, as the case may be. Overdistention must be guarded against, and a safe rule is not to allow over 8 c.c. to be used at any one time.

The type of catheter to be used may be according to the fancy of the operator. There is at present a very excellent two-way pelvic irrigating ureteral catheter, or the single tube instrument may be used and the fluid allowed to escape after filling the pelvis. This procedure should be repeated until the return fluid is clear. Should hemorrhage complicate the procedure, an irrigation of adrenalin may be used. (This has been known to apparently cure essential renal hematuria.)

After completion of the irrigation the bladder should be thoroughly washed and the patient put upon some mild genito-urinary antiseptic, of which there are several to choose from. Urotropin is the one usually given.

It would seem a wise plan to thoroughly irrigate the kidney, pelvis, and ureter before certain surgical operations. When a surgeon is going to open the kidney in the presence of pus or infected urine, before cutting down upon the kidney it is well to wash away as much as possible of the purulent matter

through a ureteral catheter and then instil some nitrate of silver or collargol, which is the silver preparation used in making pyelographic studies.

Irrigation of the pelvis and ureter has come to be a rational surgical procedure and in many cases is followed by a marked improvement in the general health of the individual, making operative procedure safer and not complicated by so marked a toxemia. In all cases when operative work is to be done on a kidney with a large pus sac of other than tuberculous origin, irrigation of the pelvis by means of one of the milder antiseptic silver solutions would seem to be a proper surgical procedure. It is especially so when the necessity may arise for opening the peritoneal cavity.

A differential diagnosis must sometimes be made between gall-bladder disease, appendicitis, and movable kidney with dilated pelvis. This is done by determining the carrying capacity of the pelvis by means of the injection of

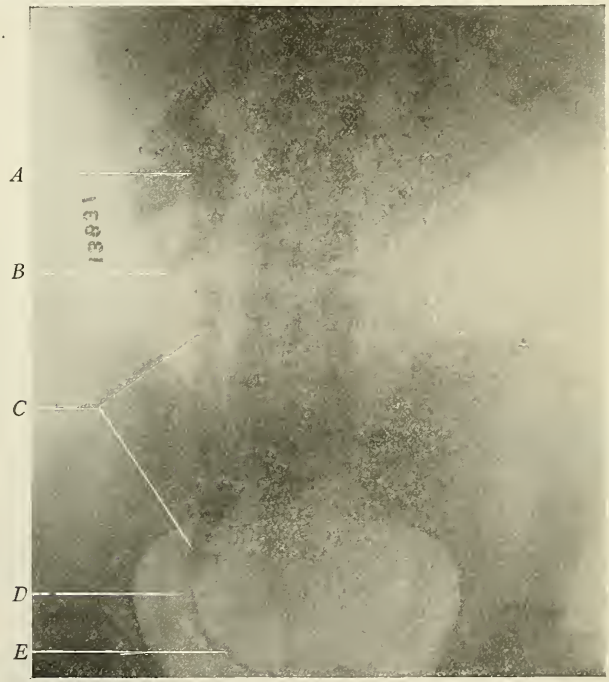



Fig. 875.—*A*, Dilated kidney pelvis filled with collargol solution; *B*, kink in ureter; *C*, dilated ureter filled with collargol solution; *D*, constriction of ureter; *E*, ureteral catheter. (Taken by Dr. W. F. Manges.)

salt solution and by making a pyelograph (Fig. 875). If the trouble is with the kidney, pain caused by injection of salt solution will be similar in character and situation to the pain of the attacks. As a rule, in movable kidney producing marked symptoms the renal pelvis is dilated. When the trouble is due to the kidney the pain induced by the distention is similar to that from which the patient has suffered (Howard A. Kelly). If a stone is present in the ureter the *x*-ray picture will usually reveal it, but here again there may arise doubt as to the exact course of the canal. Catheters passed into the ureters, each catheter with a lead wire in it, can be skiagraphed, and the radiographer will be able to determine the exact course of the ureters. Instead of a catheter containing a wire the *x*-ray catheter of Eynard may be used. Figure 876 demonstrates the practical utility of such manipulation. Phleboliths, fecal masses, appendoliths, concretions, and calcified lymph-nodes have been mistaken for ureteral stone

even after a study of skiagraphs. Appendices have been removed when the source of trouble has been impacted stone in the right ureter. In 3 cases seen by Stellwagen appendectomy had failed to cure. In each case a stone was subsequently removed from the ureter and the patient cured. An impacted stone in the ureter may often be dislodged by passing a catheter into the ureter and, if possible, beyond the stone. A few minims of sterile olive oil are then injected through the catheter, which act as a lubricant and assist in the passage of the stone. Dilatation of the ureteral orifice, by leaving the catheter *in situ* or passing a second instrument alongside of the first, will often cause the stone to pass into the bladder.

**Disinfection of Urethral Catheters.**—Metallic instruments are cleansed by boiling. Soft-rubber and elastic catheters can be sterilized by mechanical cleansing with soap and water and boiling for five minutes. The common custom of immersing a soft-rubber or elastic catheter for five minutes in a 1 : 2000 solution of corrosive sublimate is a useless waste of time, as such a

Fig. 876.—X-ray photograph, showing the course of the ureters by wires in the catheters. (Taken by Dr. W. F. Manges.)

procedure will not sterilize an infected instrument. Formalin vapor is not reliable. A catheter coated with varnish or resin cannot be placed in steam, and cannot be boiled in water if it is allowed to touch the metal of the boiler or another catheter. Woven and varnished instruments can be boiled in salt water (3 drams of salt to 1 pint of water), provided each instrument is wrapped in a piece of gauze so that it cannot touch another instrument or the side or bottom of the metal sterilizer. This plan secures the most certain sterilization. Catheters, after being cleansed mechanically and disinfected, may be kept ready for use in a glass cylinder containing calcium chlorid (R. W. Frank, in "Berliner klin. Woch.," No. 44, 1895). By this plan the catheters can be kept straight. Some prefer to keep them in a glass cylinder containing a few formalin tablets.

An excellent way to keep sterile catheters clean is to place each catheter in an individual bag of linen or waxed paper. Stellwagen boils a number of rubber condoms at the time he boils the catheters. When the instruments

and condoms are dry he encloses an instrument in each condom. Before using catheters which have been in formalin vapor they must be washed in sterile water or salt solution.

## Diseases and Injuries of the Bladder

**Retention of Urine in the Male.**—Retention of urine is not, of course, a disease; it is rather a result of one of a number of different diseases. By this term is meant an absolute inability to micturate voluntarily. The retention may be *complete*, not a drop emerging, or it may have been complete, a dribbling setting in after a time, due to paralysis of the bladder, which viscus becomes unable to contain more fluid, expulsion of the overflow from the ureters being produced by atmospherical pressure. This condition is known as *the engorgement*, *the overflow*, or *the incontinence of retention*. There may be *retained* urine in a man with enlarged prostate, a portion only of the urine being voided. This is not retention, and the urine so retained is called *residual urine*. Of course, true retention may arise in a person with enlarged prostate. Retention may be caused by: (1) *Obstruction*, resulting from urethral stricture, hypertrophied prostate, inflamed prostate, occluded meatus, impacted calculus or foreign body, urethral tumor, rupture of the urethra, perineal abscess, imperforate prepuce, congenital phimosis, tumor of the penis, tumor of the prostate, prostatic abscess, abscess of the penis, ischiorectal abscess, and pressure from a large pelvic tumor. The commonest obstructive cause is spasm of the membranous urethra arising during the existence of stricture, acute gonorrhea, or gleet. (2) *Defective expulsion*, resulting from impairment of the nervous apparatus for inducing micturition. Hysteria is a rare cause in men. We see retention without obstruction after vertebral fractures or spinal concussion, in certain diseases of the spinal cord, sometimes in shock and peritonitis, often in the continued fevers and diseases characterized by muscular wasting, from the action of certain drugs (belladonna, opium, or cantharides), and after certain surgical operations upon or about the rectum. The last-named form of retention is due either to reflex inhibition of the expulsor muscle or to reflex stimulation of the sphincter vesicæ, causing it to remain firmly contracted. *Acute retention* comes on suddenly and is sometimes the first thing that causes a sufferer from urethral stricture to seek a surgeon.

**Symptoms.**—In *acute retention* there is an agony of desire to urinate, the patient making acutely painful straining efforts, during which feces are often passed. There are severe pain and aching in the abdomen, thighs, perineum, and penis. All the symptoms rapidly increase, a typhoid state is inaugurated eventually, and death closes the scene unless relief be given. If retention is from time to time alleviated by the passage of a little water, the symptoms are slower in evolution and are less intense, and the case is said to be *chronic*. Some cases of gradual onset due to atony are very insidious, the patient feeling no particular pain and complaining only of the dribbling, which is really the overflow of retention, and is not a sign that the bladder is successfully emptying itself. In any case of retention the bladder rises above the pubes, and there is found a pyriform, elastic, fluctuating mass in the hypogastrium, which mass is dull on percussion and gradually enlarges until the bladder is evacuated or incontinence sets in. The flanks give a clear percussion-note, and the tumor is more prominent when the patient is erect than when he is recumbent. Long continuation of obstructive disease, producing partial retention with or without attacks of complete retention, disorganizes the kidneys. Acute and complete retention may induce rupture of the urethra or urinary suppression.

**Treatment of Retention of Urine.**—Place the patient upon his back and, if possible, upon a hard mattress. If the bed is so soft that the hips sink down in it, put an ironing board or some other support beneath the mattress at the level of the buttocks. This will greatly facilitate catheterization. Never attempt to use a catheter when the patient is erect; to do so may cause serious or even fatal shock. Always keep the patient protected from cold. Obtain a full history of the case and always make a gentle rectal examination of the prostate; also examine the external genitalia. Failure is often traceable to a lack of thorough examination. It is evident that retention due to prostatic disturbance is not to be dealt with in the same manner as retention due to stricture, impacted stone, etc. If instrumentation does not rapidly succeed, give an anesthetic. Be sure that every instrument is aseptic, and irrigate the urethra before and after instrumentation. Grease the instruments with liquid cosmolin. Prolonged attempts to introduce a catheter and excessive instrumentation are highly dangerous, especially in prostatic cases. Dreadful damage may be inflicted. There is no operation in surgery that requires a gentler touch. Haste, eagerness, carelessness, roughness are, alike, taboo. If a non-medical person knew the facts he would give more thought in the selection of a surgeon to relieve him of retention of urine than of a surgeon to amputate his leg. A surgeon with retention would give grave thought to the question. When the instrument enters the bladder, draw off but half of the urine, withdraw the instrument, wait a few hours, insert it again, and then empty the bladder and wash out the viscus with warm boric acid solution. To draw off all of the urine at once is dangerous, because the sudden relief of the pressure upon distended veins leads to bleeding from the mucous membrane and hemorrhage into the bladder walls. After the bladder has been emptied the patient is wrapped in blankets, a bag of hot water is placed against the perineum, and a hot-water bag is laid upon the hypogastric region. If no anesthetic was used, he is given, at once after the operation, a suppository of opium and belladonna. If an anesthetic was used, he is given the suppository when he recovers from the effect of the anesthetic. Tablets of salol and boric acid are administered during the several days which immediately follow the operation. If the cause of retention is organic stricture, try to pass an elastic, olivary pointed catheter (Fig. 877, *a*). Do not use any force until the neck of the elastic catheter is well engaged in the stricture. Then an experienced operator may warily use a certain amount of force, but never an amount which much exceeds the slightest. If it is found impossible to pass an elastic instrument, make an attempt to carry a filiform whalebone bougie into the bladder. Fig. 878 shows filiform bougies. If the history shows that the man has long had an organic stricture, do not waste time with the gum catheter, but at once proceed to use the filiform bougie. On this bougie, after it has been inserted, Gouley's tunnelled catheter can perhaps be threaded (Fig. 879) and carried into the bladder. Instead of carrying in the catheter, we can simply leave the filiform bougie in place and fasten it. The filiform bougie will act as a capillary drain, and in a few hours will empty the bladder and will also dilate the stricture. Then insert another bougie beside the first, and so on for several days, using also opium, order-

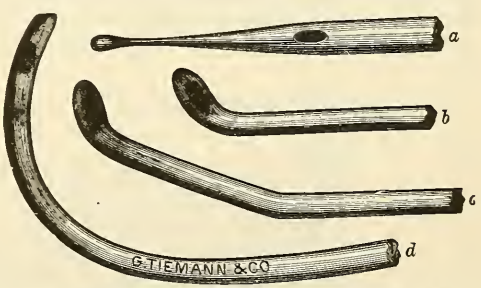



Fig. 877.—*a*, French olivary gum catheter; *b*, Mercier's elbowed catheter (coudé); *c*, Mercier's double-elbowed catheter (bicoudé); *d*, curved gum catheter.

ing rest in bed, and making no attempt to dilate the stricture forcibly until retention has ceased and inflammation has subsided. Perhaps Phillips's catheter (Fig. 883) can be passed. If no instrument can be passed, aspirate above the pubes or perform cystotomy (suprapubic or perineal). In *spasmodic stricture* hold a good-sized metal catheter firmly against the face of the spasmed area; relaxation will occur and the instrument will eventually

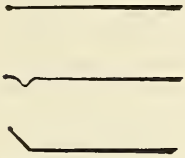


Fig. 878.—Points of Gouley's whalebone guides (filiform bougies).

Fig. 879.—Gouley's tunnelled catheter threaded on a filiform bougie.

pass. Fig. 880 shows the proper curve of a metal instrument. An individual who has an organic stricture which has given but little trouble may develop attacks of retention because of inflammatory edema of the mucous membrane and spasm of the urethral muscles. These attacks are temporary, and an instrument can usually be inserted when employed as above directed. In *inflammation* give a hot hip-bath and suppositories of opium and belladonna, and then use a hot sand-bag to the perineum and a hot-water bag

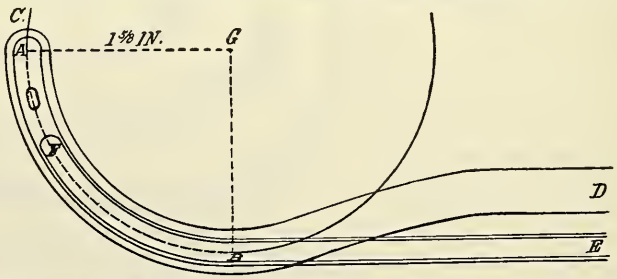



Fig. 880.—*A–B–E* shows the proper curve (reduced in size) for unyielding male urethral instruments; *C–B–D* shows an improper curve.

over the hypogastrium. If these fail or if the symptoms are urgent, pass a soft catheter. In the *occluded meatus of the newborn* incise with a tenotome. In a *congenital cyst of the sinus pocularis* pass a steel bougie, which will rupture the cyst. In *complete phimosis* split up the prepuce. In *impacted stone* try to pull out the calculus with urethral forceps; if this fails, cut the urethra or, in rare cases, push the stone back into the bladder. In *fecal impaction* scrape

Fig. 881.—English silk-web catheter.

out the rectum with a spoon. In *enlarged prostate* the rectal examination gives information as to the type of enlargement. If there is moderate enlargement of the middle lobe the coudé (Fig. 877, *b*) or the bicoudé catheter (Fig. 877, *c*) will probably pass. If these instruments fail, try the overcurved silver catheter of Sir Benjamin Brodie. This metal instrument has a large curve and will probably succeed, but it is a dangerous tool and one capable of inflicting grave injury. In enlargement of one lateral lobe with possible deflection of the

urethra and valve formation try, in order, the woven silk catheter (Fig. 881), the Nélaton catheter (Fig. 882), strengthened by having a filiform passed in its lumen nearly to the beak, and the rat-tailed silk instrument. In enlargement of both lobes and the middle lobe try the coudé, then the bicoudé, then a coudé and a bicoudé with olivary or rat-tailed tips. If all of these fail, the overcurved metal catheter of Brodie must be used gently. In *retention from expulsive defect* use a soft catheter (Fig. 882). Cases of retention after catheterization require warmth, confinement to bed, the administration of laxatives, free action of the skin, and the use of such drugs as salol, boric acid, urotropin,

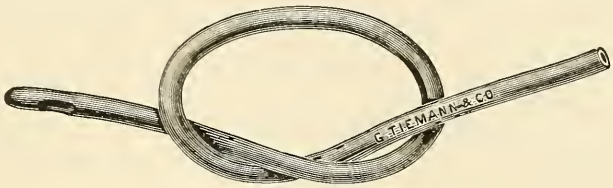



Fig. 882.—Nélaton's catheter.

and quinin to asepticize the urine. In some few cases no instrument can be inserted in the bladder. In most of such cases aspirate—which may be done several times if necessary—and in a day or two, when swelling and congestion abate, an instrument can be passed. The parts are asepticized. A small aseptic trocar or aspirator needle is pushed into the bladder, the trocar or needle being inserted in the median line, just above the pubes, and taking a course downward and backward. After the completion of the operation the puncture is dressed with iodoform and collodion. Only half the urine is with-

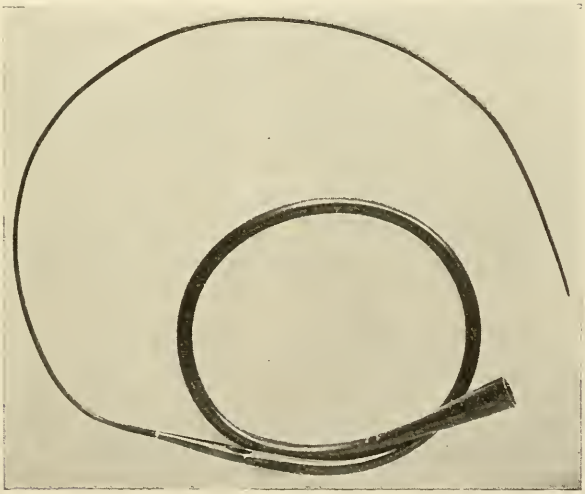


Fig. 883.—Phillips's catheter.

drawn at a first aspiration. Rectal puncture is now obsolete. If incision is necessary in retention, the perineal route is usually chosen. In some cases the operation is done with, in some without, a guide. In prostatic retention not relievable by a catheter, make suprapubic drainage or do prostatectomy.

**Congenital Defects of the Bladder.—Exstrophy of the bladder** (*ectopia vesicæ*) is a condition of defective development commoner in males than in females. The anterior abdominal wall has failed to close, the anterior wall of the bladder is absent, the arch of the pubes has not developed,

epispadias exists, and in many cases the testicles have not descended into the scrotum. In this condition the posterior wall of the bladder projects into or beyond the gap in the abdominal wall; the urine constantly flows and renders the condition of the patient dreadful. The condition shortens life and only 30 per cent. of the victims live beyond the twentieth year, death being due to pyelonephritis.

The only treatment which offers hope is operation, and operation often fails. If possible, operate when the patient is about five years of age. Various operations have been suggested for this condition, viz.: covering with skin-flaps; implanting the ureters into the bowel (Maydl, Albert, Roux, Simon, and others); division of the posterior ligaments of the sacro-iliac joints, bringing the arch of the pubes forcibly together, the patient wearing a support until the parts become firm, when the greatly narrowed defect is closed in by integumentary flaps and suturing the bladder edges (*Trendelenburg's operation* or *synchondroseotomy*); osteotomy through one ilium or both ilia instead of separation of the sacro-iliac joints (*Berg's operation*), or after extirpating the bladder, loosening the ureters from the bladder, drawing them down and attaching them to the end of the penis (*Sonnenberg's operation*).

A bladder closed in by autoplasty, by Trendelenburg's operation, or by Berg's operation is never really continent, although when the patient is erect and wears a light compress he may perhaps be able to hold water two or three hours. The above methods are suited to young children and are far safer than ureteral transplantation. Tuffier showed, nearly twenty years ago, that transplantation of the cut ureters into the bowel was certain to be followed by fatal infection of the kidney, and that the only way to prevent this was to retain the ureteral orifices which contain valves.

Maydl introduced his operation in 1892. He implanted the trigone with the ureters into the sigmoid flexure, extirpating the rest of the bladder. I have twice performed a modified Maydl's operation, with one death and one recovery. Buchanan ("Surg., Gynecol., and Obstet.," Feb., 1909) has collected 80 cases. In this collection there were 23 deaths (28.7 per cent.); 7 died of peritonitis and 9 of pyelonephritis. Bregenhem in 1894 devised extraperitoneal implantation of the ureters and a portion of bladder through two separate openings into the rectum.

**Diseases and Injuries of the Bladder.**—This viscus is so deeply situated, and the abdominal walls are so elastic, that it is rarely injured when empty. If the bladder is full and the abdomen tense—which is common in alcoholic intoxication—force applied upon the abdomen may injure the bladder.

**Contusion of the Bladder.**—In this condition there are noted vesical hematuria, tenesmus, severe cystitis, and an impediment to the flow of water because of clots. Hemorrhage may be very severe and sepsis may arise, even causing death. When contusion exists retention is relieved by means of a clean soft catheter; if this fails because of occlusion of the eye of the catheter with blood-clot, at intervals there must be passed through the catheter from a fountain-syringe a solution of sodium bicarbonate in cooled boiled water. Gross's blood-catheter can be used or the evacuator of Bigelow may be employed. The patient is put to bed, a hot-water bag is applied to the hypogastrium, morphin is administered in moderate doses, the bladder is washed out several times a day with boric acid solution or a solution of bicarbonate of sodium to disintegrate and remove blood-clots, and the urine is diluted and rendered aseptic by the stomach administration of salol, boric acid, and the free use of bland fluids. Hemorrhage usually ceases on relieving distention; if it does not, some more radical measure must be employed (see Hematuria).

**Wounds of the Bladder.**—Besides being contused, the bladder may be injured by bullets; by stabs or punctures through the abdomen, the vagina, or the uterus; or by penetration by a fragment of a fractured pelvic bone. The symptoms of such conditions are those of rupture of the bladder (*q. v.*). In an intraperitoneal wound at once open the abdomen, suture the wound in the bladder wall, irrigate and drain the peritoneal cavity, and drain the bladder by means of a retained catheter, perineal section, or suprapubic cystotomy. In an extraperitoneal wound drain the wound by a tube, and drain the bladder by a retained catheter, perineal section, or suprapubic opening.

**Rupture of the bladder** occurs in three forms: (1) intraperitoneal—a rupture involving the peritoneal coat; (2) extraperitoneal—a rupture of a portion of the bladder not covered by peritoneum; and (3) subperitoneal—a rupture of the mucous and muscular coats, the urine diffusing under the peritoneal investment. The *causes* are of two kinds, predisposing and exciting. *Predisposing* causes are: distention of the bladder; drunkenness; cystitis; ulceration; degeneration or atony of the bladder-coats; prostatic enlargement, and urethral stricture. Distention of the bladder is the great predisposing cause. It causes the bladder to rise from the pelvis and so become exposed to a direct blow, it places the organ under tension, and force tends to rupture the weakest point. In about one-third of the cases collected by Bartels the individual was intoxicated at the time of the accident. Drunkenness predisposes because a drunken man is very liable to injury and is apt to have a distended bladder and a rigid belly wall. Males are much more liable to rupture than females (10 to 1). Most cases are between the ages of twenty and forty. Of Besley's 25 cases, 1 was a child of three, 1 a man of forty-nine, 3 were in the first and second decades, 5 were between twenty and thirty, 7 between thirty and forty, 6 between forty and fifty, and in 2 the age is not given (paper before Chicago Surg. Soc., Feb., 1907). The condition is very rare in children, but 1 of Besley's cases was three years of age, and King recorded the accident in a fetus with imperforate urethra.

*Exciting* causes are: obstruction to the outflow of urine (by stricture or enlarged prostate); external violence; falls upon the feet or the buttocks, as well as upon the abdomen; lifting; straining at stool, in micturition, or during parturition; and the forcing of injections into the bladder. A distended bladder may be ruptured by a concussion. The most usual cause of the injury is a crush. The mechanism of the injury is in dispute. It is certain that the bladder must have lost its elasticity by distention. When force is applied to fluid (fluid is incompressible) the bladder tears at its weakest point. The weakest point is not identical in all individuals. It may be weak anywhere from disease. The most common site of the tear is at the posterosuperior aspect, but it may be in front, at either side, or at the pubic or prostatic ligament (Staubenranch). The mucous membrane or the peritoneum may give way first and the tear may be anteroposterior, oblique, or longitudinal. Alexander maintains that the most usual cause of the injury is a crush which forces the distended bladder against the sacral promontory, but Besley's (Ibid.) experiments do not indicate that this is correct. A common complication, especially of extraperitoneal rupture, is fracture of the pelvis, due to the same force that ruptured the bladder.

*Symptoms, Diagnosis, and Treatment.*—The symptoms are not always definite, and every characteristic one may be for a time absent, the patient seeming in some rare instances of extraperitoneal and intraperitoneal rupture to possess the power of retaining his urine and of voiding it. As a rule, however, there are found some or all of the following symptoms, following an accident or occurring during the progress of a causative disease: severe pain in the bladder and in the suprapubic region, collapse; inability to walk or great difficulty

in walking; excessive desire to micturate, but inability to do so (sometimes a little pure blood or bloody water is squeezed out by painful effort); a catheter, when used, brings away pure blood or a very little bloody urine; the catheter occasionally slips through the tear into the cavity, and more bloody water comes away. In some reported cases clear water has been withdrawn. If a measured amount of boric acid solution is injected, it is improbable that all of it can be withdrawn by the catheter, although in some cases it may all come away (Alexander, in "Annals of Surgery," August, 1901). Injecting fluid fails to lift the bladder into the hypogastric region so as to be recognizable on percussion. In a patient suffering from retention of urine in whom rupture occurs there is first a temporary sense of relief from retention, but very soon severe hypogastric pain and rectal tenesmus. In intraperitoneal rupture reaction may be obtained after a few hours or a number of hours. The evidences of peritonitis will be noted soon (rapid pulse, perhaps vomiting, rigidity, distention, obstruction of the bowel, elevated temperature, etc.). Shock in vesical rupture is so severe that death may ensue; if reaction follows, there may be delirium and often septicemia; extensive infiltrations of urine may occur. In *intraperitoneal rupture* general peritonitis is certain to arise, but its appearance may be postponed for several days if the urine is healthy. In these cases the extravasation is noted as a simple swelling, probably on one side only. In *extraperitoneal rupture* the urine may infiltrate the perineum, the scrotum, the thighs, and under the integuments of the abdomen and the back, and may soon induce sloughing. In *subperitoneal rupture* peritonitis is apt to arise.

In doubtful cases some surgeons pump air or hydrogen into the bladder. To insert air a bicycle pump can be used (Brown) or a Davidson syringe (Keen). Keen's directions are to insert a catheter, empty the bladder of urine, and connect to the catheter a disinfected Davidson's syringe, a mass of absorbent cotton being fastened over the distal end of the syringe. Air after it has filtered through the cotton is pumped into the bladder; an unruptured bladder will rise above the pubes as a pyriform tumor, tympanitic on percussion. A ruptured bladder will not so rise. In intraperitoneal rupture the air will pass into the general peritoneal cavity and distention will occur. In extraperitoneal rupture injection will produce emphysema of the extravesical connective tissue. On removing the syringe the air rushes out again if the bladder is unruptured, but little if any comes away if it is ruptured. Alexander considers gaseous distention unreliable, and claims that it adds to shock and disseminates infection. His rule is the wisest to follow; that is, in a case of suspected rupture of the bladder, make a suprapubic incision and inspect the prevesical space for signs of extraperitoneal rupture. If extraperitoneal rupture is not found, open the belly and explore.

*Treatment.*—In extraperitoneal rupture, after incision down to the bladder insert a drainage-tube. In intraperitoneal rupture, place the patient in the Trendelenburg positon, expose the tear in the bladder by abdominal incision, and suture the opening in the viscus.

*Results.*—Baron Larrey was the first surgeon to state that a wound through all the coats of the bladder might be followed by recovery. In intraperitoneal ruptures if operation is not performed the mortality is 98 per cent. If operation is performed many cases recover. Of the 78 cases collected by Dambriu and Papin in 1904, 34 died, a mortality of 43 per cent. (Besley, paper before Chicago Surg. Soc., Feb., 1907). Galactionoff ("Rovssky Vratch," Nov. 12, 1910) reports 15 cases operated upon for intraperitoneal rupture; 5 recovered (3 of them were operated upon during the first twenty-four hours; 1, after thirty-six hours, and 1, after forty-eight hours). In extraperitoneal rupture without operation there are 11 per cent. cures and with operation 30 per cent.

(See Daniel N. Eisendrath, "Jour. Amer. Med. Assoc.," Oct. 25, 1902; Samuel Alexander, "Annals of Surgery," Aug., 1901.)

**Atony of the bladder** is a condition in which the expulsive power of the bladder is diminished or lost because of impairment of muscular tone. The bladder is very thin, and the muscles are flaccid and often the seat of fatty degeneration. Sometimes the viscus is very large and sometimes it is very small. A slight degree of atony is physiological after middle age.

The **causes** are senility, distention from true paralysis, chronic overdistention from obstruction, and acute overdistention. In most cases there is obstruction of the urethra. In that rare condition known as *idiopathic atony* there is no evidence of obstruction. Walker ("Annals of Surgery," Nov., 1910) reports 12 cases of idiopathic atony. He says the patients were between the ages of twenty-two and forty. He regards the condition as due to a lesion in the reflex centers.

**Symptoms.**—In atony of the bladder the patient passes water frequently (a symptom probably existing for some years), and especially at night; he may even do so while asleep. The stream, when voluntarily passed, has little projection, but seems to drop at once from the end of the penis. Residual urine exists for years and may at any time set up cystitis, and retention with incontinence is apt to occur. This condition is *not* vesical paralysis resulting from a lesion of the nervous system.

**Treatment.**—In treating atony of the bladder measure the residual urine: if it amounts to 4 oz., use a soft catheter night and morning; if it amounts to 6 oz., use the catheter every eight hours; if it amounts to 8 oz., use the catheter every six hours (J W. White). The patient should be taught how to use the catheter and how to keep it sterile. (For Methods of Disinfecting Catheters see article on page 1311.) The bladder is from time to time washed out with 3 gr. to the ounce of boric acid solution at a temperature of 100° F. Strychnin, electricity, ergot, and urotropin may be ordered.

**True Paralysis of the Bladder.**—Vesical paralysis results from a lesion of the nervous system causing paralysis of the motor nerves or of the motor paths from which they are prolongations (fracture of the vertebræ, spinal meningitis, syphilis of the cord, myelitis, and hemorrhage about or into the spinal cord). A traumatic paralysis comes on suddenly. Hysteria may be responsible for temporary palsy. If the detrusor muscles alone are palsied there is complete retention of urine. If the sphincter is paralyzed the urine dribbles constantly. Even when there is dribbling a quantity of urine is retained below the level of the internal meatus. In such a case there is incontinence with partial retention (the *overflow* of Sir Henry Thompson). If the patient sits down or assumes the knee-chest position he may empty the bladder by contracting the abdominal muscles. In cases of real retention the detrusors may be temporarily paralyzed. In such a case the sphincter may finally relax from fluid pressure, and atmospherical pressure or contraction of abdominal muscles may cause urine to dribble, the bladder remaining full (the *overflow of retention* of the elder Gross). In some cases of vesical paralysis there is retention; in some cases, incontinence of urine. If the sensory as well as the motor path is involved in a lesion, the patient has no sensation to notify him of dribbling or of retention.

**Treatment.**—Treat the cause. Employ regular aseptic catheterization by a soft instrument. In some cases the bladder may be subjected to faradism (one electrode in the bladder and one in the perineum or on the abdomen above the pubes). Kilvington ("Brit. Med. Jour.," 1907, vol. i) pointed out that the nerve-supply of the bladder and rectum is from the second and third and sometimes also from the first sacral nerves. He suggested treating some otherwise incurable cases of vesical paralysis by anastomosing a nerve-root above

the lesion to certain sacral nerves below the lesion. Bird did the first operation, but it failed. Mills and Frazier (see page 863) performed intradural anastomosis of the last lumbar nerve to the third and fourth sacral nerves. The patient was much benefited ("Jour. Amer. Med. Assoc.," Dec. 21, 1912).

**Foreign Bodies in the Bladder.**—The term "foreign body" is, at best, a poor one, since a stone is a foreign body. The term, however, is generally used to designate material held within the viscus and of purely extraneous origin. There have been a great variety of foreign bodies removed from both the male and female urinary bladder. We have seen a lead-pencil, hair-pins, a willow twig, a glass rod, and a piece of chewing gum removed, as well as portions of catheters and filiform bougies. For reports of foreign bodies found in the bladder the reader is referred to text-books upon genito-urinary diseases.

Most of the foreign bodies are found in the bladders of masturbators. In them the glans penis or the clitoris have become so inured to friction that they must seek deeper for sensation, and consequently men resort to titillation of the verumontanum and posturethra, and women, to irritation of the vesical neck. The object used to stir sensation may slip from the grasp and pass backward into the bladder. The careless use of the filiform bougie is a great danger. Always test the filiform for defects and for tensile strength before using it. Should you be using the Gouley catheter, see that the tip through which the filiform must lace is not sharp or square, else it may shear off a portion of the bougie. All foreign bodies sooner or later induce cystitis. Every foreign body if allowed to remain will become encrusted with urinary salts and form a nucleus for stone formation.

**Diagnosis, Symptoms, and Treatment.**—*Diagnosis* is made by the history, if given correctly, of the case. The stone-searcher, the *x*-ray, and the cystoscope are most useful in diagnosis. The *symptoms* are similar to those of stone in the bladder. The *treatment* is removal, with subsequent care of the resultant cystitis.

**Vesical Calculus, or Stone in the Bladder.**—The salts normally in solution in the urine may deposit as calculi and may be imprisoned in any portion of the urinary tract. The commonest primary calculi are those composed of uric acid, urates, calcium oxalate, and fusible phosphates. In 80 per cent. of cases primary calculi are composed of uric acid and urates. A primary calculus may become coated with another material (secondary calculus). The formation of uric acid and urate calculi is explained under Renal Calculus (see page 1283). Vesical calculi are usually renal calculi that have passed the ureter and become enlarged by new accretions. New accretions from an alkaline urine will cause the formation of a secondary phosphatic stone. Phosphatic calculi may be formed in the bladder when chronic cystitis causes and maintains an alkaline urine. Uric-acid calculi are smooth, round or oval, and hard, but easily broken. On section they present the color of brick-dust and are marked by concentric rings. Their nuclei are dark by comparison. They are soluble in dilute potassium hydrate and in nitric acid. They are combustible and leave scarcely any ash. Urate of sodium and urate of ammonium often occur together in stones, and these calculi are not in rings, are not so hard as the uric-acid stones, and are fawn colored on section. Oxalate of lime stones are round, with many projecting nodes like the mulberry, hence the term "mulberry calculus." They are very hard, and section shows the color to be brown or green and that they possess wavy, concentric rings. This form of calculus is soluble in hydrochloric acid. The so-called fusible calculus of the early writers, which is composed of a phosphate of magnesium, ammonium, and calcium (*triple phosphate*), constitutes the commonest form of phosphatic stone and of large stone. It is light, soft, smooth and white, and shows no laminæ on section. Some rare forms of primary stone are composed of xan-

thin, cystin, indigo, urostealith, calcium phosphate or calcium carbonate, or blood concretions.

A stone having layers of different substances may be formed; for instance, there is often found a uric-acid nucleus surrounded by phosphates, the latter surrounded by some uric acid or urates, and these again by phosphates. In some cases oxalate of lime alternates with uric acid, urates, or phosphates (Bowlby). Bowlby states that the alternating uric-acid and phosphatic layers are due to the altering reactions of the urine; that when the urine is acid uric acid is deposited on the stone, but when cystitis makes the urine alkaline the stone receives a phosphatic coat.

Anything that favors the formation of an excessive urinary deposit may cause vesical calculus, and among such causes are defective digestion, failure in processes of oxidation, excess of solids and nitrogenous elements in the diet, deficient exercise, etc. If to the urinary condition established by the above factors catarrh of the genito-urinary tract is added, pus or mucopus in the

Fig. 884.—Stone in bladder shown by *x*-rays.

concentrated urine may induce stone. Children are predisposed to uric-acid stones, and old people to phosphatic stones. In an old man with enlarged prostate and chronic cystitis a stone forms rapidly about any accidental nucleus. The nucleus may be phosphate crystals glued together by mucus, a blood-clot, uric-acid gravel, or a foreign body. Stone is rare in females because of the shortness, the large diameter, and the ready dilatability of the urethra. Stone is very rare in the negro. Gout, rheumatism, lithemia, enlarged prostate, vesical atony, urethral stricture, and catarrhal inflammation of the kidney, the ureter, and the bladder are predisposing causes.

**Symptoms.**—In not a few cases the vesical symptoms are antedated by an attack of nephritic colic. Hence the necessity for cystoscopy after renal colic if no stone passed from the meatus, and the removal of any retained stone by the evacuator or the cystoscope. The severity of the symptoms of stone in the bladder depends more on the roughness of the stone than on its size. A small, rough calculus will produce intolerable anguish, whereas several large, smooth

stones will cause but moderate pain. A patient with stone in the bladder complains of frequency of micturition, particularly in the daytime, the desire being sudden, uncontrollable, and invoked or aggravated by exercise. This symptom is more positive in youth than in old age. Pain of a sharp, burning character is experienced at the end of micturition, due to the contraction of the empty bladder upon the stone or stones. It disappears gradually as urine enters and distends the bladder. The usual seat of this pain is the under surface of the head of the penis, a little behind the meatus, and the pain may continue for some time. By pulling on the penis to relieve this pain the prepuce of a child may become pendulous. The pain varies in severity, being much worse during an attack of cystitis and after exercise; it may be absent in encysted stone; it may be present early in a case, but almost disappear as a case progresses, and it is always worse in the young than in the old. Stone in chronic cases of atony and in cases of vesical paralysis causes neither marked pain nor frequency of micturition.[1] In a case of enlarged prostate pain *precedes* the act of micturition, in urethral stricture it *accompanies* it, and in stone, as already stated, it *follows* it (P. J. Freyer, in "The Practitioner," Feb., 1898). The symptoms may be somewhat confused by the coexistence of vesical calculus and prostatic hypertrophy. Attacks of cystitis in a man with calculus are spoken of as *attacks of stone*. When a stone is small, it may during micturition roll into the urethral orifice, and so cause a sudden interruption of the flow of urine, the stream again starting when the patient changes his position. This symptom is seldom met with and is particularly rare in the old, the stone in them dropping into the sac back of the prostate

Fig. 885.—Thompson's calculus sound.

and *below* the urethral orifice. Even if this symptom occurs, it is not conclusive, as a stalked tumor, a blood-clot, or a mass of pus or mucus may block the urethral orifice and cut off the stream. Hematuria may or may not be noted; it is most usual after exercise, and occurs at the end of the urinary act, the first urine passed being clear, the later urine being blood-tinged, and at the end of the act some drops of pure blood emerge. It is not one of the earliest symptoms. When it occurs it puts the patient in a great fright. It does not appear suddenly and profusely, but as gradual and trivial bleeding and with micturition. Blood appearing between acts of micturition comes from either the urethra or prostate (P. J. Freyer). The bleeding from a bladder tumor is profuse and the urine is mixed with blood and blood-clots and tumor fragments. Bleeding from a tuberculous ulcer of the bladder often resembles the bleeding caused by stone. Pus or mucopus will be observed if cystitis occurs with calculus disease. Priapism occurs in some cases. Pain of a reflex nature may be felt in the rectum, in the perineum, or in some distant part.

The above symptoms, even if all are present, do not prove that an individual has a stone in the bladder. To prove the presence of a stone the object must be pictured by the *x*-rays, seen through a cystoscope, or be touched by a sound. Simple touching by a sound is not sufficient, the contact must be felt and heard. To sound a patient, have the bladder well filled with boric acid solution or salt solution, and place him recumbent, with the knees drawn up. Never sound a person while he is standing, because of the danger of syncope. In an ordinary case

[1] "American Text-Book of Surgery."

in a male use a sound with a very slight curve (Fig. 885); in a man with hypertrophied prostate use a sound with a short and decided curve. The caliber of a stone-sound is No. 13 of the French scale. The instrument is carefully boiled and anointed with yellow liquid cosmolin. Examine the entire bladder systematically, and be sure a stone is present only when contact with the sound is both heard and felt. The stone may be difficult to find, or it may elude the instrument entirely when it is encysted, when it rests in a diverticulum, when it is fixed to the roof or anterior wall of the viscus, or when it is crusted with lymph or blood-clot. In doubtful cases always insist on a second examination, giving ether if the first was very painful. Occasionally, as Freyer pointed out in 1884, a small stone will be found by using a Bigelow evacuator, the current causing the calculus to knock against the tube. In many cases stone in the bladder may be detected by means of the *x*-rays (see Fig. 884). Use the cystoscope in all cases of suspected stone. If a stone is fixed in a diverticulum or projects from the ureter, or is in a sac back of the prostate, it may be missed by sound and evacuator tube, but be shown by the *x*-rays and the cystoscope. A stone, when it is detected, should always be measured by Thompson's instrument, an arrangement looking something like a small edition of a lithotrite, but having very delicate blades. The composition of the stone is assumed from an examination of fragments which pass by the urethra or which adhere to the measure. Remember that the outer layer of a calculus may be soft phosphate and the inner portion may be the harder uric acid, urate, or oxalate.

**Stone in Females.**—Calculus in the female is a rare complaint. In over 900 patients operated upon by Freyer for stone there were only 20 females. Pain and increased frequency of micturition, which are symptoms of stone in men and women, are in women commonly caused by other conditions, notably by uterine disease and displacement. A straight sound is used to examine a female for stone. If the surgeon is still uncertain after sounding, he uses a cystoscope or dilates the urethra and explores the bladder with his little finger.

**Stone in children** can occur at any age, and congenital cases have been placed on record. The uric-acid stone is most common. The symptoms are like those of the adult. The pain causes the male child to pull at the penis and the prepuce becomes pendulous. If in a child with stone the stream of urine is blocked from time to time, the child strains to empty the bladder, and after a time a hernia may form or prolapse of the rectum take place.

**Treatment.**—In people predisposed to stone (for instance, by lithemia) the physician should forsee the danger and antagonize it. Insist on the urine being kept dilute by the freest use of water and of milk, and reduce to a minimum the amount of alcohol, meat, sugar, and fat which is taken. Let the patient live chiefly on green vegetables, salads, bread, fruit, eggs, fish, poultry, weak tea or coffee, water, milk, and, if desired, a little red wine. Continued purging does harm by concentrating the urine, though a laxative may be employed when indicated. Moderate open-air exercise is of immense importance, sunshine and fresh air being Nature's correctives for a condition of imperfect oxidation power. If the urine be very acid, use piperazin, 15 to 20 gr. daily, liquor potassii citratis, phosphate of sodium, or borocitrate of magnesium. If the urine be phosphatic and alkaline, order mineral acids and strychnin, or, what seems to be very efficient, urotropin. Urotropin is given in 5-gr. capsules four times daily. If the urine be filled with oxalate, use the mineral acids with an occasional course of phosphate of sodium. Travel and rest at the seaside or at some spa are often of service in all forms. Always endeavor to prevent cystitis, and treat it promptly when it does occur. When a stone is once formed, it is an idle dream to think of dissolving it. An operation must be done. Some very small stones may be

crushed in view through the cystoscope by one of the several intravesical forceps, but in an immense majority of cases a very much more formidable operation is required. The operation selected depends upon the age, the state of the bladder and the prostate, the dilatability of the urethra, the kidney condition, the size and composition of the stone, and the number of calculi present (see Operations on the Bladder).

**Bacteriuria.**—Most urines contain bacteria. If bacilli are present in numbers, the condition is called *bacilluria*. The variety and number vary greatly in different individuals. In typhoid fever typhoid bacilli are found. In many cases of nephritis bacteria are present. In pulmonary tuberculosis, vesical tuberculosis, and renal tuberculosis bacilli may be found in the urine. In some cases numerous colon bacilli are found. They may seem to do no harm; they may cause nephritis; they may cause cystitis.

If a urine contains bacteria, inflammation of the genito-urinary tract may or may not exist, but even if it does not exist it is apt to occur at any time. In some persons the urine is found swarming with colon bacilli. Colon bacilli are capable of causing a very severe type of cystitis and nephritis. Bacterial urine explains many cases of urinary fever (see page 1369).

**Cystitis.**—Inflammation of the bladder is, as a rule, a complication of some other disease of the genito-urinary tract, but it may arise after exposure to cold and wet. Traumatism from a catheter, the presence of a stone, the spread of a urethral inflammation, pus infection, vesical tuberculosis or cancer, and the use of such drugs as cantharides, turpentine, alcohol, urotropin, and sandalwood oil in large doses may produce it. It appears not unusually during an exanthematous fever or in conditions of vesical paralysis; it often follows retention; frequently accompanies enlarged prostate and urethral stricture, and sometimes arises from concentration of urine or accompanies bladder growths. Acute cystitis causes discoloration and swelling of the bladder walls, and there is present a catarrhal discharge which is mixed with urinary elements, serum, mucus, often pus and epithelial débris. Ulceration, sloughing, or false-membrane formation may occur. Chronic cystitis is an inflammatory condition always due to bacteria. We frequently speak of a chronic cystitis as due to stone in the bladder, hypertrophy of the prostate gland, or tumor of the bladder. These conditions do not cause chronic cystitis, but act by rendering the bladder vulnerable to micro-organisms. Among the causative organisms we may mention the Bacillus coli communis, the gonococcus, the Bacillus tuberculosis, the Bacillus typhosus, the Urobacillus liquefaciens septicus, and the various pyogenic bacteria (Leonard Freeman). These bacteria may gain entrance on instruments or by way of the ureter, urethra, the lymph-vessels, and, in rare instances, by the blood.

In chronic cystitis there is an enormous production of thick, sticky mucus and the urine becomes alkaline. The excessive secretion of mucus and the great number of bacteria convert the urea into carbonate of ammonium, and this product, being irritant to the bladder walls, makes the inflammation worse. In chronic cystitis the bladder is contracted and has very thick walls, and the mucous membrane is thick, edematous, congested, and filled with large veins. The bladder may be ulcerated or encrusted with urinary salts. The urine contains bacteria, triple phosphate, pus, blood, and mucus, the blood emerging with the last drops of urine. Pyelitis may arise as a result of chronic cystitis.

What was formerly called inflammation of the neck of the bladder is an inflammation of the vesical trigone and of the posterior urethra. It is now usually called *urethrocystitis* or *trigonitis*.

**Symptoms of Acute Cystitis.**—Great frequency of micturition, with the passage, at each act, of a very small quantity of urine; the desire to urinate is

almost constant, and there is intensely painful straining (*tenesmus*). The pain is acute and scalding, and may be felt in the head of the penis, above the pubes or in the perineum; it often runs into the loins and the thighs and radiates over the sacrum. Pain above the pubes indicates involvement of the fundus, and pain in the perineum and in the head of the penis points to inflammation of the bladder trigone. The urine, at first clear, loses its transparency, becomes full of thick mucus, and often contains a little blood or pus. The patient not unusually has some fever. A rectal examination causes violent pain. If retention arises, there will probably be a chill and high fever, and anuria may occur.

**Treatment.**—In treating acute cystitis endeavor to remove the cause. By allaying an irritation or removing an obstruction the bladder will often become able to empty itself of retained urine, which urine causes congestion of the bladder and thus renders infection probable or may be itself filled with bacteria. If cystitis arises after the administration of cantharides, put the patient in bed and give him liquor potassii citratis. If it arises after the use of a clean sound, order rest in bed, suppositories of opium and belladonna, diluent drinks, and ammonii benzoas or lupulin. If the inflammation is septic (as from the use of a dirty sound) or is very acute, put the patient in bed, keep him warm, and use a hot sand-bag to the perineum and hot fomentations or poultices to the hypogastrium. Hot hip-baths may be used. The hips should be elevated, and the bowels should be emptied by the administration of salines and by glycerin enemata. An exclusive milk-diet is desirable. The patient should drink copiously of sweetened water containing a few drops of aromatic sulphuric acid or of milk of almonds. Sterilize the urine by the administration of urotropin, giving a capsule containing $7\frac{1}{2}$ gr. of the drug three times a day. Other remedies which may be of service in sterilizing the urine are quinin, boric acid, salol, borocitrate of magnesium, and salicylate of sodium. A valuable remedy consists of 15 gr. of salicylate of sodium and 15 gr. of benzoic acid, given three times a day in a little chloroform water. If the pain and straining still continue, order—

℞. Ext. hyoscyami..... gr. viij;
Ext. cannabis indicæ..... gr. viij;
Sacchar. alba..... gr. xlviij.—M.
Div. in pulv. No. xxiv.
Sig.—One powder every four hours.

Or,

℞. Camphoræ..... gr. viij;
Ext. cannabis indicæ..... gr. viij;
Sacchar. alba..... gr. xlviij.—M.
Div. in pulv. No. xx.
Sig.—One powder every three hours. (Von Zeissl.)

Suppositories of extract of belladonna are of great value. Suppositories each of which contains 1 gr. of ichthyol are of service, and one may be used every four hours. Opium, unfortunately, constipates; when it is given, secure evacuations by the use of glycerin suppositories, by the administration of saline cathartics, or by the employment of enemata. If opium is necessary, it is given in a suppository containing 1 gr. of powdered opium and $\frac{1}{6}$ gr. of the extract of belladonna every three or four hours. Hypodermatic injections of morphin may be required. Wash the bladder out daily with warm normal salt solution or warm boric acid solution. This can be done through a soft catheter or, better, by hydrostatic pressure. If retention occurs, use a soft catheter. If much blood is passed, give internally the tinctura ferri chloridi and blister the perineum. In urethrocystitis (trigonitis) the instillation of solutions of

nitrate of silver (5 to 10 gr. to the ounce) often do good. A very acute cystitis is rarely arrested within a week or ten days.

**Symptoms of Chronic Cystitis.**—This condition may be a legacy from acute cystitis or it may appear without any acute precursory phenomena. There will be found frequency of micturition, but not so great as in the acute form. There will be slight tenesmus and moderate pain from time to time, usually radiating toward the head of the penis. Constitutional symptoms may arise when kidney damage has become pronounced or sepsis has occurred from absorption. The urine is ammoniacal, fetid, and turbid; it is filled with viscid, tenacious mucus or with mucopus; it contains a great excess of phosphates, and occasionally clots of blood. The condition of chronic cystitis with the production of immense quantities of thick mucus is often called *chronic catarrh of the bladder*. Chronic cystitis may eventuate in the formation of stone or in the production of serious disease of the bladder, the ureters, and the kidneys. It is very apt to cause retention of urine.

**Chronic Tuberculous Cystitis.**—Chronic cystitis may be due to tuberculosis. Primary vesical tuberculosis is very uncommon. When it does occur it will often be found that it was preceded by gonorrheal infection of the bladder. Most cases of vesical tuberculosis are secondary to renal tuberculosis or to tuberculosis of the prostate, seminal vesicles, or epididymis. Some cases come on rapidly, many tubercle bacilli being found in the urine. Other cases come on more gradually, and in them the urine may contain few tubercle bacilli. In many such cases no tubercle bacilli are found. The tuberculous products caseate and ulcers form or fibrous organization takes place. A cystitis for which no cause can be found, and which is accompanied by pyuria and severe and lasting pain, is possibly tuberculous. Pyuria is usually present, but in some cases the urine is perfectly clear. In some cases the patient has painful paroxysms of varying duration and feels well between the atacks. Finding tuberculosis of the kidney, prostate, vesicle, or epididymis, increases the probability that tuberculous cystitis exists. The diagnosis is made by the cystoscope. Tuberculous ulceration is most common in the trigone and about the inner orifice of the urethra. A tuberculous ulcer is small. The adjacent mucous membrane is not inflamed, but contains grayish-white nodules (Louis E. Schmidt, "Jour. Amer. Med. Assoc.," July 19, 1902).

**Treatment of Chronic Cystitis.**—In treating chronic cystitis remove the cause, if possible (get rid of a stone, evacuate frequently residual urine, dilate a stricture, and remove a tumor). For chronic cystitis certain remedies are taken by the mouth. Water is drunk in large amounts, also iron spring-water (Marienbad, etc.). Salol and boric acid, 5 gr. of each four times a day, are very valuable. Salol in fluidextract of triticum repens does good; so does chlorate of potassium, 10 gr. daily. Either borocitrate of magnesium, quinin, or salicylate of sodium with benzoic acid may often be used with benefit. Alum, tannic acid, uva ursi, copaiba, cubebs, buchu, and turpentine have all been recommended, and possibly may be of some benefit. Urotropin is useful in many cases. This drug prevents the development of bacteria in the urine (Nicolaier) and antagonizes the tendency to sepsis and urinary poisoning. It is given in 5-gr. capsules, from four to six being given daily. Colon bacillus cystitis is treated by giving sodium benzoate and urotropin internally. In obstinate cases a vaccine should be made and given. In cases of chronic cystitis (even the tuberculous form) Stellwagen has had excellent results from the following capsule: 3 min. of creosote, 6 min. of oil of sandalwood, 1 min. of oil of cinnamon, 1 gr. of pepsin. One capsule after each meal. Whatever remedy is used, see that the bowels move once a day and that the skin is active. Champagne and beer must be avoided. If residual urine gathers, a soft

catheter must be regularly employed. If it is possible to introduce a catheter of considerable size, catheterization may be all that is needed in the case. In some cases of chronic cystitis the retention of a catheter from three to five weeks is of the greatest service. If the case is very severe, the bladder must be washed out daily with peroxid of hydrogen (25 to 40 per cent. solution), nitrate of silver (1 : 8000), boric acid (5 to 10 per cent.), carbolic acid (1 : 500), corrosive sublimate (from 1 : 20,000 to 1 : 5000), or permanganate of potassium (1 : 4000). If nitrate of silver or permanganate of potassium is used, first rinse out the bladder with distilled water. If any other agent is used, first wash out the bladder with either boiled water or normal salt solution. The daily injection of a 2 per cent. solution of ichthyol may prove useful. Collin uses a 1 per cent. solution of guaiacol carbonate in sterile olive oil. Some surgeons occasionally employ, at intervals of a number of days, strong silver solutions (30 or 40 gr. to the ounce). If a strong solution is used, after the drug flows away wash out the bladder with a solution of common salt. The bladder is usually washed out by attaching to the free end of a soft catheter, the other end of which is in the bladder, a tube which is connected with a graduated bottle, the force being obtained by elevating the reservoir (*fountain irrigation*). The bladder can be irrigated without using a catheter, the resistance of the compressor muscle of the urethra being overcome by the pressure of a column of water. The reservoir is raised to the height of 6 feet. The patient sits in a chair. The tube of the reservoir has upon it a clamp to control the flow, and in its end a large bulbous tip which will fill the meatus (Valentine's instrument). The tip is inserted into the urethra, the clamp on the tube is loosened, and the patient is directed to take a deep inspiration. In a short time the bladder fills with water, the tube is removed, and the patient empties the viscus naturally. In some cases it is necessary to wait quite a while for the column of water to tire out the muscle. If the fluid will not enter, direct the patient to make efforts as in micturating, the pressure of the fluid on the anterior surface of the cut off muscles being kept up. If this fails, direct him to urinate, and then the surgeon makes another attempt to get the fluid to enter. After a little practice a patient learns how to admit the fluid.

If the cystoscope discloses tuberculosis of the bladder and there is known to be tuberculosis of one kidney, cure of the bladder is impossible without preliminary nephrectomy. In tuberculous cystitis collargol may be injected once a day. A 1 per cent. solution is used and it is allowed to remain for a long time. The method is painless. Collin advises the instillation of 30 min. of the following mixture into the bladder and posterior urethra: 5 gm. of guaiacol, 1 gm. of iodoform, 100 gm. of sterile olive oil. About 30 min. of this are injected once a day. Rovsing, of Copenhagen (Meeting of French Urological Assoc. of 1910), uses, in tuberculosis of the bladder, a fresh solution of carbolic acid (3 to 6 per cent.). He injects from 25 to 50 c.c. through a catheter, allows it to remain in the viscus for two or three minutes, lets it run out, and repeats this procedure until the fluid emerges clear. This treatment is carried out at first every other day. When, on the intervening day, the urine remains clear the interval between treatments is lengthened. Rovsing claims to have cured 28 cases in from four to six weeks. During treatment he makes a cystoscopic examintion every other week to determine the progress of the case. The injections are usually very painful and would be intolerable without a preliminary injection of a local anesthetic (25 c.c. of a 1 per cent. solution of eucain). Sometimes, in tuberculous ulceration of the bladder, curetting through a cystoscope is useful. In other cases the bladder must be opened, the ulcer curetted, and the viscus drained.

If the ordinary methods of treatment fail to cure chronic cystitis; if the bladder resents catheterization and irrigation; if in spite of irrigation the

urine does not become clear; and if there are evidences of infection of the patient and breaking down of his general health, drain by perineal or suprapubic cystotomy and through the incision wash the bladder frequently and thoroughly. If a persisitent cystitis is due to stricture which dilatation fails to cure, perform external perineal urethrotomy and employ perineal drainage.

**Ulcer of the bladder** may be due to injury, cystitis, tuberculosis, malignant tumor, or gonorrhea. A form of ulceration particularly common in anemic women is a solitary, punched-out ulcer (Louis E. Schmidt, "Jour. Amer. Med. Assoc.," July 19, 1902). Ulcers may be single or multiple. Perforation may occur.

A perforation may occur into the peritoneal cavity or into the perivesical cellular tissue. In the former case, after the onset of marked hematuria, there are shock, abdominal pain, and peritonitis. In the latter case there is extravasation of urine or abscess formation.

Tuberculous ulcer is discussed on page 1327 and cystoscopic ulcer on page 1301.

Schmidt (Ibid.) points out that gonorrheal ulceration is apt to be multiple, and causes severe pain and bloody, turbid urine. As a rule, when the bladder is ulcerated, the urine contains blood, blood-clots, or tissue débris, but the urine may be clear when there is a tuberculous ulcer or solitary ulcer.

**Diagnosis** is usually made by the cystoscope. In some cases it is made by exploratory suprapubic incision.

**Treatment.**—If there is one ulcer, or if there are a few ulcers, curet through an operating cystoscope, use irrigations, and keep the urine aseptic. In widespread ulceration perform suprapubic cystotomy, curet the diseased mucous membrane, and insert a drainage-tube. In some cases of malignant growth the cautery is used as a palliative measure. Perforation is treated as is rupture of the bladder (see page 1318).

**Tumors of the Bladder.**—These growths are usually said to be very rare, but in Guyon's statistics they are found to constitute 3.9 per cent. of all cases of genito-urinary disease. They are almost 5 times as common in males as in females. They are most frequently met with between the ages of fifty and sixty, although myxoma is met with only in childhood and sarcoma is most common in the young (Lincoln Davis, in "Annals of Surgery," April, 1906). Persistent vesical irritation may, perhaps, be an element in causing tumor. Tumors of the bladder may be either innocent or malignant, the latter being the commonest. Innocent tumors which may arise from the bladder are papillomata or villous tumors, adenomata, mucous polypi (myxomata), fibrous polypi, myomata, and angiomata. A myoma may attain a great size (even that of a child's head). The common form is intravesical. There is an interstitial form and a peripheral form. Papilloma is far and away the most common form of innocent tumor. Cysts may also arise. Malignant tumors are sarcoma (comparatively rare) and carcinoma (encephaloid, rare; epithelioma, common). Munwes ("Zeit. f. Urology," Nov., 1910) collected 107 cases of sarcoma. Sarcoma begins in the submucosa. The majority of bladder carcinomata are secondary to growths of the rectum, prostate, or uterus. Adenocarcinoma and scirrhous carcinoma are practically always secondary to rectal, prostatic, or uterine tumors. Papillary cancer and epithelioma are not unusually primary (Mandlebaum, in "Surg., Gynecol., and Obstet.," 1907). Primary carcinoma, like primary sarcoma, most commonly arises in or near the trigone. Any tumor of the bladder, innocent or malignant, will eventually cause death if allowed to remain. Papilloma is very apt to become cancerous.

**Symptoms.**—The innocent tumors rarely cause cystitis or irritation, though by obstructing the ureters or the urethra they may induce disease of the kidneys. Hematuria is almost invariable present at some time during the

existence of a bladder tumor. It is apt to be profuse, and the urine contains blood, blood-clots, and perhaps fragments of tumor. The bleeding is intermittent, may occur even when the patient is at rest, and, except in malignant disease, is seldom preceded or accompanied by pain. Bleeding usually occurs at the termination of micturition, the first urine being clear and the last red or clotted. Often hemorrhage is the only phenomenon produced by a papilloma or a mucous polypus. Hemorrhage may occur from a myoma. Malignant tumors cause cystitis, and the urine contains mucus, blood, and pus. The growth may become crusted with salts from the urine. Cancer is decidedly and often horribly painful. In malignant disease the rectum usually becomes involved. Hydronephrosis may be caused. Metastases to the lungs are common. Ulceration may occur into the peritoneal cavity or rectum. A malignant tumor progresses much more rapidly than an innocent growth, although in vesical cancer metastases are not formed so early as in some other regions. Innocent tumors are felt with difficulty with the sound, but malignant tumors are easily felt. In some cases a tumor can be detected by a bimanual examination (a finger in the rectum and the fingers of the other hand on the abdomen). Make a careful study to determine whether or not a growth has infiltrated the prostate, the seminal vesicles, the rectum, or the perivesical tissues. Bleeding follows the use of a sound. There may be difficulty in starting the stream in micturition, there may be interruption or irregular halts in the stream. The latter condition is called *stammering of the bladder*. The urine should be examined microscopically to see if it contains villi, portions of fibroma, colonies of cancer-cells, or fragments of epithelioma. A cystoscope should be employed in order to reach a diagnosis. If the urethra is too narrow for the cystoscope, this channel must be dilated. If there is profuse bleeding an irrigating cystoscope must be employed. In doubtful cases exploratory suprapubic cystotomy is advisable.

**Treatment.**—Some innocent tumors may be cured by fulguration. Papillomata are particularly amenable to this treatment. Recurrence is rare. Some innocent tumors (for instance, peripheral myomata) are treated by suprapubic incision and removal of the growth and a portion of the bladder wall. A papilloma which recurs after fulguration should be treated by excision with a portion of the mucous membrane and submucous tissue of the bladder wall. The perineal operation only enables the surgeon to reach and remove growths of small size, pedunculated growths, and growths near the neck of the bladder. (See Operations on the Bladder.)

Among the operations practised for malignant disease are *partial cystectomy* with resection of one or both ureters, partial resection of the bladder wall, removal of the growth without resection of the wall (a useless procedure), curetting (which is futile). Complete extirpation of the bladder (*total cystectomy*) for cancer has been performed by Bardenheuer, Heresco, and others. Goldenberg reported 26 cases, with a mortality of over 60 per cent. It is usually done in two stages, in the first operation the ureters of a man being transplanted into the rectum, the ureters of a woman into the rectum or vagina. About three weeks later the bladder is removed. The adjacent lymph-nodes along the internal iliac vessels and in front of the sacrum must be removed in all cases. The surgeon should bear in mind that vesical scirrhus and adenocarcinoma are practically always secondary growths, and if he cannot remove the primary growth he should not extirpate the bladder. The complete procedure has been carried out successfully at one operation (Tuffier and Dujarier, "Rev. de Chir.," April, 1898). Some surgeons prefer preliminary double lumbar nephrostomy; others transplant the ureters to the skin surface. The operation of complete extirpation is of questionable value. In most cases it has proved a fatal failure. Munwes ("Zeit. f. Urology," Nov., 1910) re-

ported 69 radical operations for sarcoma. Only 3 patients lived for more than a brief time.

In Rafin's collection of 30 cases of cancer there were 17 deaths. One case was alive five years after operation, 1 fifteen months, and 1 seven months after. Watson's table of 25 cases of cancer shows 14 deaths and only 2 of the survivors were alive and free from recurrence after three years.

Complete extirpation should be employed only when cancer involves the bladder extensively. I agree with Berg ("Annals of Surgery," Sept., 1908), that if less than one-third of the bladder is involved the operation should be partial cystectomy with implantation of the ureters into the portion of bladder remaining. The surgeon removes the anatomically related lymphatic area and always bears in mind that vesical adenocarcinoma and scirrhus are secondary growths. Partial cystectomy saves the performance of the fatal operation of transplanting the ureters as is ordinarily done, or the questionable operation of lumbar nephrostomy with all of its unpleasant consequences. In Rafin's collection of 96 cases there were 21 deaths (a mortality one-half that of complete cystectomy); 50 cases were traced—5 were well over three years and 16 over six months (Berg, Ibid.). Henry Morris lays down the following rule: "When an infiltrating growth is felt, *per rectum* or *per vaginam*, or with the sound, to be involving a large surface of the bladder wall, to be infiltrating its coats, especially in the neighborhood of the ureters and neck of the bladder, no operation whatever should be proposed unless the hemorrhage is copious or the symptoms of cystitis severe, and then an incision for palliative purposes only should be made" (Treves's "System of Surgery").

Many surgeons content themselves in vesical cancer with suprapubic cystotomy, removing the growth and a portion of the bladder wall. If removal is not possible, they curet, cauterize, and drain.

**Operations On the Bladder.—Lateral Lithotomy.**—*Lithotomy* is the removal of a stone from the bladder. *Lateral lithotomy* is an operation which was once a glory of surgery, which is every year becoming less popular, but which is still at times employed by surgeons, especially for stone in children. This operation should not be performed if the stone is over 2 inches in its short diameter; it is rarely justifiable if the stone weighs 3 oz. or more (Cage); and it must not be performed for encysted stone, or on a person with a deep perineum, a narrow pelvic outlet, or an enlarged prostate. For one week before the operation keep the patient in bed, wash out the bladder daily with hot boric acid solution, and administer salol and boric acid by the mouth, 5 gr. of each four times a day. The night before the operation give a saline, order a hot bath, and have the perineum, the scrotum, the buttocks, and the inner sides of the thighs cleansed and dressed antiseptically. In the morning an enema is to be given. At the time of operation the bladder should contain several ounces of boric acid solution. The instruments required are a lithotomy knife, a straight probe-pointed bistoury, a grooved staff, a stone-sound, stone-forceps and scoops, a tenaculum, an aneurysm needle, a fountain-syringe, curved needles and a needle-holder, hemostatic forceps, a tube with chemise (see Fig. 229), a Paquelin cautery, a Clover crutch, and a lithotrite.

Place the patient upon his back, anesthetize him, and find the stone by sounding. If the stone is not discovered by the sound at that time, *do not operate*. Place the buttocks so that they project beyond the edge of the table, introduce the staff into the bladder, flex the legs and thighs, and fasten the patient in the lithotomy position with a crutch. During the first incision the handle of the staff is held toward the belly; after the first cut the staff is set perpendicularly and is hooked up under the pubes. An incision is made, starting just to the left of the raphé of the perineum and $1\frac{1}{4}$ inches in front of the edge of the

anus, and passing downward and outward to between the anus and the ischial tuberosity, but one-third nearer the former than the latter. In the adult this incision is 3 inches in length. The first incision is superficial and does not reach the staff, but it is this incision which may cut the rectum. After making the first cut the nail of the left index-finger feels for the groove of the staff, the staff is hooked up, the knife is entered into the groove and is pushed into the bladder, and as it is withdrawn the wound is enlarged. As the knife enters the bladder there is a gush of fluid. The finger follows the knife and stretches the wound, the staff is withdrawn, and the stone is felt for and extracted with forceps. Liston showed years ago the value of keeping the finger in the wound. This maneuver retains some water in the bladder, and as a consequence causes the stone to rest at the lowest part of the viscus, and when the forceps are introduced they at once come upon the stone. In withdrawing the stone make traction in the axis of the pelvis, and do not rotate the calculus until it is entirely out of the prostatic urethra. Wash or scrape away débris or incrustation from the wall of the bladder, see that no other stone is present, syringe out the viscus with warm salt solution, insert a tube, apply antiseptic dressings around the tube, and put on a T-bandage. The end of the tube which is external to the dressings is fastened to the tails of the T-bandage. A rubber cloth is put on the bed, under the body and legs, and the patient's buttocks rest upon a mass of old linen, the scrotum being raised on a pad. The knees are bent over pillows. Change the linen as soon as it becomes wet. Remove the tube in forty-eight hours. The urine begins to come by the urethra from the eighth to the twelfth day. In children the incision is not so long, it is dilated by forceps instead of by the finger, and no tube is required. In lateral lithotomy the prostatic and membranous portions of the urethra are opened, the prostate gland is partly divided by the knife, and the wound is dilated by the finger. One objection to the operation is that it is possible to cut the rectum, and another is that inflammation may occlude the ejaculatory ducts and cause sterility.

**Suprapubic Lithotomy.**—This operation is the removal of a stone through an opening above the pubes. It is in many instances the preferable operation. The mortality of this operation is higher in children than that of lateral lithotomy; in adults and in individuals beyond middle life the mortality is decidedly less than is that following the lateral operation. It is used for the removal of multiple calculi, for very hard stones, for stones above 2 inches in their short diameter, for calculi in men with enlargement of the prostate, for foreign bodies incrusted with sediment, when the perineum is deep, when the pelvic outlet is narrow, for encysted stones, for calculi associated with a vesical tumor, and when the urethra will not permit the use of a lithotrite. Before doing the operation determine the carrying capacity of the bladder when the patient is not anesthetized. This gives the safe limit of distention. Under an anesthetic the bladder will receive 3 or 4 more ounces than the safe amount. The patient is prepared as for lateral lithotomy, except that the pubes are shaved, and the lower part of the abdomen and the upper part of the thighs are disinfected. During the operation the penis is kept wrapped in a piece of antiseptic gauze.

In performing the operation place the patient in the Trendelenburg position. It is necessary to distend the bladder and raise it in order to have the prevesical space uncovered by peritoneum. In most cases this is accomplished by distention of the bladder and the Trendelenburg position. In a few cases in which the bladder holds very little fluid a rectal bag is used to lift the bladder. If a rectal bag is to be used an assistant oils it and pushes it (empty) above the sphincters. It is filled after the bladder has been injected. Draw off the urine with a soft catheter, wash out the bladder with a solution of

silver nitrate (1 : 8000 to 1 : 5000), and inject the bladder with the same solution. In a child under the age of five inject 3 to 4 oz.; in an adult it is usual to inject 10 to 12 oz. Withdraw the catheter and tie a tube around the penis to prevent the escape of fluid. After injecting the bladder with fluid, if the viscus is not well lifted, inject the rectal bag with water and clamp its tube with forceps. In a child inject from 2 to 4 oz. of warm water into the rectal bag; in an adult inject 10 oz. Bristow suggested the injection of air into the bladder. Some surgeons simply inject air by means of a catheter and a brass syringe or a Davidson syringe. Air injection is not recommended if fluid can be used. In operating on an air-distended bladder there is greater danger of trauma, shock, and postoperative bleeding. If air is injected, a rectal bag is never used, and the patient is placed on his back rather than in the position of Trendelenburg. The best method of injecting air is that of F. Tilden Brown,[1] by means of a bicycle pump. A catheter is introduced, the bladder is washed out, the catheter is fastened to a bandage, the bicycle pump is attached, the operation is proceeded with, and when the transversalis fascia is exposed the bladder is filled with air, the soft catheter is clamped, and the bladder is opened. Make a 3-inch longitudinal incision in the median line of the hypogastric region, terminating over the symphysis. When the prevesical connective tissue is reached push it up with a gauze-covered finger. In close association with this tissue are the prevesical fat and peritoneum. The pushing up must be done slowly and gently with a sweeping motion. Any roughness may be responsible for disaster. If the peritoneum should appear, push it up. Hold the wound edges apart by retractors. The large veins are seen, giving the bladder a blue color. Avoid these veins if possible, but even if they should be cut bleeding will usually cease when the bladder has been opened and the rectal bag has been emptied and removed.

Fig. 886.—Cathcart drainage. The Y-tube is of glass and is darkened in order to be shown against a white background.

Clamp bleeding vessels. Pass a stay suture of strong silk on each side of the contemplated incision in the bladder. Catch each suture by a hemostat and let it hang. Open the viscus in the middle line above, and cut toward the pubes. Explore the bladder, remove the stone or stones, scrape away incrustations, ligate bleeding vessels outside the bladder, and irrigate the viscus with hot saline solution. Introduce a double tube into the bladder, and attach to its external end a long tube to siphon off the urine. The bladder can be drained very satisfactorily by a siphonage apparatus (Fig. 886). Suture the muscles and fascia at the upper part of the wound. Dress with dry antiseptic gauze and a rubber-dam, the dressings and binder being split to go around the tube. Catch the urine which siphons over in a bottle containing some antiseptic fluid. Change the dressings as often as they become wet.

[1] "Annals of Surgery," Feb., 1897.

Take out the tube in four or five days, and allow the wound to heal by granulation. The patient may get up in two weeks. Many Continental surgeons advocate immediate suture of the bladder after incision. Albert, Vincent, Bassini, DeVlaccos, and others advocate immediate suture. The suture material should be catgut. After suturing a catheter is kept in the bladder to drain the viscus. Immediate suture may be employed in patients of any age, but should not be used if the urine is very septic or if pyelonephritis exists. In some cases the attempted closure will fail; in others it will only partially succeed; in others it will prove successful; but even if it only partially succeeds it will tend to prevent dissemination of urine in the prevesical cellular tissue. The chief causes of death after suprapubic lithotomy are septicemia, secondary hemorrhage, cellulitis, peritonitis, and suppression of urine. J. W. White estimates the relative mortality of suprapubic and lateral lithotomy as follows: In children the suprapubic operation gives a mortality of 12 per cent.; the perineal, of 3 per cent. In adults the suprapubic gives a mortality of 12 per cent.; the perineal, from 8 to 12 per cent. In old men the suprapubic gives a mortality of 25 to 30 per cent.; the perineal, 30 to 40 per cent.

**Crushing of Vesical Calculi.**—This is now done in one sitting, the old operation of Civiale, which required repeated crushings, being obsolete. In every case of suspected stone (as before stated) the cystoscope should be used.

*Contra-indications to Litholapaxy.*—Papilloma, malignant tumor, projecting prostatic lobes, encysted stone, and diverticuli.

**Litholapaxy** (*Bigelow's operation*, or *rapid lithotrity*) is the operation for removing a stone from the bladder in one sitting by thoroughly crushing the stone and completely washing away the fragments. This operation is wonderfully successful if done by an expert. Few of us do it sufficiently often to learn how to perform it with great rapidity, certainty, and safety. It is the best operation in most cases if performed by a very skilful man. It is the operation in the majority of cases for even the general surgeon to select, but the general surgeon will have better results in certain difficult cases after suprapubic lithotomy than after litholapaxy. Sir H. Thompson says this method is suited to 29 cases out of 30. Litholapaxy should be employed if the bladder will hold at least 4 oz. of fluid and is in a fairly healthy condition; if the urethra is tolerant and penetrable by instruments; if the stone is not too hard, does not weigh over $2\frac{1}{4}$ oz., and is not over 2 inches in diameter. It is not suitable for multiple calculi, for large and hard calculi, for encysted stones, or for a patient with marked enlargement of the prostate gland, with vesical atony, or with cystitis. An easily dilatable stricture need not prevent the surgeon doing litholapaxy. The stricture can first be dilated, and later Bigelow's operation can be performed, but firm, gristly strictures demand a cutting operation. If the urethra is intolerant to instrumentation, the patient being prone to febrile attacks when it is attempted, cut instead of crushing. An individual laboring under kidney disease will do better after this operation than after cutting (Cage). In diabetes, locomotor ataxia, and conditions of exhaustion patients are best treated by Bigelow's operation, unless cystitis exists.

The Indian surgeons have had the most admirable results from litholapaxy. It has often been claimed that such results were due to racial peculiarities of the patients and various factors regarding their habits, diet, etc. The fact, however, that some of these very surgeons have returned to England and repeated their triumphs in London shows how large a part masterly dexterity played in obtaining success.

J. A. Cunningham ("Brit. Med. Jour.," Aug. 7, 1887) reports upon 10,073 Indian cases of litholapaxy. The mortality was 3.96 per cent.

Cabot, of Boston, in 116 cases had but 4 deaths, and 2 of these were due to pneumonia.

The preparation of the bladder is the same as for lithotomy. Be sure to measure the stone, and to ascertain also whether a lithotrite can readily be introduced and manipulated. The instruments required are a stone-sound (see Fig. 885), lithotrites (several sizes, Figs. 887–889), an evacuating bulb and tubes (straight and curved, Figs. 890, 891), soft catheters, a glass irrigator to inject the bladder, and instruments in case the surgeon is forced to cut. The patient is anesthetized and is placed upon his back, a pillow is inserted under the pelvis, and he is well wrapped up. The urine is drawn and a measured amount of warm boric acid is allowed to flow into the bladder. This plan is better than having the patient retain his urine, as in the latter case there is no certainty as to the amount of fluid in the viscus. It is well to introduce at least 5 or 6 oz. of fluid, if possible. If the bladder will not hold 4 oz. the operation is unsafe (Thompson). The lithotrite, preferably the instrument of Forbes (Fig. 889), is now introduced,

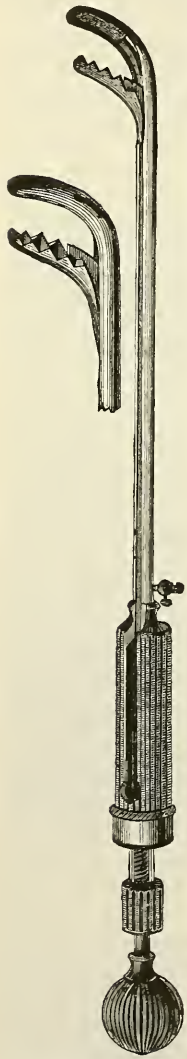


Fig. 887.—Bigelow's lithotrite.

Fig. 888.—Thompson's lithotrite.

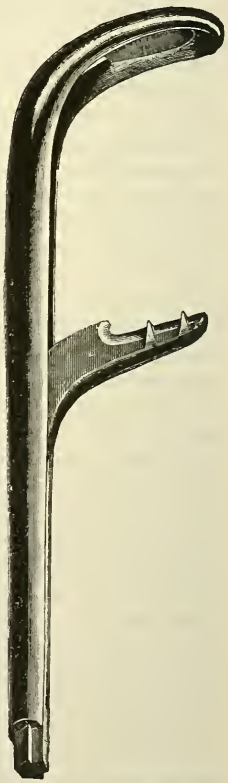


Fig. 889.—Forbes's lithotrite.

the handle being gradually raised to a vertical position as the penis is drawn up on the shaft, but not being depressed until the instrument has passed by its own weight into the prostatic urethra. Thompson's plan for catching the stone is as follows: After introducing the lithotrite, let its lower end rest for a few seconds on the bottom of the bladder, so that currents will subside; then draw back the male blade, wait a moment, close the blades, and in almost every

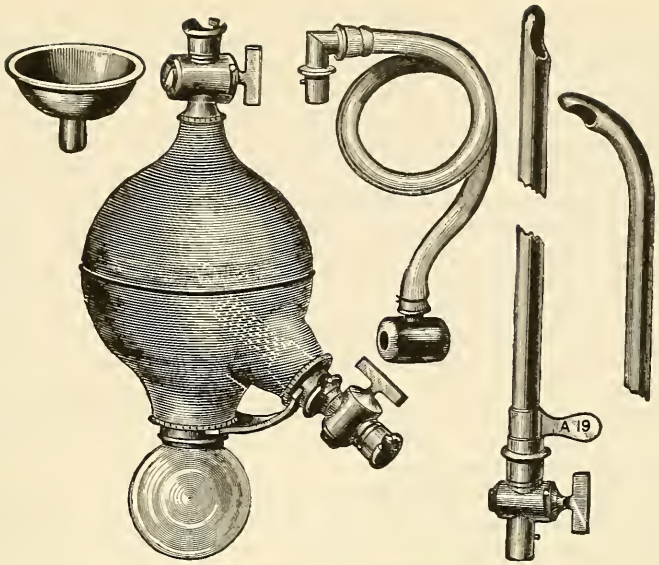



Fig. 890.—Bigelow's latest evacuator.

instance the stone will be caught. If the stone is caught, press firmly to see that the calculus is well held, lock the instrument, and break the foreign body by screwing. When resistance suddenly ceases the stone has either slipped or has been crushed; if crushed, the blades should have been felt forcing through the stone and the calculus should have been heard to break. When resistance ceases catch and crush again as above directed. Rapid movements with the lithotrite are improper, as they establish currents which are apt to push away the stone. If the above maneuver does not catch the stone, see if the calculus be near the neck of the bladder. Pull the instrument close to the vesical neck, and open it, not by pulling the male blade, but by pushing the female blade. If the operator still fails to catch the stone, or if, after crushing, a large fragment knocks against the evacuator, which fragment cannot pass, conduct a careful search: turn the blades to the right side, open, and close; then to the left side, open, and close; next turn the point around behind the prostate, open, and close. After catching a stone with the lithotrite, turn the instrument very slowly, so as to detect the catching of the bladder wall if it has occurred, and crush the stone in the middle of the bladder with the blades up. After crushing several times, proceed to evacuate. Fill the aspirator with warm saline fluid. Insert an evacuating catheter, its point being in the center of the bladder, let the fluid and fragments run out, and attach the aspirator to the

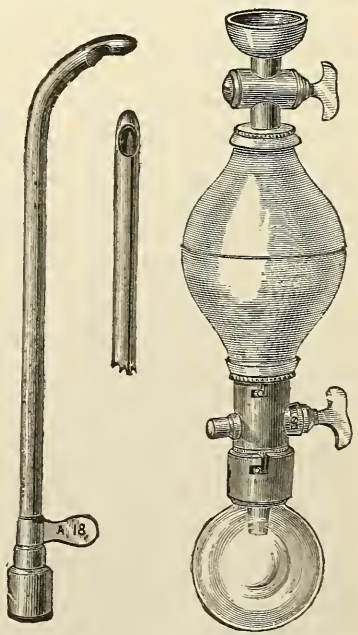



Fig. 891.—Thompson's evacuator.

catheter; turn the valve, and compress and relax the bulb so that an ounce or more of fluid is forced in at each squeeze, the compression coinciding with expiration. The débris falls into a bulb, and the pumping is continued until the fragments cease to pass, whereupon the point of the catheter is pushed against the floor of the bladder and another trial is made. If fragments which cannot gain exit are felt knocking against the tube, withdraw the evacuator, crush again, and again use the aspirator. When no more débris comes away and no more fragments are felt, withdraw the tube and carefully sound the bladder. Keyes advises the operator to seek for a final fragment by listening with a stethoscope while pumping at the bulb and searching the bladder with the tube. The amount of blood usually obscures a cystoscopic view. This operation will rarely occupy over forty minutes, though Bigelow has protracted it for three hours, the patient recovering. A serious complication is severe bleeding, due to damage done with the instrument or to the presence of a tumor which easily bleeds. The injection of moderately hot water or of adrenalin solution (1 : 10,000) usually checks hemorrhage, but if bleeding is dangerous in amount the operation of litholapaxy should be abandoned and suprapubic lithotomy be performed.

If clogging of the lithotrite with fragments occurs, forcible pushing of the blades together repeatedly will probably amend it; but it will never happen if the surgeon uses a proper form of instrument. A lithotrite with a fenestrated blade will not lock. Forbes's lithotrite is a very powerful instrument, the blades of which will not lock. If the blades of a lithotrite should become forcibly and hopelessly locked, make a perineal section, clear out the blades, close them, and then withdraw the instrument.

*After-treatment.*—Put the patient to bed, apply a bag of hot water to the hypogastrium, and give him a hypodermatic injection of morphin as he recovers from ether. Give a hot hip-bath every night, and administer liquor potassii citratis in moderate doses every day. If urethral fever occurs, use quinin and morphin, wash out the bladder several times daily with warm boric acid solution, and tie in a rubber catheter. If retention occurs, use the catheter. If cystitis appears, treat as in an ordinary case. The urine ceases to be bloody in two or three days, and the patient may get up in a week. Before the case is discharged cystoscopy should be practised to be certain that no fragments of stone remain.

**Litholapaxy in Male Children.**—It used to be the teaching that a child, because of the small size of the bladder, the small diameter of the urethra, and the readiness with which the mucous membrane is lacerated by even slight violence, is a poor subject for crushing. Lateral lithotomy is known to be eminently successful when performed upon children. The elder Gross did this operation upon 72 children, with only 2 deaths. Keegan, however, has persuaded the profession that rapid lithotrity is perfectly applicable to children: He shows that the bladder of a child of even less than two years of age is quite large enough to allow the surgeon to manipulate an instrument; that the mucous membrane is in no danger if the operator is careful, and that the urethra is by no means so small as was supposed. The urinary meatus must often be incised, and after doing this, Keegan states, there can be passed in a boy of from three to six years a No. 7 or 8 lithotrite (English), and in a boy of from eight to ten years a No. 10 or even a No. 14. It is, however, just to state that the operation is more delicate than a like procedure on older persons, and that no one is justified in doing it who has not had considerable experience in adult cases. Furthermore, it should be noted that Keegan's mortality by this operation has been 4.3 per cent., while Gross's mortality from lateral lithotomy on children was under 3 per cent.

*Special points relating to litholapaxy on male children* are as follows: use well-

fenestrated lithotrites; have a stylet to punch out the fragments blocking the evacuator; and crush the stone to a fine mass. There can usually be employed a No. 8 lithotrite and a No. 8 evacuating tube (English scale).

**Perineal Lithotrity** (*Keith's Operation*).—This operation is employed by some surgeons in dealing with very hard or very large calculi in male adults, or in cases in which it is impossible to introduce a lithotrite into the bladder. Keith's operation consists in opening the urethra from the perineum, passing a lithotrite through the wound, into the urethra and along the urethra into the bladder, and crushing the stone, introducing an evacuator and removing the fragments. In Keith's operation the incision is median and opens the membranous urethra. In very large stones Milton thinks the surgeon should open the bladder as in ordinary lateral lithotomy, introduce a lithotrite through the incision, and crush the stone before extracting it, thus avoiding the infliction of injury upon important structures.

**Operation for Stone in Women.**—If the stone be small, give the patient ether, place her in the lithotomy position, dilate the urethra by the uterine dilator until it admits the index-finger, and remove the stone by the finger, the scoop, or the forceps. If the stone is found to be too large to pass, crush it by a lithotrite and get rid of the débris by the evacuator. Large stones (2 oz.) may require suprapubic lithotomy. Vaginal lithotomy is never required. If done, it is very likely to leave as a legacy a vesicovaginal fistula. In female children dilate the urethra, crush the stone, and evacuate.

**Cystotomy or Cystostomy.**—These terms mean the opening of the bladder. If the opening is made for diagnosis or treatment and is then closed or allowed to close, the operation is a cystotomy. If the wound is deliberately kept open it is a cystostomy. The bladder may be opened for drainage, for diagnosis, for the removal of stones or tumors, or for the treatment of ulcers. This opening may be done by (1) a suprapubic cut (as in suprapubic lithotomy), (2) a lateral perineal cut (as in lateral lithotomy), or (3) a median perineal cut (as in median lithotomy).

The operation may be completed in one sitting, or the bladder may be only exposed, the opening of it being delayed for several days until it becomes adherent to the margins of the wound (Senn's operation). Senn's operation prevents infiltration of urine into the prevesical space, and it is advisable to select it if the urine is very foul.

A sinus may persist after suprapubic cystotomy, but usually the wound heals unless it is kept open by some expedient.

The effects of suprapubic drainage are very beneficial in cases of chronic cystitis associated with hypertrophy of the prostate gland, the urine being foul. Drainage causes the urine to become clear and the mucous membrane of the bladder to become normal. If the opening is made as a permanent drain, there will usually be incontinence, as the new channel has no sphincter action (Dandridge). Figures 895 and 896 show tubes for prolonged drainage.

**Suprapubic Cystotomy (or Cystostomy).**—The operation is employed to allow the surgeon to explore the bladder, to treat an ulcer, to provide drainage, or to remove a tumor. If the operation is for calculi, it is known as suprapubic lithotomy (see page 1331). After the bladder is opened its interior can be illuminated by the rays of an electric lamp, which appliance is fastened with a mirror to the forehead of the operator. If an ulcer is found, it is scraped with a curet or a spoon. Most cases of tumor require suprapubic cystotomy. It is true that a small single growth at the vesical neck is accessible by median cystotomy, but the area for manipulation is very narrow and the growth cannot be seen. Every large growth, all cases of multiple tumors, and all cases of tumor in individuals with great depth of perineum or with enlarged prostate require suprapubic cystotomy, an operation which allows one to feel and to

see the growth, which gives room for manipulation, and which permits thorough exploration of the entire bladder. The patient is put in the Trendelenburg position if water distention is used, but is placed horizontally if air distention is employed. After opening the bladder as for stone (see page 1332), hold the edges of the incision apart by means of a speculum (speculum of Keen or Watson) or by retractors, and reflect the electric light into the wound. Growths when seen can be twisted off, a pair of forceps holding the base and another pair being used to twist, but after removal by twisting they will always recur unless the base and the mucous membrane about the base is removed. Broad malignant growths may require partial cystectomy. Some growths (as inoperable cancer) are removed piece by piece with Thompson's forceps (Figs. 892–894), the base of the tumor being scraped. Such a procedure is merely palliative. Soft growths are scraped away by a curet, a spoon, or the finger-nail. If bleeding is severe, check it by pressure, by hot water, by a 1 : 10,000

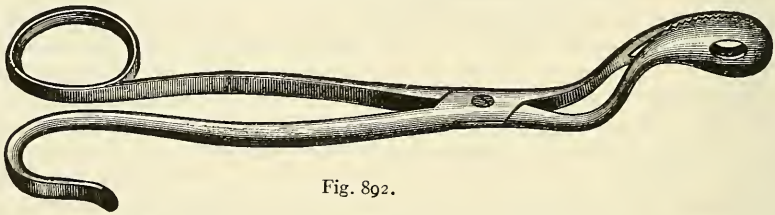


Fig. 892.

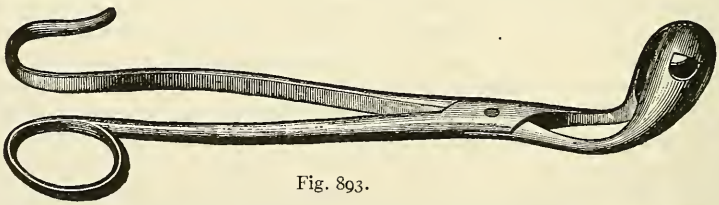


Fig. 893.

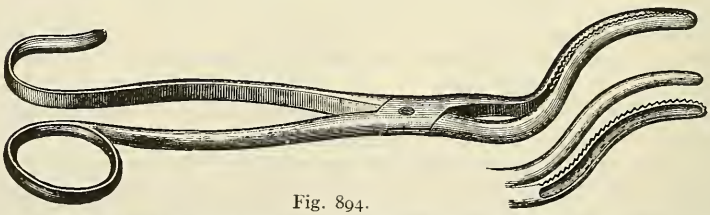


Fig. 894.

Figs. 892–894.—Thompson's vesical forceps for removing growths in the bladder; for growths close to the neck of the bladder, with separation of the blades, to avoid nipping the neck of the bladder.

solution of adrenalin chlorid, or even by the actual cautery. In some cases the wound is allowed to heal rapidly. In others the bladder is drained for a considerable time. In some it is kept open permanently. Permanent drainage is desirable in some cases of enlarged prostate, and in such cases Senn's tube (Figs. 895 and 897) or Stevenson's tube (Figs. 896 and 898) may be employed.

**Median Cystotomy (or Cystostomy).**—The same incision is made in the perineal raphé for median cystotomy as for median lithotomy. A grooved staff is introduced and is hooked up under the pubes; an incision is made into the membranous urethra, and is extended backward for $\frac{3}{4}$ inch, and a finger is carried into the bladder. If searching for a growth, find it by the finger. The usual rule has been to catch it in Thompson's forceps and twist it off. Such an operation is totally inefficient. Soft growths may be scraped away. Stop bleeding by digital pressure or by injections of hot water or adrenalin

chlorid (1 : 10,000). Median cystotomy does not allow anything like the freedom of access given by suprapubic cystotomy, and the latter operation is the best for tumor cases. The median operation may be used for drainage.

Fig. 895.—Senn's silver tube.

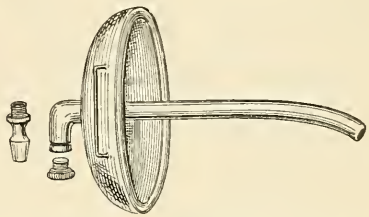


Fig. 896.—Stevenson's suprapubic drainage-tube.

**Growths In the Female Bladder.**—It was long the custom to dilate the urethra as in a case of stone, and scrape, twist, or pull the growth away or ligate it. This plan is inefficient, as by it the base of the tumor is not removed. It is usually best, if fulguration fails or will evidently be useless, to perform a suprapubic operation. If the growth is large or if there are multiple growths, always perform suprapubic cystotomy.

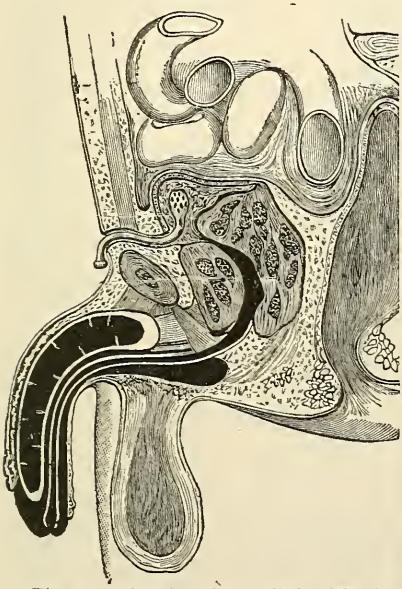


Fig. 897.—Senn's tube applied. The instrument does not press upon the sensitive neck of the bladder.

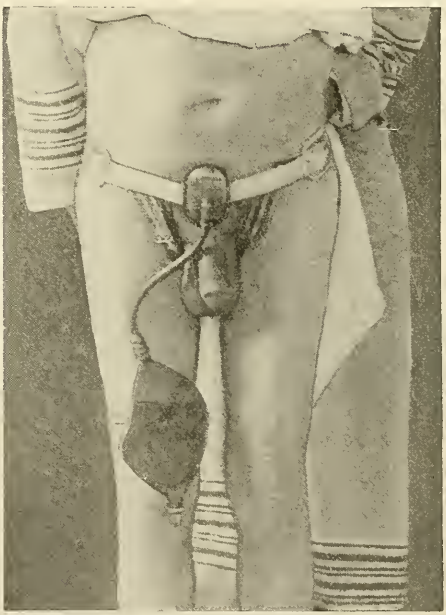


Fig. 898.—Stevenson's suprapubic drainage-tube in place and attached to a receptacle for urine.

## Diseases and Injuries of the Urethra, Penis, Testicle, Prostate, Seminal Vesicle, Spermatic Cord, and Tunica Vaginalis

**Injuries of the penis and urethra** may arise from traumatism to the perineum or the penis, from cuts and twists of the penis, from the popular "breaking" of a chordee, from tying string around the organ, from forcing rings

over it, from the passage of instruments, or from the impaction of calculi. Violence inflicted upon an erect penis may fracture the corpora cavernosa. The writer saw one man with a glass rod broken off in the canal, he having been in the habit of introducing it at the dictate of morbid sexual excitement. A patient in the Insane Department of the Philadelphia Hospital pushed a ring over his penis, which organ ulcerated into the urethra. These injuries are treated on general principles.

**Perineal Bruises.**—If the perineum is bruised without rupture of the urethra, the perineum and scrotum swell and become discolored; water is passed with difficulty because the extravasated mass of blood in the periurethral tissues compresses the canal more or less; the water is not bloody; and there is severe pain and much shock. Some authors include under rupture those cases in which laceration of the spongy tissue occurs, without involvement of the mucous membrane or of the fibrous coat, but they are properly contusions.

**Treatment of Bruises and Wounds.**—Place the patient in bed and establish reaction, and when reaction is complete employ opiates for the relief of pain. Apply an ice-bag to the perineum. If, notwithstanding these measures, swelling continues, introduce a silver catheter (No. 12 English), tie it in, and make pressure upon the perineum by a firmly applied T-bandage or by a crutch braced against the foot-board of the bed. Even when swelling is slight, retention of urine may occur from projection of a submucous blood-clot into the canal of the urethra. In some cases it may become necessary to incise the perineum and evacuate the blood-clot. After twenty-four hours have passed, if hemorrhage has ceased, substitute a hot-water bag for the ice-bag, and empty the bladder regularly by a soft catheter. Occasionally, though rarely, an abscess forms. *Punctured wounds of the urethra* require ordinary dressings. *Incised wounds of the urethra*, when longitudinal, are closed by suture. Healing is rapid and ill consequences are not to be feared. Stricture does not follow. When the wound is transverse, introduce a catheter, suture the wound over the instrument, and remove the catheter at the end of the third day. If a catheter cannot be introduced, employ sutures, but at the first evidence of extravasation open the wound, and if drainage is not free perform external perineal urethrotomy.

**Rupture of the Urethra.**—By this term is meant a lacerated or a contused wound of the urethra, destroying partially or entirely the integrity of the canal. A lacerated wound may be induced by fracture of the cavernous bodies during erection, the symptoms being severe hemorrhage, intense pain, retention of urine, and inability to pass an instrument; infiltration of urine occurs, and gangrene is a common result. The writer has seen 1 case of rupture of the penile urethra due to a man's slipping while shaving, the penis being caught in a partially open drawer, the drawer being shut by his body coming against it. Rupture, however, is almost invariably located in the perineum, and it arises when the urethra is suddenly and forcibly pressed against the arch of the pubes by a blow, by a kick, or by falling astride a beam or a fence-rail or on a wagon wheel. Retention of urine due to stricture may lead to extravasation of urine. The lesion of urethral rupture consists in some cases of laceration of the spongy tissue and the mucous membrane, a cavity being formed which communicates with the canal, and which fills with urine during micturition. In other cases not only the spongy tissue and the urethral mucous membrane are rent asunder, but the fibrous coat is also torn, the canal opening directly into the perineal tissues, among which a huge cavity forms, that fills with blood and later with urine and pus. The urethra may be torn entirely across, but in most cases a small portion at least of its circumference is uninjured. Rupture never occurs primarily and alone in the prostatic urethra. Some think it is extremely rare in the membranous urethra unless due to pelvic fracture. I believe that it occurs not unusually in the membran-

ous urethra. When we recall that this is the fixed portion of the tube we would expect rupture here rather than elsewhere. It is very unusual in the penile urethra. The seat of rupture in the great majority of cases is in the region of the bulb. Very rarely is the skin broken.

The **symptoms** of rupture of the fixed urethra are considerable pain, aggravated by motion, pressure, and attempts to pass water; decided shock; in some cases micturition is still possible, blood preceding and also discoloring the stream, for some blood usually runs into the bladder; retention of urine quickly arises; in a vast majority of the cases retention is absolute from the very first, and it is due to the interruption in the integrity of the canal and to the occlusion of the channel by blood-clots. Bleeding, which is usually free, lasts for several hours, some little blood generally appearing externally and much being retained in the perineum, inducing progressive swelling. The presence of a large swelling is regarded as evidence of urethral rupture. The blood which is effused in the perineum may extend under the fascia to the penis and scrotum. The swelling soon becomes reddish, purple, or even black, pressure upon it is apt to cause blood to run from the meatus, and it is augmented in volume when attempts are made to urinate. After a time, if the surgeon does not act, the urine fills the perineal cavity and widely infiltrates, and there ensue gangrene, sloughing, and sepsis, life being endangered or fistulæ being left as legacies. The course of the extravasated urine will often enable one to locate the seat of injury. In rupture of the membranous urethra, if uncomplicated, the urine remains between the two layers of the triangular ligament until a channel is opened for it by sloughing or by the knife. When extravasation occurs behind the posterior layer of the ligament the urine finds its way to the perineum in the neighborhood of the anus. When the rupture is in front of the anterior layer of the ligament the urine, directed by the deep layer of the superficial fascia, finds it way into the scrotum and up on the belly, but does not pass into the thighs (Fig. 899). A contusion is distinguished from a rupture by the facts that in the former the perineal swelling is not very extensive and does not enlarge on attempting micturition, while in the latter it is extensive and does enlarge on attempting to pass water. Furthermore, contusion does not cause urethral hemorrhage, while rupture does. A contusion sometimes, but not often, prevents the passage of a catheter; a rupture almost always, but not invariably, does so. The mortality from severe rupture with extravasation is about 14 per cent. (Kaufman).

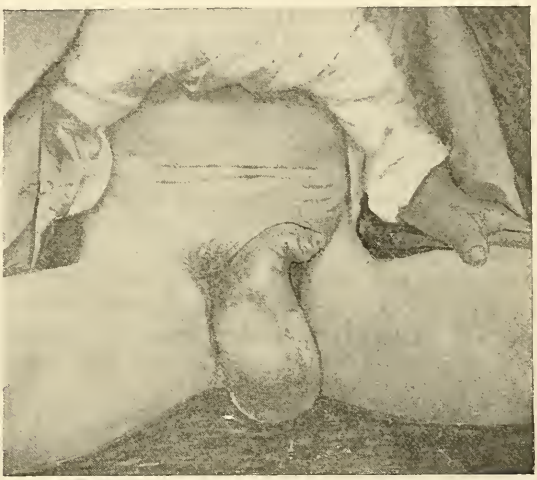


Fig. 899.—Rupture of the urethra and extravasation of urine.

**Treatment.**—In some very rare cases it is possible to suture the urethra, and this procedure should be carried out when possible. In order to suture, perform suprapubic cystotomy and also make a perineal section. Find the posterior end of the ruptured urethra in the perineum by passing a catheter from the bladder into the urethra. Suture by way of the perineum and with silk.

The sutures pass through all of the coats of the urethra. The roof of the canal is sutured first, then a steel sound is introduced from the meatus, and the urethra is sutured around the instrument. The sound is withdrawn and the bladder is drained by Cathcart's siphon as illustrated in Fig. 886 or by a double tube. In recent cases of ruptured urethra the usual treatment is as follows: Immediately perform median perineal section and turn out the clot; trim off lacerated edges; find the proximal end of the urethra, pass a catheter from the meatus into the bladder, and leave it *in situ* until healing has begun around it. If the catheter cannot be passed from the meatus, open the bladder above the pubes and find the proximal end of the torn urethra by retrograde catheterization. In retrograde catheterization we push an instrument from the bladder into the wound and use it to guide a catheter from the meatus into the bladder. When rupture occurs back of a stricture it is a good plan to excise the cicatricial tissue. In cases with extravasation make a median incision and numerous transverse cuts to secure drainage for areas of retained urine or pus. Then at once perform suprapubic cystotomy. Drain suprapubically and from the perineum for about two weeks, by which time sloughing tissue will have separated. Then find the posterior urethra by retrograde catheterization and do a perineal operation to repair the damaged urethra. (See Eugene Fuller, in "New York Med. Jour.," Nov. 23, 1901.) The wound is packed with iodoform gauze, and the bowels are tied up with opium for a few days. Some surgeons strongly disapprove of the custom of retaining the catheter, believing that the instrument does no real good, as urine is certain to get between the catheter and the walls of the urethra. They think it is quite enough to stuff the wound with gauze, the patient urinating through the wound for the first few days, after which time a catheter is used at regular intervals. Whatever method is employed, healing will require from six to eight weeks, and the patient must during the rest of his life, from time to time, introduce large-sized bougies.

**Foreign Bodies in the Urethra.**—These bodies may be calculi, bodies introduced by injury, as shot, bone, etc., bodies entering from a fistulous opening into the rectum, or bodies introduced from the meatus, as broken bits of catheters, straws, pins, etc.

The **symptoms** and **treatment** vary with the size and the nature of the body. Sometimes there are almost no symptoms; at other times there are found great pain, retention of urine, and hemorrhage. Examination is made by the urethroscope by feeling carefully with a finger in the rectum and by searching very gently with a sound, taking care not to push the body back. In some cases the body can be removed by aid of the urethroscope. Employ this plan if possible. If it is not possible, try the following plans: If the bladder is well filled with water when the body becomes impacted, inject a little oil into the meatus, close the lips with the fingers, and direct the patient to forcibly attempt urination, the surgeon opening the meatus when the urethra is widely distended, the foreign body being often forced out. If this maneuver fails, and the foreign body is impacted in the pendulous urethra, prevent its backward passage by at once tying a rubber tube around the penis. Try to squeeze the body out, and, if unsuccessful, endeavor to catch it with a wire loop, with a scoop, or with the long urethral forceps. If these methods fail, cut down upon the body and remove it, dividing any existing stricture. If it is lodged just back of the meatus, incision of the meatus will permit extraction. If a hairpin is in the canal, the points of the pin are almost always pointing to the meatus; to prevent them catching on attempted withdrawal, the penis must be squeezed to approximate the feet of the pin, and when they are adjacent a part of a silver catheter is slipped over to retain them in this position, when the pin can be extracted. If this fails, drag the penis against the belly, by rectal touch force the sharp ends of the pin out through the integument, cut one

end off, and then withdraw the other. An ordinary large-headed pin is forced out in the same way, and when the head is turned externally it is extracted by way of the meatus. If a hard or sharp foreign body is lodged in the prostatic urethra, do not catch it with an instrument and try to drag it forward. To do so will be apt to tear the membranous urethra. It is better to push it into the bladder and remove it later by cutting or, if it be a stone, by crushing (H. Hartmann, in "La Presse Méd.," July 24, 1901). If a lithotrite loaded with fragments be caught in the urethra, the surgeon must perform a perineal section, to enable him to clean and close the blades. After the blades have been closed the instrument may be easily withdrawn.

**Urethrorrhea** is not urethral inflammation, but is a condition of sensitiveness of the urethra and oversecretion of the glandular elements. It may be due to masturbation, sexual excess, and also, as Sturgis points out, to withdrawal during sexual intercourse, and to ungratified sexual passion. A drop or two of transparent mucus is found at the meatus in the morning, and a considerable amount may flow away while straining at stool or upon the diminution of an erection. This flow at stool is often called defecation spermatorrhea. This discharge stains, but does not stiffen linen (Sturgis). The discharge contains mucus, mucous corpuscles, epithelial cells, but no gonococci or pus organisms. The patient may be well in all other respects, but in many cases there are neurasthenic symptoms, sexual weakness, or even impotence. It is well to explain to the patient that the overaction of the muciparous glands is Nature's effort to facilitate the passage of the fluid of the orgasm and to alkalinize an acid urethra which would inhibit the activity of spermatozoids. Quacks fatten on these unfortunates. This condition may be readily distinguished from prostatorrhea by the absence of Böttcher's crystals (see page 1377) and from that very rare condition spermatorrhea by the absence of spermatozoids.

**Treatment.**—In an uncomplicated case improvement or cure will follow upon the abandonment of evil habits and the systematic passage of steel sounds. If complications arise, they must be treated.

**Urethritis, or Inflammation of the Urethra.**—Urethral inflammations can be divided into two classes: (1) *simple* or *non-specific*, in which infection is due alone to pyogenic cocci (particularly the Bacillus coli communis and the Staphylococcus pyogenes), and (2) *specific*, in which the gonococcus is present.

**Non-venereal, non-specific, pyogenic,** or **simple urethritis** may be due to several causes, such as traumatism; great acidity of the urine; chancre in the urethra; contact with menstrual fluid, leukorrheal discharge, the discharge from malignant disease of the uterus, ordinary pus, or acrid vaginal discharge; the passage of instruments; the administration of irritant diuretics; strong injections; worms in the rectum; a febrile malady; venereal excess and masturbation; the passage or impaction of foreign bodies, and papillomata of the urethra. A temporary and mild urethritis sometimes accompanies early syphilitic eruptions. Simple urethritis is less severe and prolonged than gonorrheal urethritis, though clinically in the early stage the physician cannot invariably distinguish between the two forms. The diplococci of gonorrhea are never found in the discharge of simple urethritis, although there may be numerous other diplococci. In medicolegal cases testimony is not admitted as to the presence or absence of diplococci, as judges do not admit that their presence proves or their absence disproves gonorrhea. In the non-specific inflammation pus is not always present, many cases stopping short of pus formation after a varying period of catarrh, but any catarrh may become purulent. A simple urethritis may be caused or may be prolonged for an indefinite period by the presence of large amounts of oxalate in the urine or the existence of the uric-acid diathesis (see Gouty Urethritis).

*Treatment.*—Seek for the cause and remove it. Correct any abnormal condition of the urine by means of suitable diet, drugs, and mode of life. Mild astringent injections are useful. It may be necessary to flush the urethra repeatedly with a solution of silver nitrate (1 : 8000).

**Traumatic Urethritis.**—The onset of pain in traumatic urethritis is coincident with the introduction of the foreign body. The discharge, which may be bloody, mucous, mucopurulent, or purulent, comes on within twenty-four hours.

*Treatment.*—If the inflammation is slight, prescribe diluent drinks, paregoric, a saline, or the following:

℞. Tinct. belladonnæ..... f℥ss;
Sodii bromid..... ℥iv;
Tinct. opii camphorat..... f℥j;
Syrupus zingib..... f℥ss;
Aquæ destil.....q. s. ad. f℥vj.—M.
Sig.—A tablespoonful every six hours.

If the inflammation is severe, put the patient to bed, apply hot fomentations to the perineum, give diluent drinks, employ suppositories of opium and belladonna, and watch for fever and other complications.

**Gouty Urethritis.**—This condition first manifests itself in the posterior urethra, not in the anterior, as does clap. Its symptoms are great vesical irritability; pain on urination; discharge, usually scanty, associated with uric acid in the urine and perhaps joint symptoms of gout. The *treatment* comprises dieting and the usual remedies for gout. Purgatives are given freely, and full doses of colchicum, piperazin, urotropin, or the alkalis; hot baths, low diet, diluent drinks, and diaphoretics are indicated. A chronic discharge from the prostatic region is apt to linger; for this there is nothing better than the usual gouty remedies and saline waters with copaiba, cubebs, or sandalwood oil. In many cases it is necessary to flush the urethra once a day with a solution of silver nitrate (1 : 8000).

**Eczematous Urethritis.**—Berkley Hill states that this disease is very obstinate, is probably associated with gout, and is met with in adults of full habit or who are beer-drinkers and who have eczema of the surface of the body. He states also that the glans penis near the meatus is red and tender, and that the interior of the urethra is in the same condition. Pain is constant, and it is aggravated by micturition. The discharge is scanty. The *treatment* comprises injections of cold water or irrigation with iced water, and internally the administration of arsenic with the alkalis.

**Tuberculous urethritis** is due to a tuberculous ulcer, which is most apt to be seated near the vesical neck. There is a little pain on micturition, but there is intense pain at one spot on passing a bougie. The discharge is slight and at times bloody. The bladder is very irritable, and severe cystitis arises and persists. The *treatment* includes warmth, nutritious diet, and cod-liver oil, curettement, and local applications of iodoform through a urethroscope, and living as much as possible out of doors. The climate of southern California is peculiarly well suited to these cases. The bladder is washed out once a day with boric acid solution. Iodoform emulsion is injected daily. Tuberculin may prove of value. After a time the surgeon will probably be forced to drain by perineal or suprapubic cystotomy (see Tuberculous Cystitis, pages 1326 and 1327).

**Pyogenic Chancroidal Urethritis.**—This condition is sometimes seen in association with chancroid of the urinary meatus.

**Pyogenic Urethritis of Urethral Chancre.**—A urethritis may occur in this condition.

**Pyogenic Syphilitic Urethritis.**—A temporary and mild urethritis sometimes accompanies early syphilitic eruptions.

**Examination of a Patient in Whom a Urethral Discharge Exists.**—Learn accurately the history. Obtain some of the discharge and examine an unstained slide and a slide stained for gonococci. In some cases take cultures. Learn the amount of the twenty-four-hour urine and study a sample chemically and microscopically, being sure to determine the amount of urea. Learn if the discharge discolors or stiffens linen; if it is only found in the morning; if it simply glues the lips of the meatus together; if it is seen during the day; if it is noted particularly or only after sexual excitement or straining at stool. Inquire as to pain, frequency of micturition, passage of blood, nocturnal emissions, manner of urinating, etc. In many cases insert a finger in the rectum, feel the prostate and vesicles, massage them, and see if discharge appears at the meatus after stripping the penis. If discharge does appear, collect a specimen and examine it. In some cases it is necessary to pass a sound. Carefully cleanse the meatus, glans, prepuce, and urethra before passing a sound. Cleanse the meatus, glans, and prepuce with a 1 : 6000 solution of corrosive sublimate. Irrigate the urethra with boric acid solution and fill and clean urethra with emulsion of iodoform and glycerin (5 per cent.), and after using the instrument irrigate again with boric acid solution (Valentine's method). Examine the urine by the three-glass test.

**The Three-glass Test** (*Valentine's Plan*).—Take as many 3-oz. tubes as are required to receive all the urine from the bladder. The first tube contains the washings from the anterior urethra. The second and other tubes, additional material from the bladder. The last tube contains material expressed from the posterior urethra, prostate, and seminal vesicles. Examine the urine and the sediment in the first two glasses and in the last glass. Note particularly if *shreds* are present. The shreds of gonorrhea are white in color and of variable length, and float in the urine. They are composed of pus-corpuscles and of epithelial cells which have undergone fatty degeneration. Some of these shreds form in the ducts of Cowper's glands, some in the glands of Littré, some in the prostatic sinuses, some in the utricle, some in the folds around the verumontanum, and some from inflammatory patches along the entire length of the urethra. A 1 per cent. solution of methylene-blue injected into the anterior urethra will stain the urethral shreds, while those from the bladder will not be stained by it.

**Gonorrhea (Clap; Specific Urethritis; Tripper; Venereal Catarrh).**—Gonorrhea is an acute inflammation of the genital mucous membrane, nearly always of venereal origin, due to the deposition and multiplication of gonococci in the cells of the membrane and a mixed infection with the cocci of suppuration. The disease is inaugurated by gonococci. After a few days or more secondary pyogenic infection develops and complications may result from the gonococci or from the bacteria causing the mixed infection. The disease attacks with the greatest ease surfaces covered with squamous epithelium. The gonococci enter into and multiply in the superficial epithelial cells and pass between the deeper cells, where they lodge and multiply as the superficial cells are cast off. The pus from the urethra contains epithelial cells with gonococci on or inside of them, and also pus-cells with gonococci within them as a result of phagocytosis. Cultures are made with difficulty. Gonococci do not stain by Gram's method, but stain best with a weak, watery solution of an anilin dye. These bacteria are said not to be pathogenic to animals, although some observers deny this assertion. Gonorrhea is one of the most common and widely disseminated diseases. Probably one-half of all sterile women and many sterile men have been rendered so by this disease. It is responsible for not a few cases of abortion, for an enormous majority of female pelvic diseases, and it causes many cases of blindness by infection of children's eyes during delivery.

**Gonorrhea in the Male.**—In the male, clap begins within the meatus and fossa navicularis and extends backward throughout the length of the urethra. The mucous membrane swells and becomes hyperemic, and there is a discharge, first of mucus and serum, and then of pus. In severe cases the discharge is bloody (*black gonorrhea*) or green. For a week or more the inflammation increases, then becomes stationary for a time, and then declines, the discharge growing less profuse and thinner, a watery discharge lasting for a considerable time. An ordinary case of genuine gonorrhea lasts from six to ten weeks, and even a case limited purely to the anterior urethra will rarely be cured within four or five weeks. During the acute stage the entire penis swells and the corpus spongiosum becomes infiltrated with inflammatory exudate. An interesting fact is that gonorrhea may induce mild septicemia without demonstrable complications, the condition causing, according to Thayer ("Amer. Jour. Med. Sci.," Nov., 1905), a continued fever which, perhaps, lasts a number of weeks. In true gonorrheal septicemia the blood must contain gonococci (*gonococcemia*). In the case recorded by Thayer and in the one recorded by Blumer and Hayes, and in the one recorded by Stellwagen ("Therapeutic Gazette," April 16, 1910, page 248) cultures were obtained from the blood. Gonorrhea may produce grave septicemia with systemic complications. It tends particularly to attack serous membranes or other endothelial structures (joints, pericardium, endocardium, pleura, tendon-sheaths, intima of vessels, etc.). Among the complications are gonorrheal arthritis, myelitis, poliomyelitis, and multiple neuritis. There are 3 cases of gonorrheal myositis on record (Martin W. Ware, "Amer. Jour. Med. Sci.," July, 1901). Phlebitis may arise. Mild endocarditis may arise or severe endocarditis may occur, identical symptomatically with ulcerative endocarditis due to other bacteria. In 6 reported cases of endocarditis gonococci were obtained by cultures from the blood *intra vitam* (Thayer, Loc. cit.). Cerebral embolism may result. Cerebrospinal meningitis can occur (fluid obtained by lumbar puncture contains gonococci).

Gonorrheal rheumatism is discussed on page 636. Gonorrheal peritonitis is rare. Infection of the peritoneum through the blood is very rare. The majority of cases of gonorrheal peritonitis occur in women and are due to direct extension from the Fallopian tubes. Gonococci have not been found in the exudates of cases of pleuritis and pericarditis supposed to be of gonorrheal origin. A child may contract gonorrheal ophthalmia during delivery, and any person may develop it by getting gonococci into the eyes.

**Symptoms of Acute Inflammatory Gonorrhea.**—The period of incubation of gonorrhea is from a few hours to two weeks. The usual period is from three to five days, when symptoms of the *prodromal stage* or *stage of onset* begin. The patient notices on arising a drop of thin fluid which glues the lips of the meatus together, and he feels some heat and itching or tickling about the meatus or in the navicular fossa. There may be uneasiness or actual pain unconnected with urination, and there is sure to be scalding pain on urination. The meatus is red and swollen, has a glazed appearance, may be covered with a little mucopus, and the lips are glued together by the discharge. It may be possible to squeeze out a drop or two. Even so early the fluid contains gonococci. The urine appears clear, but on shaking some flakes are noted. They are epithelial cells. Within forty-eight hours the *first stage*, the *florid stage*, the *acute stage*, or the stage of increase, becomes established. The meatus is now red, swollen, and everted (*fish-mouth meatus*); the entire glans may be red and swollen; if the prepuce is long, it becomes swollen, reddened, and constricted, and in many cases very edematous; the lymphatics by the frenum and on the dorsum of the penis may be red, swollen, tender, and cord-like; micturition causes severe pain (*ardor urinæ*), which is due to distention of

the inflamed urethra and to stinging by the acid urine. Bumstead thus described the act of micturition in acute gonorrhea: "During the act the patient involuntarily relaxes the abdominal walls, holds his breath, and keeps the diaphragm elevated in order to diminish the pressure on the bladder and lessen the size and force of the stream" ("Venereal Diseases," by Robt. W. Taylor). Because of the narrowing of the canal the stream of urine becomes narrow, weak, twisted, forked, or is delivered in little bursts or drops. Retention may result from spasm of the muscles. When the acute or florid stage is fully developed, the entire urethra is inflamed from the meatus to the triangular ligament; there is constant uneasiness or actual pain in the penis and perineum, increased by walking and by sitting down suddenly or carelessly. Insomnia is common; *chordee* occurs, especially when the patient is warm in bed. By "chordee" we mean a condition of painful erection in which the penis is markedly bent. The rigid infiltration of the corpus spongiosum prevents it distending to accommodate itself to the enlarged corpora cavernosa, and in consequence the organ curves. There is frequent micturition, with tenesmus and a profuse creamy discharge, which is yellow, greenish, or even bloody. The discharge soils and stains the victim's linen and may crust upon the linen, the meatus, or the glans. The commonest complications of this stage are *balanitis* (inflammation of the mucous membrane of the glans penis), *balanoposthitis* (inflammation of the surface of the glans and the mucous membrane of the prepuce), *phimosis* (thickening and contraction of the foreskin, so that the glans cannot be uncovered), and *paraphimosis* (catching and fixation of the retracted prepuce behind the corona glandis, with such swelling of the glans and prepuce that it is impossible to bring the prepuce forward over the glans). This is a dangerous condition and it should be reduced at once. In the *second* or *stationary stage*, which lasts from the end of the first to the end of the second week, the acute symptoms of the first stage continue. The most common complications of this stage are *peri-urethral abscess* or *phlegmon* (infection of a urethral gland or of submucous structures), *folliculitis* (inflammation of the follicles of Littré), hemorrhage, retention of urine (which is rare), gonorrheal arthritis, lymphangitis, and diminutive bubo on the dorsum of the penis (*bubonulus*), solitary and painful bubo of the groin, which may suppurate, *Cowperitis* (inflammation of Cowper's glands), inflammation of the prostate or of the bladder, gonorrheal ophthalmia, and *chordee* (painful erection with downward bending of the penis). In the *third* or *subsiding stage* the symptoms gradually abate, the discharge becoming scantier and thinner, and finally drying up. This stage is of uncertain duration, and in it there may occur *epididymitis*, or inflammation of the epididymis. Among possible complications we may mention peri-urethral abscess or phlegmon, Cowperitis, cystitis, prostatitis, bubonulus, folliculitis, gonorrheal arthritis (see page 636), infective endocarditis, tenosynovitis, pyelitis, purulent ophthalmia, perichondritis, and peritonitis.

**Examination for Gonococci.**—Every urethral discharge should be examined for gonococci in order to make a positive diagnosis. This examination is made several times during the progress of the case, so as to determine when the organisms disappear. Many non-gonorrheal conditions are due to cocci strongly resembling gonococci. Free pus and numerous cocci usually mean gonorrhea. So do complications. The examination of the smear is not absolutely conclusive. Cultures are conclusive. If there is a free discharge, fluid for examination can be easily obtained. If the discharge is scanty or occasional, have the patient partially empty the bladder. Then the surgeon massages the prostate and urethra and smears fluid on the slide. Then the patient empties his bladder into two glasses. Each specimen is to be centrifuged and examined for pus (Keyes, "Amer. Jour. Med. Sci.," Jan., 1912).

The taking of a smear and its examination are easy. Place a drop of discharge upon a cover-glass, lay another cover-glass over this, and slide the glasses apart. Dry and fix the slides in the flame of an alcohol lamp. Bring the cover-glasses in contact with a saturated solution of methylene-blue in 5 per cent. carbolic acid water. The staining material is allowed to remain in contact with the slides for five or ten minutes, the glasses are washed with water, are then placed in a solution of 5 drops of acetic acid to 20 c.c. of water, and kept there "long enough to count one, two, three slowly," and are again washed with water. Examination with the microscope shows the gonococci stained blue.[1] In doubtful cases, especially when the microscope fails to show gonococci, make cultures. Cultures must be made in suspected gonorrhea in a child, from the fluid of an inflamed joint, from the discharge in gleet or purulent ophthalmia, and from the blood in obscure infections.

**Subacute** or **catarrhal gonorrhea** develops in men who have previously had gonorrhea, as a result of prolonged or repeated coition or of contact with menstrual fluid or leukorrheal discharge. There is profuse mucopurulent discharge, but very little pain on micturition, and seldom chordee or marked irritability of the bladder.

**Irritative or Abortive Gonorrhea.**—In this disease the symptoms, which are identical with those of beginning clap, do not increase, but disappear within ten days.

**Chronic Urethral Discharges.**—**Chronic urethral catarrh,** which may follow gonorrhea, is characterized by the occasional presence of a drop of clear, tenacious liquid. This discharge becomes more profuse as a result of sexual excitement or the abuse of alcohol.

The persistence of a small amount of milky discharge, because of localization of inflammation in one spot or the production of a granular patch or a superficial ulcer, characterizes *chronic gonorrhea*. There is some scalding on urination; erections produce aching pain; there are pain in the back and redness and swelling of the meatus. All the symptoms are intensified by sexual excitement, by coitus, by violent exercise, or by alcoholic excess.

**Gleet.**—If a chronic gonorrheal urethritis lasts over ten weeks, it is called gleet. In gleet the lips of the meatus are stuck together in the morning, and squeezing them discloses a drop of opalescent mucopurulent fluid (the *morning drop*). During the day the discharge is rarely found. The discharge is yellow or has a yellowish hue; it stains the linen distinctly, and contains pus shreds, epithelium, and at times gonococci. The urine is clear and contains pus, gonorrheal shreds, and comma-shaped hooks. The discharge is not obviously purulent, and contains amyloid corpuscles. There are frequency of micturition, pains in the back, and dribbling of urine, and a bougie may find a stricture of large caliber, or at least will discover that the urethra is rigid from inflammatory infiltration. A discharge may be maintained by *chronic prostatitis*. In this condition there are frequency of micturition; a sense of weight or dull pain in the perineum; diminished projectile force of the stream of urine; there is often a tendency to sexual excitement and premature emission. In *prostatorrhea* a milky discharge gathers in the urethra during sleep and flows during muscular effort or while the patient is at stool. The linen is stained but slightly and the lips of the meatus are not glued together on waking. There is a history of masturbation or sexual excess. The condition is not aggravated particularly by alcohol or sexual intercourse. In chronic anterior urethritis there is a discharge from the meatus or sticking together of the lips in the morning. In chronic posterior urethritis there is no discharge of pus from the meatus. If the three-glass test is made, it will be found that in a case of chronic anterior urethritis only the first portion will be cloudy and show shreds; if there is

[1] Schutz's method, as set forth by R. W. Taylor in his work upon "Venereal Diseases."

posterior urethritis of not very long standing, both portions will be a little clouded, the first containing clap shreds, the last hool-shaped shreds. In a very chronic case neither sample will be cloudy, but the first portion will contain shreds. In gleet the rigidity of the urethra causes the retention of small quantities of urine after each act of micturition, back of the thickened areas. This retained urine decomposes and adds to inflammation. Indulgence in alcohol, sexual excitement, or sexual intercourse aggravates the condition.

**Treatment of Acute Gonorrhea.**—*General Care.*—Wash the hands after touching the parts and dry them on an *individual towel*, which is not used upon the face. Wear a suspensory bandage. Avoid violent exercise, especially bicycle riding, and also wet. Moderate exercise is allowable. The patient must not only refrain from sexual intercourse, but must not permit himself to indulge in sexual excitement, and must not drink a drop of liquor, malt, vinous, or spiritous, unless he is a heavy or a regular drinker, in which case we should permit the moderate use of well diluted rye or Scotch whisky. Some men become actually ill without their regular daily stimulants and such men should have them in moderation. Some surgeons permit the moderate use of claret to all patients. At least twice a day wash the penis for five minutes in a cup of warm water containing 1 dram of salt. Passing the urine while the penis is immersed in warm fluid lessens ardor urinæ. Cut a small opening in a square piece of old linen, slip the linen over the glans, catch it back of the corona, and bring the ends forward with the prepuce. Never permit the cotton or linen to stick fast and plug up the lips of the meatus. If the lips tend to become sealed up, grease them with sterile vaselin. If the glans is completely naked, pin the foot of an old stocking upon the undershirt, put absorbent cotton in the toe, and place the penis within this bag. Never tie or fasten any material about the penis. The patient should drink freely of plain water or of water containing a little bicarbonate of sodium. He should obtain one bowel movement every day.

*Diet and Instruction List* (Dr. T. C. Stellwagen, Jr.).—*Meats.*—May have white meat of chicken, boiled fish, lamb.

Must not take beef, beef steak, veal, pork, liver, kidney, etc.; nor salt fish, meats, smoked, canned, or potted foods.

*Vegetables.*—May have rice, hominy, string beans, fresh peas, spinach, beans, baked potatoes (sweet and white), lentils, etc.

Must not take rhubarb, tomatoes, asparagus (Guitéras's rule. See Begg, in "Phila. Med. Jour.," June 7, 1902), lemons, oranges, limes, or preserved fruits.

*Drinks.*—Nothing but plain water and milk.

Must not take alcoholic beverages or carbonated drinks.

Must not take salt, spices, condiments, cheese or pickles.

*Desserts.*—Only of the simplest kind, made from milk and eggs, and take them only sparingly.

*General Rules.*—Put on a suspensory bandage and wear it in conjunction with a piece of linen or a bag to catch discharges.

Wash the hands after handling the parts, and keep the fingers away from the eyes.

Keep the linen from contaminating the family wash.

Wash the organ frequently in warm salt water (1 teaspoonful of table salt to 1 glass of water).

When urination causes burning, immerse the organ in a basin of warm water and allow the urine to flow.

Keep the body warm and free from chilling.

Should not go about on rainy or snowy days without ample protection.

Should go to bed early.

Must not take cold baths. May take warm ones.

Should not eat between meals—it is unwise to overload the stomach.

Should drink freely of plain water or milk.

Should urinate when the desire comes.

Should keep away from females socially and sexually—and any other exciting influences, such as erotic sights, thoughts, and literature.

Must keep bowels loose.

A smoker should decrease the consumption of tobacco.

Never put pieces of cotton over the end of organ, as cotton so used prevents the free drainage of discharge. Should the lips of the opening seal together, wash clean in warm salt water and grease with vaselin.

A tea or coffee drinker may have a small amount once daily, but it will be better not to partake of either.

Should the patient be using an injection and find that the desire for urination becomes frequent at night, he must stop the injection and consult his physician.

The injection is never to be forced into the organ; it is to be introduced gently, and the bladder is to be empty when an injection is given.

*Abortive treatment* may be tried if the case is seen early. The use of strong solutions of powerful germicides has been abandoned because of the great pain they produce and the inevitable subsequent crippling of the urethra.

Abortive treatment is applicable only to specially selected cases and even then usually fails. It should never be used after the gonococci have invaded the submucosa, but only during the prodromal stage, when it is hoped that the germs are upon and not in the mucous membrane. This stage usually lasts but a few hours, usually not over forty-eight. The patient generally presents himself after this period has passed. When the symptoms as described in the prodromal period prevail, the abortive treatment may be tried, after explaining to the patient that it will cause some pain and discomfort and in the end may fail or even aggravate the inflammation.

Germicides may be used and with some chance of killing the infection, for as yet the organisms are growing upon the superficial strata of epithelium much as a sod of grass upon soil. When the deeper structures have been invaded it is folly to attempt to abort the disease.

The method advocated by White and Martin and which has proved successful in Prof. Hiram Loux's clinic in a limited number of selected cases is as follows: After urination 4 drops of a 4 per cent. solution of eucain are injected into the urethra; after which 2 drams of $\frac{1}{4}$ per cent. solution of protargol are instilled and retained for three minutes. The injections are repeated every two hours while awake.

Each time the bottle is half emptied it is replenished with sterile water to its full capacity, until the end of the third day. If successful, recovery is accomplished in about seven days. Should a mucoid discharge persist an antiseptic astringent injection is employed to complete the cure. During the treatment the patient must be kept at rest. The diet should be bland and light and the usual balsamic remedies are administered. If the symptoms become hyperacute, stop the treatment at once and give a sedative.

Another abortive method is the use of hot retro-injections of corrosive sublimate solution (1 : 20,000), 2 pints being run through the urethra once a day. If in seventy-two hours the symptoms are not greatly improved, abortive treatment should be abandoned. Recent studies render it almost certain that there is no real abortive treatment. Abortive treatment, to be efficient, would have to be carried out before the gonococci penetrated the epithelial cells; in other words, would need to be instituted before the distinct symptoms of the disease appear. Janet says that we must alter our conception as to what

constitutes abortive treatment, and he doubts if a case of true gonorrhea was ever really aborted.[1] The method of irrigation with solutions of permanganate of potassium is really a prophylactic treatment. Janet applies his treatment as evidences of trouble present themselves, and before acute symptoms appear, and claims that in most persons the disease can be arrested in from eight to twelve days. The same plan of treatment is useful in a well-developed case.

*Irrigation in Gonorrhea.*—Irrigation can be used in an incipient or in a well-developed case. Janet's method is as follows: An irrigator is filled with a warm solution of permanganate of potassium (1 : 4000). The patient after emptying his bladder is seated upon a chair and his sacrum rests upon the extreme front edge of the chair (Valentine's position). The reservoir is joined to a glass nozzle by a rubber tube. The nozzle is introduced into the meatus, and the fluid is permitted to run gradually at first, with full force later. In anterior trouble the fluid is allowed to run out of the meatus by the side of the nozzle. The anterior urethra is always irrigated first, the reservoir being 2 feet above the chair.

In posterior urethritis, after the anterior urethra has been irrigated, the reservoir is raised from 6 to 7 feet above the bed, the meatus is held tight about the nozzle, and the fluid overcomes the force of the compressor muscles of the urethra and the bladder sphincter and enters the bladder. If the muscles do not quickly relax, continue the hydrostatic pressure for several minutes, when relaxation will usually occur; but if it does not do so, tell the patient to breathe slowly and deeply, and to make efforts at urination (Valentine's plan). When the bladder is full the tube is withdrawn and the patient micturates. This procedure is practised once or twice a day for five or six days, or even longer, and the strength of the solution is gradually increased up to 1 : 1000. It has been claimed that after one or two weeks of this treatment gonococci permanently disappear in the majority of cases. Figure 900 shows the ingenious and very useful irrigator devised by Ferd. C. Valentine, of New York. Valentine[2] has constructed the table on page 1352, which is of use to a practitioner who wishes to employ irrigations with permanganate of potassium in the treatment of acute gonorrhea. I followed the method set forth in the table in a great number of cases, and regarded it an extremely useful systematic plan.

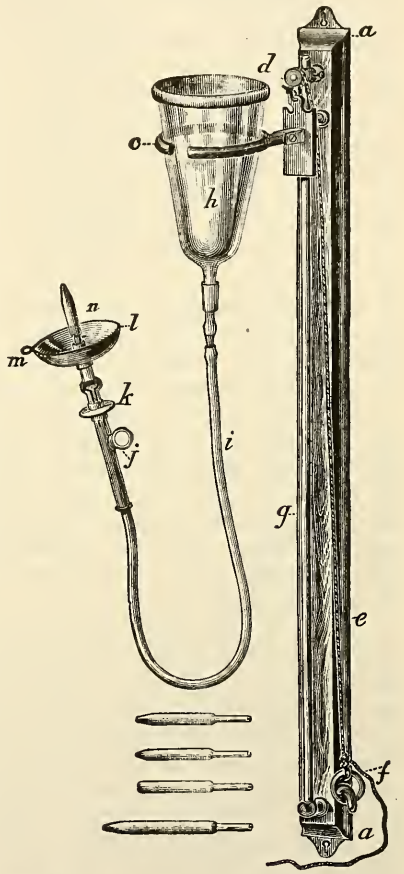



Fig. 900.—Valentine's urethral and intravesical irrigator: *a*, Board with attachments to be screwed to wall; *c*, open collar; *d*, pulley; *e*, cord; *f*, ring to suspend percolator; *g*, brass rod; *h*, percolator; *i*, rubber tube; *j*, ring for fourth finger; *k*, flange to graduate pressure; *l*, shield; *m*, ring to suspend shield; *n*, nozzle attached.

[1] "Ann. d. mal. d. org. gén.-urin.," 1896, p. 1031.
[2] "The Irrigation Treatment of Gonorrhea."

| | | | |
|---|---|---|---|
| First day, first visit. | | Anterior irrigation | 1 : 3000 |
| First day, | 7 P. M. | Anterior " | 1 : 4000 |
| Second day, | 9 A. M. | Anterior " | 1 : 3000 |
| Second day, | 7 P. M. | Anterior " | 1 : 4000 |
| Third day, | 9 A. M. | Intravesical " | 1 : 6000 |
| Third day, | 7 P. M. | Anterior " | 1 : 5000 |
| Fourth day, | 9 A. M. | Intravesical " | 1 : 5000 |
| Fourth day, | 7 P. M. | Intravesical " | 1 : 5000 |
| | | Anterior " | 1 : 2000 |
| Fifth day, | Noon. | Intravesical " | 1 : 5000 |
| Sixth day, | Noon. | Intravesical " | 1 : 5000 |
| Seventh day, | Noon. | Intravesical " | 1 : 5000 |
| Eighth day, | 9 A. M. | Intravesical " | 1 : 5000 |
| | | Anterior " | 1 : 3000 |
| Eighth day, | 7 P. M. | Intravesical " | 1 : 5000 |
| | | Anterior " | 1 : 2000 |
| Ninth day, | 9 A. M. | Intravesical " | 1 : 4000 |
| | | Anterior " | 1 : 1000 |
| Ninth day, | 7 P. M. | Intravesical " | 1 : 4000 |
| | | Anterior " | 1 : 1000 |
| Tenth day, | 9 A. M. | Intravesical " | 1 : 4000 |
| | | Anterior " | 1 : 1000 |
| Tenth day, | 7 P. M. | Intravesical " | 1 : 5000 |
| | | Anterior " | 1 : 500 |

For full directions regarding this method see Valentine's book, "The Irrigation Treatment of Gonorrhea." If a stricture exists, it is not advisable to employ this treatment. Excellent results can be obtained by irrigations with fluid containing silver nitrate (1 : 12,000 to 1 : 8000).

When a patient is treated by irrigation, after the entire subsidence of acute symptoms, a thin, colorless discharge may persist. This can be cured by the use of astringents. Two or three times a day an astringent is injected by means of a ½-ounce syringe. Dalton's formula is very useful: Zinc oxid and lead acetate, of each, ½ to 3 gr.; tincture of catechu, from 10 to 30 min.; glycerin, from ½ to 1 dram; and water to make 1 oz. Ultzmann's formula is: Zinc sulphate, 16 gr.; pulverized alum, 8 to 12 gr.; carbolic acid, 3 gr.; and water to make 4 oz.

Many writers oppose the irrigation treatment, claiming that it increases the liability to complications, especially prostatic inflammation, and enhances the danger of recurrence. I believe in the method. I do not think it shortens materially the duration of the disease, but do believe that it mitigates its intensity, makes the patient much more comfortable, and quickly causes the discharge to become mucopurulent. That it increases complications and the danger of reinfection is very doubtful. Much of the trouble which has followed its use has been due to raising the reservoir to too great a height.

*Irritative gonorrhea* will subside in a few days. The above directions should be followed, and the anterior urethra should be washed out several times daily with diluted peroxid of hydrogen, or irrigated once a day with a hot solution of permanganate of potassium (1 : 4000). In *catarrhal gonorrhea* at once order injections (1 gr. to the ounce of sulphate of zinc; or zinci sulphas, 8 gr., plumbi acetas, 15 gr., water 8 oz.; or 5 gr. of sulphocarbolate of zinc to 1 oz. of water; or White's prescription of 1 dram each of acetate of zinc and tannic acid, 8 drams of boric acid, 6 oz. of liq. hydrogen peroxid). For injecting use a blunt-pointed hard-rubber syringe of a capacity of 3 or 4 drams. Let the patient urinate and then sit on a chair, his buttocks hanging over the edge; throw a syringeful of the solution into the urethra and let it run out at once, and throw in another syringeful and hold it in from three to five minutes.

In *ordinary acute gonorrhea* we follow an old rule when we order balsams. The common custom is to give two capsules three times a day, each capsule

containing 5 gr. of salol, 5 gr. of oleoresin of cubebs, 10 gr. of balsam of copaiba, and 1 gr. of pepsin. Clinical observation indicates that the balsams are of distinct value in gonorrhea. When used early, the discharge tends to become mucopurulent and the acute symptoms usually soon begin to subside (S. Behrmann, in "Dermatologisches Centralblatt," Berlin, Nov. and Dec., 1901). Many practitioners will not use balsams until the third week. Bacteriological studies indicate that copaiba, when eliminated in the urine, has a certain amount of power in inhibiting the growth of gonococci, but that cubebs and oil of sandal have not such power. Yet oil of sandal is more useful than copaiba as a remedy. Salol is distinctly germicidal, hence it is given with the balsams. In a case treated by balsams an astringent injection is usually employed. The injection is used two or three times a day, immediately after micturition. As the inflammation subsides the strength of the injection should be gradually increased. A good plan is to order an 8-oz. bottle and 8 1-gr. powders of sulphate of zinc. Direct the patient to fill the bottle with water, in which one powder is dissolved; when this is used dissolve 2 powders in a bottleful of water, and so progressively increase the strength. When the discharge ceases stop the injections gradually. Whenever a syringeful is taken from the bottle a syringeful of water is put into the bottle, and thus pure water is soon obtained, at which point injection is discontinued. If a simple astringent injection causes much pain, use a sedative injection—2 drams of boric acid, 8 gr. of aqueous extract of opium, and 8 oz. of liquor plumbi subacetatis dilutis. I have had about as much success with the above simple method as with the most complicated of plans. Complication and complexity are not criterions of usefulness.

Argonin, which is a combination of albumin, silver, and an alkali, is highly recommended by some authors as a local remedy for gonorrhea (Schäffer, Guthiel). A solution of this material is non-irritant, the silver is not precipitated by chlorids, and the agent destroys gonococci. It is used by injection or irrigation. If used by irrigation, employ a 1 : 500 solution twice a day. If used as an injection, employ a 1 : 200 solution six or eight times a day. When the discharge is found free from gonococci and remains free for three days, stop the argonin and use an astringent injection.

Protargol, metallic silver combined with a protein, is a yellow powder soluble in water, the solution not being acted on by light. It is a non-irritant germicide. Neisser, after demonstrating the presence of the gonococcus, administers protargol by injection, the first injections being of a strength of 0.25 per cent., the strength being gradually increased to 0.5 per cent., and finally to 1 per cent. In the beginning he orders three injections a day, each injection being retained from fifteen to thirty minutes; after several days, when the symptoms improve, he gives only one or two injections a day, and these are continued for ten days after gonococci disappear from the discharge. After protargol is abandoned an astringent injection should be used for a time. Some surgeons use a 1: 1000 solution of protargol, and irrigate the anterior urethra and flush the bladder twice a day. A silver salt used by many is argyrol, or silver vitellin. The injection used at first may be of a strength of 2 per cent. The drug should be retained in the urethra four or five minutes, and three or four injections should be given each day. The strength of the injection can be gradually increased to 5 per cent. or even more. I have not been impressed with the value of this preparation except in the earliest stages of gonorrhea. Picric acid has been highly commended as an injection. The strength of solution is 1 : 200, and it is to be retained in the urethra three or four minutes (de Brun's method).

Methylene-blue internally is occasionally of service in gonorrhea. A capsule containing 1 or 2 gr. of the drug is given three times a day. It makes the

urine greenish blue and occasionally induces strangury. Urotropin renders the urine sterile. Salicylate of sodium may be of value late in the case.

In his clinic in Jefferson Hospital my colleague, the late Prof. Horwitz, introduced the following plan of treatment: A capsule containing balsam of copaiba, salol, oil of sandal, and methylene-blue is given half an hour after each meal:

℞. Methylene-blue ..... gr. xxx;
Balsami copaibæ }
Olei santali } ..... āā f℥iss.—M.
Pone in capsulas No. xxx.
Sig.—One after each meal.

The patient begins at once to take, by hand injection, a 10 per cent. solution of argyrol. He takes it three times a day,

At each daytime injection the fluid is retained in the canal for five minutes. At the bedtime injection the fluid is retained for fifteen minutes. At first only about 1 dram is injected at a time, but as the urethra becomes accustomed to fluid distention the amount is gradually increased, until finally 4 drams may be given.

When the stage of decline begins (toward the end of the second week) a combined astringent and antiseptic treatment is used.

The capsules of methylene-blue, copaiba, and sandal, and the injections of argyrol are discontinued. A capsule containing oleoresin of cubebs, oil of sandal, and balsam of copaiba is given before each meal, and a capsule of urotropin is given after each meal. An injection of protargol ($\frac{1}{4}$ of 1 per cent.) is given three times a day. This remedy is germicidal and also astringent. The strength is gradually increased until a 1 per cent. solution is used. By the end of the fourth week the patient has reached the terminal stage. Now capsules of sandal and salol are substituted for the cubebs, sandal and copaiba and an astringent injection is ordered. The following injection, recommended by J. Wm. White, is very satisfactory:

℞. Hydrarg. chlor. corros. ..... gr. $\frac{1}{8}$;
Zinci sulphocarbolas ..... ℥ss;
Acidi boraci ..... ℥ij;
Acidi carbolici ..... ♏xv;
Boroglycerid (25 per cent.) ..... f℥ij;
Aquæ destil. ..... q. s. ad. f℥vj.—M.
Sig.—Inject. Dilute if it causes much pain.

When the mucoid condition predominates in the discharge the following is useful:

℞. Zinci sulph. ..... gr. xv;
Plumbi acetati ..... gr. xxx;
Glycerol tannin }
Hydrastin (Lloyd's) } ..... āā f℥iv;
Mucil. acaciæ ..... f℥iv;
Aquæ destil. ..... q. s. ad. f℥vj.—M.
Sig.—As injection.

The formula of the "injection Brue" is as follows:

℞. Plumbi acetat. ..... gr. xxx;
Zinci sulphat. ..... gr. xvj;
Ext. krameriæ fl. ..... f℥iv;
Tinct. opii ..... f℥iij;
Aquæ destil. ..... q. s. ad. f℥vj.—M.
Sig.—As injection.

When all symptoms of clap have disappeared, any injection in use is gradually diluted. Whenever a syringeful of the fluid is taken from the bottle, a syringeful of water is put in. When the fluid becomes pure water the injection is discontinued. Balsams are stopped by gradual diminution in the number of daily doses. For three weeks after the entire disappearance of all symptoms alcohol is forbidden and sexual indulgence is prohibited. Should a relapse occur the patient is at once placed upon treatment as for the acute stage, and when the stage of decline again ensues he is placed again on antiseptics and astringents. Relapses are caused by a localized lesion or lesions in the anterior or posterior urethra, hence as soon as all acute symptoms subside an endoscopic examination is to be made and proper treatment is to be applied to any localized lesion. If during the treatment of gonorrhea a complication develops, local treatment of the urethra is at once discontinued and constitutional treatment suited to the new condition is prescribed.

If the onset of gonorrhea is marked by violent inflammatory symptoms (chordee, hemorrhage, severe pain, swelling, profuse purulent discharge), local treatment of the urethra is contra-indicated.

If the invasion is hyperacute, no local treatment of the urethra is permissible until the disease assumes the character of ordinary gonorrhea.

Not unusually gonorrhea passes into a low grade of anteroposterior urethritis that proves most rebellious to treatment. This condition is especially common when too stimulating treatment has been used. A mild astringent injection should be used three times a day. Every second or third day an injection of nitrate of silver (1 : 4000) should be given. If the silver salt sets up an acute inflammation the treatment used in the acute stage of gonorrhea is given until the symptoms abate.

A valuable plan in rebellious anteroposterior urethritis is daily irrigation of the anterior urethra with a warm solution of permanganate of potassium (1 : 6000), followed by the passage of a soft catheter and the filling of the bladder with a like solution. The patient empties his bladder after the catheter is withdrawn and thus flushes the entire urethra with the permanganate. The strength of the solution is gradually increased up to 1 : 2000.

Weiss uses corrosive sublimate (1 : 20,000) in the manner just described. It is particularly valuable in cases of bacterial contamination, the gonococci having disappeared.

**Treatment of Complications.**—*Ardor urinæ* is relieved by urinating while the penis is immersed in hot water and by administering alkaline diuretics. *Chordee* requires a bowel movement in the evening, and sleeping in a cool room, under light covers, and on a hard mattress; bromid of potassium is given several times daily, and a considerable dose is given at night; it may be necessary to use suppositories of opium and camphor or to give hyoscin. *Balanitis* requires frequent washing of the head of the penis and prepuce in warm water, drying with cotton, and dusting with borated talc, stearate of zinc, or boric acid and subnitrate of bismuth (1 : 6). *Balanoposthitis* requires soaking in hot water, and injections of black wash under the prepuce until edema of the foreskin subsides, and then cleanliness and the application of a drying powder. *Phimosis* requires soaking the penis in hot water, and injections of hot water beneath the foreskin, followed by black wash. If this fails, circumcision must be performed. If *paraphimosis* occurs, grasp the head of the penis with the left hand, squeeze the blood out, and try to push the head back, while with the right hand the penis is pulled upon as if the surgeon intended to lift the individual by the organ. If this fails, cut the collar on the dorsum with scissors; or, what is better, for it gives free exposure, incise each side of the prepuce between the middle of the dorsum and the frenum. *Bubo* requires the application of iodin, ichthyol, or blue oint-

ment, the use of a spica bandage, and rest. If a bubo suppurates, it must be opened or aspirated. *Acute posterior urethritis* is treated by rest, and if the symptoms are severe, by rest in bed. If the balsams do not irritate, they are given; if they do, they are withdrawn. Urotropin or salol is given and the patient is placed upon a milk-diet with orders to drink largely of flaxseed tea. Alkaline fluids do harm by favoring ammoniacal decomposition of the urine. Injections and irrigations are abandoned. Pain and vesical spasm are controlled by suppositories of opium and belladonna. If retention of urine occurs, have the patient urinate while in a hot bath; if this fails, use a soft catheter. *Acute vesiculitis* is treated as is acute prostatitis. Chronic vesiculitis is considered on page 1375. *Pyelitis* is treated by rest in bed, hot baths, wet cupping of the loin, milk-diet, diuretics, the taking of a large quantity of bland liquid, and the administration of salol or urotropin, and in some cases lavage of the kidney pelvis through a ureteral catheter with one of the silver preparations (1 : 15,000 of nitrate of silver). *Folliculitis* is treated by rest and the application of a hot-water bag to the perineum (if that be the part involved). If pus forms, evacuate by incision. Later the follicle may be dissected out or destroyed by cauterization. If the follicle opens into the urethra it may be cauterized through an endoscope. *Peri-urethritis* is treated by rest and hot applications. If pus forms, an incision must be made. If the abscess is permitted to break into the urethra, rest and hot fomentations may be used, but at the first sign of urinary extravasation make an external incision. *Cowperitis* is treated in the same way as peri-urethritis. Gonorrheal rheumatism is considered on page 636. *Acute prostatitis* and *cystitis* require confinement to bed, a milk-diet, the use of diuretics, hot applications to the perineum and hypogastrium (bags of hot sand), suppositories of opium and belladonna or of ichthyol, leeching the perineum, the discontinuance of balsams and injections, and the administration of urotropin or salol. Heat can be applied by means of a stream of warm water which circulates through a metal instrument introduced in the rectum. *Abscess of the prostate* requires instant incision. In *retention of urine* the patient should try to pass the urine while in a hot bath; if this fails, a soft catheter is used. After relieving the bladder put the patient to bed and apply hot sand-bags as for acute prostatitis. *Chronic prostatitis* requires cold hip-baths, cold-water enemata, deep urethral injections, massage of the prostate, plain diet, avoidance of alcohol and overexertion, counterirritation of the perineum, and the relief of stricture or phimosis. Great benefit is occasionally derived from passing a soft bougie covered with blue ointment or with a 1 per cent. ointment of silver nitrate. If there are pus pockets, gonorrheal phylacogen may perhaps be of service. If *epididymitis* arises, put the patient to bed, abandon injections, shave the hair from the groin, leech over the cord, elevate the testicles, and early in the case apply an ice-bag. Give a cathartic, a fever mixture, and suitable doses of bromid of potassium and morphin. The local application twice a day of 20 drops of guaiacol in 1 dram of cosmolin or olive oil gives relief. When swelling lingers, after tenderness subsides strap the testicle with adhesive plaster. A lingering case is benefited by the internal use of iodid of potassium and the local application of ichthyol. In *gonorrheal ophthalmia* secure a watch-crystal over the unaffected eye, put the patient in a darkened room, rub the infected conjunctival sac with cotton soaked in a 2 per cent. solution of silver nitrate, wash out the affected eye repeatedly with hot boric acid solution, keep the pupil dilated with atropin, leech the temple, and give purgatives. Always send for an ophthalmologist.

**When is Gonorrhea Cured?**—It is said that Ricord declared: "We know when clap begins, but God alone knows when it ends." When actual discharge ceases a patient considers himself cured, and yet he may have residues of

infection which are liable at any time to awaken into activity and produce anew an acute condition. Gonococci are frequently retained in the urethral glands and follicles or in areas surrounded by indurated mucous membrane. Gonococci may linger in these haunts for many months, some say for years. Keyes, however, maintains that they never persist in the male urethra over three years and that in 90 per cent. of cases, with or without treatment, they disappear within one year (Edward L. Keyes, Jr., in "Amer. Jour. Med. Sci.," Jan., 1912). It is customary to consider a man well when, after he has been without treatment for one week, shreds and pus disappear from the urine, when an examination of expressed mucus on three successive days fails to find gonococci, and when there has been no discharge for ten days. Furthermore, we must be sure that the prostate, Cowper's glands, and the seminal vesicles are free from disease. Never declare a man well under three months from the start of the disease unless gonococci are positively absent from the urine and the expressed discharge.

If a patient has "no morning drop," has been apparently well for three or four months, has no pus in the morning urine or in the expressed secretion of the urethra, prostate, and seminal vesicles, we can, without making cultures, affirm that he is well (Keyes, Ibid.).

Keyes also maintains that a man with a "pearly morning drop" who passes a urine containing pus-shreds, but who has no free pus in expressed secretions, is probably free from gonorrhea.

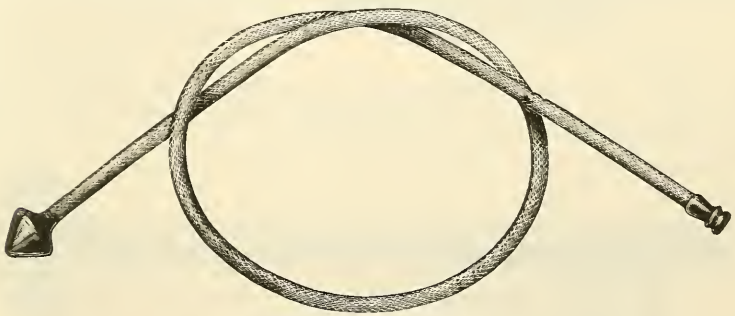


Fig. 901.—Bougie-à-boule.

**Treatment of Chronic Gonorrhea and of Chronic Urethritis Following Gonorrhea.**—The first thing to do is to determine the *cause* of the prolongation of the discharge. Valentine's list of causes should be borne in mind ("Med. Record," June 29, 1901). They are as follows: (1) Lack of treatment; (2) misdirected treatment; (3) insufficient treatment; (4) overtreatment; (5) infraction of dietetic or hygienic regulations; (6) constitutional disturbances; (7) congenital or acquired deformities and complications; (8) involvement of the urethral adnexa; (9) marital reinfection. In a case in which a discharge persists or recurs the symptoms and general condition must be closely studied, the discharge must be examined microscopically, the condition of the urine must be determined, and the urethra must be explored.

*Exploration* of the urethra is inaugurated by inspection and external palpation. Palpation detects induration, peri-urethritis, follicular abscess or inflammation, Cowperitis, etc. The prostate and seminal vesicles are examined by a finger in the rectum. The interior of the urethra is explored by a soft bougie-à-boule (Fig. 901). On withdrawing this instrument the shoulder catches in any contracture. It is to be borne in mind that a large steel sound can often be introduced with ease when the bougie-à-boule makes evident that a contracture exists. The emergence of the instrument is arrested

by a patch of thickening, a granular area, a zone of epithelial proliferation, a papilloma, or a stricture. In fact, anything which lessens the urethral caliber interferes with the withdrawal of the bougie-à-boule. It does not do to conclude that stricture exists simply because some lessening of caliber is appreciated. The bougie-à-boule finds its chief use in exploring the anterior urethra. If introduced into the deep urethra its emergence will be normally checked as its shoulder comes against the posterior layer of the triangular ligament.

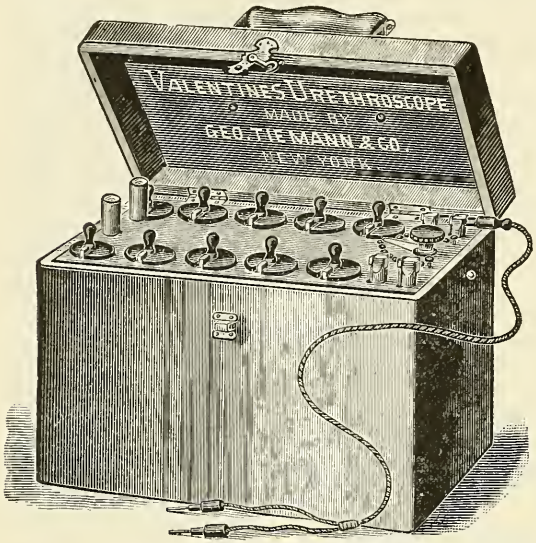



Fig. 902.—Valentine's urethroscope.

In most cases the diagnosis is only certainly determined by the use of the urethroscope. This instrument has been perfected of recent years and is now an absolutely essential part of our armamentarium. I have long used Valentine's instruments (Figs. 902–905). Marks's air-dilating urethroscopes (Figs. 906, 907)

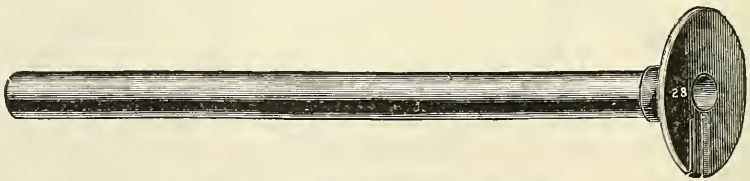


Fig. 903.—Valentine's urethroscopic tube.

are highly satisfactory. The anterior and posterior urethra can be thoroughly examined and with the utmost ease. Before inserting a urethro-

Fig. 904.—Valentine's obturator.

scopic tube place the patient recumbent and cleanse the foreskin, glans, and anterior urethra as directed in the section on Cystoscopy. Insert a tube which

Fig. 905.—Valentine's light carrier.

readily passes the meatus, first cleansing the tube and obturator by burning alcohol upon them. Carry the tube to the anterior layer of the triangular liga-

ment. Withdraw the obturator and insert the light. Turn on the light, mop the urethra with bits of cotton wrapped on a stick, and slowly withdraw the tube, examining the urethra as its walls fall together back of the retracting tube. After withdrawal of the tube irrigate the anterior urethra. To examine

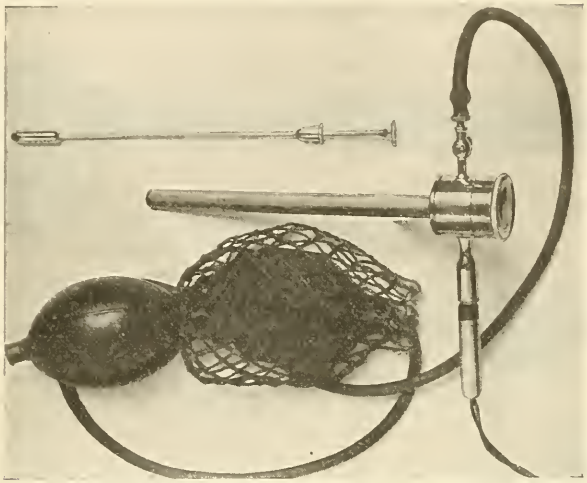


Fig. 906.—Marks's air-dilating urethroscope for examination of the anterior urethra.

the deep urethra carry the instrument through the prostatic urethra. After the examination give an intravesical irrigation.

When the cause of a discharge is once determined, rational treatment can be instituted, and to determine the cause the electric urethroscope is indispensable. An erosion of the mucous membrane or a granular patch requires

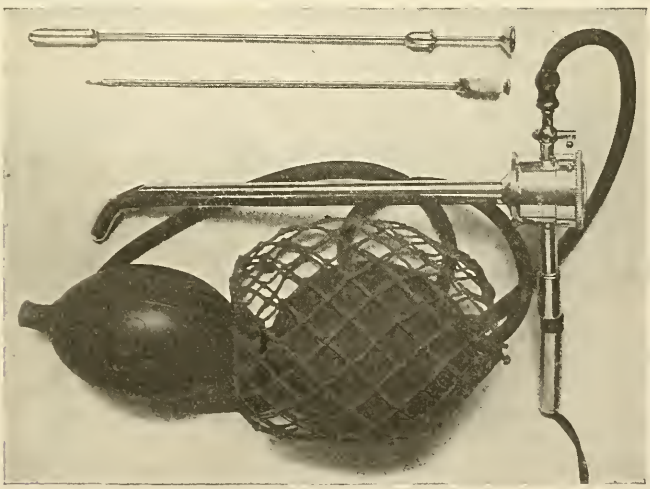


Fig. 907.—Marks's air-dilating urethroscope for examination of the posterior urethra.

touching from time to time with a solution of silver nitrate (1 or 2 per cent. or even much stronger). These applications are made through the tube of the urethroscope. A stricture or an infiltration is treated by gradual dilatation. This combines pressure and massage. If the caliber of the urethra is less than

No. 21 of the French scale, conical steel sounds are used twice a week. If there is much hyperesthesia they are retained but a brief time; but as hyperesthesia diminishes the period of retention is lengthened, until an instrument can be kept in place without causing severe suffering for ten or fifteen minutes. It is not desirable to use cocain. Its use is not free from danger and, further, it

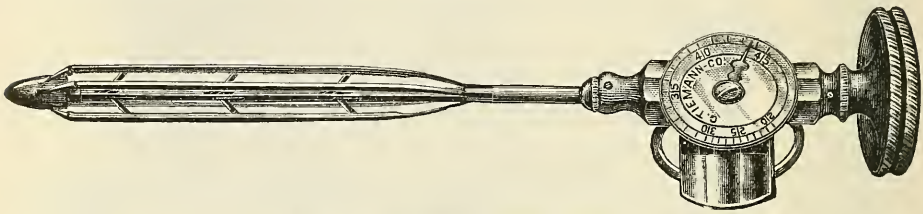


Fig. 908.—Kollmann's anterior dilator.

Fig. 909.—Oberlander's anterior dilator.

Fig. 910.—Kollmann's anteroposterior dilator.

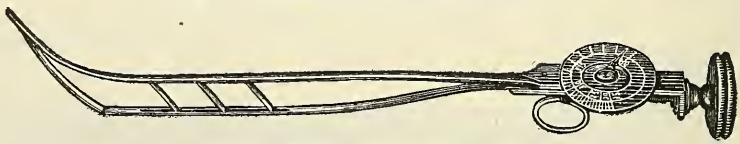


Fig. 911.—Oberlander's anteroposterior dilator.

obtunds the sensibility so that undue violence may be used, and it increases postoperative inflammation. Before and after using an instrument the urethra must be cleansed by irrigation with salt solution or permanganate of potassium.

When the urethra becomes tolerant to instrumentation, a special dilator is employed to act particularly on the area of disease. If in the beginning of treatment the caliber of the urethra is equal to or greater than No. 21 of the

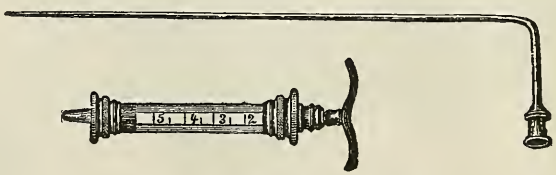


Fig. 912.—Kollmann's gland syringe.

French scale, it is rarely necessary to precede the dilator by the use of conical sounds. Figs. 908–911 show various dilators. A dilator unless of a very modern type should be inserted in a sterile rubber cover before being used, otherwise it will cut, tear, or pinch the urethra when closed and withdrawn. Kollmann's dilator (new style) will not injure the mucous membrane and can

be used without a cover (Fig. 908). A dilator should be lubricated with lubrichondrin, synol soap, or liquid cosmolin. If a two-bladed dilator is used at first, a four-bladed dilator must be subsequently employed.

A dilator is cleansed by scrubbing its blades with soap and water, sticking them in alcohol, withdrawing, and burning the alcohol retained in the instrument.

The following rules for dilatation are of the first importance (Ferd. C. Valentine, in "Med. Record," June 29, 1901):

1. The first dilatation must stop at that point at which the first resistance to further dilatation is felt by the operator's fingers turning the screw that separates the blades.

2. Dilatations, if done by a novice, must in the beginning of treatment be repeated no oftener than every three or four days.

3. Each dilatation, in point of time, must reach no greater duration than two minutes over that of the preceding session.

4. No dilatation must exceed one-half number Chariére above the number attained at the next prior séance, regardless of any lack of resistance that may be present.

As a rule, glandular and follicular infiltrations are cured by the use of the dilator. If they are not, they must be treated through the tube of the urethroscope. The interior of a follicle may be cauterized by a heated electric wire, subjected to electrolysis, fulgurated by the Oudin current, or touched with a 3 per cent. solution of silver nitrate. A thickened crypt, gland follicle, or an area of induration may be slit by a knife. A polyp can be removed by a snare, the cautery, or special forceps. In a chronic inflammation of the urethra, in which the inflammation is superficial and in which the glands are not involved, irrigations, urethral and intravesical, constitute the best treatment. (See Valentine's treatise on "The Irrigation Treatment of Gonorrhea, its Local Complications and Sequels.")

In any lingering case of gonorrhea examine the urine, and direct suitable treatment for oxaluria, lithemia, or phosphaturia, if any one of these conditions exists. Such morbid states of the urine are occasionally responsible for great prolongation of the inflammation. In some cases a discharge is kept up by inflammation of the seminal vesicles (see page 1375).

**Gonorrhea of the anus and rectum** occasionally, though very rarely, occurs. It may result from pederasty, or in a woman from a flow of infectious material from the genitalia to the anus. It causes severe burning pain, aggravated by defecation. The parts are red, swollen, and tender. The discharge is profuse, being at first cream white, and then thicker and greenish. The diagnosis rests upon the history and the finding of gonococci in the discharge. The disease rarely extends above the anus. I have seen one undoubted case of rectal gonorrhea.

**Treatment.**—If the anus only is involved, spray it several times daily with peroxid of hydrogen, wash with salt solution, irrigate with permanganate of potash (1 : 4000), dust with talc powder, and interpose a piece of iodoform gauze between the inflamed surfaces. An ulcer, a fissure, or an excoriation is touched with lunar caustic. If the rectum becomes involved, secure a daily bowel movement and irrigate the rectum twice a day with boric acid solution or permanganate of potassium (1 : 4000).

**Gonorrhea of the Mouth.**—This is a very uncommon malady. It occurs in infants more often than in older people. Infection in infants may take place during birth if the mother has gonorrhea. The symptoms are those of violent stomatitis. The diagnosis is suggested by the condition of the mother and is proved by finding gonococci in the discharges from the mouth.

**Treatment.**—Wash the mouth frequently with boric acid solution, and swab the diseased areas at intervals with a 10 per cent. solution of argyrol.

**Gonorrhea of the Nose.**—It is alleged that this condition can arise, but an absolutely authentic case does not seem to be on record.

**Gonorrhea in the Female.**—There is much dispute as to the parts infected. Some observers maintain that the vaginal epithelium never contains gonococci and that gonococci found in a vaginal discharge have come from the cervix or uterine canal. Beyond a doubt, however, when young women who have not borne children contract gonorrhea the vulva and vagina usually suffer. In older women and in women who have borne children the vaginal tissues are altered and the cells are not nearly so prone to infection; hence in such subjects the vagina usually or at least often escapes. The initial infection is in many cases in the cervical canal, in some in the vulva or urethra. No matter what part was first attacked, other parts usually become quickly involved in the acute process. The urethra is involved in almost every case. Chronic gonorrhea is prone to linger in the urethra, in the glands of Bartholin, in the cervical canal, or within the uterus or in the Fallopian tubes. The great danger of gonorrhea in the female is the development of ascending infection of the lining membrane of the uterus, which may reach the tubes, ovaries, and peritoneum.

When infection occurs during pregnancy or when pregnancy occurs during infection of the cervical or uterine canal, abortion may take place. Again, a pregnant woman may not abort, but may go on to term and the child may receive a conjunctival infection during delivery and rapidly develop purulent ophthalmia.

In some cases when pregnancy occurs during the existence of gonorrhea the disease seems to pass away, and yet the child gets conjunctival infection during delivery or the mother subsequently develops pus-tubes.

**Treatment.**—Place the patient in bed during the acute stage of the disease, give hot hip-baths, keep the bowels open by means of saline purgatives, insist on a fluid diet consisting chiefly of milk, and flush out the urethra by having the patient drink considerable quantities of water. The external genital organs should be sprayed with peroxid of hydrogen every two or three hours, and after spraying should be dried with absorbent cotton and dusted with equal parts of starch and powdered oxid of zinc, or with powdered stearate of zinc. Pads of cotton fixed in place by a bandage are used to catch the discharge. If urethritis exists in this stage, we may give alkalis, balsams, and astringent urethral injections.

When the acute symptoms have somewhat abated an attempt should be made to prevent ascending infection from the cervical canal. The mucous membrane of the canal may be curetted away or be destroyed by pure carbolic acid or nitrate of silver. A wiser plan is to paint the cervical canal daily with iodin or a 10 per cent. solution of argyrol, painting the vaginal portion of the cervix at the same time with the same drug. The vagina is irrigated twice a day with a warm solution of permanganate of potash (1 : 4000) and is lightly packed with iodoform gauze. When the vulva is particularly involved, treat that part by applying acetate of aluminum (2 per cent.) locally or paint the vulva with silver solution (40 gr. to 1 oz.). If the vulvovaginal gland suppurates, open it.

If vaginitis exists and continues in spite of the treatment suggested above, wash out the vagina every two hours, first with 1 pint of hot solution of bicarbonate of sodium, next with 1 pint of hot water, and finally with 1 pint of astringent solution (1 teaspoonful of lead acetate, 1 teaspoonful of zinc sulphate, 1 teaspoonful of alum, or 4 teaspoonfuls of tannin to 1 pint of hot water). As the attack subsides, use vaginal suppositories, each containing

5 gr. of tannic acid. In some cases apply solutions of silver nitrate (1 : 200) or of argyrol (10 per cent.), and insert tampons of ichthyol (8 per cent.) moistened with boroglycerid (Le Blonde).

In chronic cases of urethritis use strong solutions of silver nitrate and irrigate the urethra and bladder with silver nitrate (1 : 8000).

For *uterine gonorrhea* observe the same general management. Swab out the uterus with tincture of iodin or nitrate of silver and insert tampons of iodoform gauze.

**Gonorrhea in Children.**—**Male Children.**—This disease is not very common. When it affects children under twelve it is usually due to some abandoned and diseased female having brought the child's penis in contact with her sexual organs. It may result from introducing infected materials into the penis. The symptoms are similar to, but more acute than, those met with in an adult. The finding of the gonococci is clinical but not absolute legal proof of the existence of gonorrhea, and it is to be remembered that boys may suffer from catarrhal urethritis as a result of introducing irritants, from balanoposthitis, or from overacid urine. Legal proof is afforded by the growth of the suspected micro-organisms on artificial blood-serum.

The *treatment* consists in confinement to bed during the acute stage, bland drinks, light diet, etc. Circumcision is necessary if phimosis exists. When the acute symptoms subside, injections are used as in an adult.

**Female Children.**—Gonorrhea is more common in female children than in male children, and the vagina is involved as well as the vulva and urethra.

A female child may suffer from catarrhal inflammation of the vulva, as a result of the contact of foul urine, of feces, of the presence of seat-worms, or of neglect of bathing. In such a case the vagina and urethra escape. Involvement of the vagina and urethra strongly suggests gonorrhea. A recently born child or a young infant may acquire gonorrhea directly from a diseased mother, or indirectly, by pus upon linen, from the mother's fingers, etc. A diseased nurse may infect the baby. Older children who have ceased to nurse may get the disease from infected linen, bathtubs, etc., and may by these means infect child after child in an institution. Now and then the disease arises by a diseased man or woman deliberately bringing the child's private parts in contact with their own diseased organ.

The disease is acute: the urethra, vulva, and vagina are usually involved; the discharge is profuse, purulent, and often bloody. During the first day or two the discharge exhibits leukocytes but no gonococci, and the normal flora of the urethra disappear; later gonococci appear (Harmsen, "Zeits. f. Hyg. u. Infektionskr.," 1906, vol. iii). Microscopical examination of the discharge is absolutely necessary. Dry cover-slip preparations are made so as to obtain clap shreds from the discharge. An attempt should be made to obtain cultures. The gonococcus is very difficult to maintain in culture; it must be frequently transferred, and it grows best in an incubator at a temperature of 36° C. No attempt is made to grow it upon ordinary culture-media. The finger may be sterilized and punctured, blood thus obtained being smeared upon ordinary agar. Upon this composite material growth can be obtained. Animal blood-serum is not a good medium, but human blood-serum is (Lehmann and Neumann). Human blood-serum is obtained by opening a vein or from a fresh placenta.

Lehmann and Neumann ("Atlas and Principles of Bacteriology") find the following a satisfactory medium: Agar, containing 1 per cent. peptone and 5 per cent. glycerin, which has been liquefied and cooled to 50° C., is mixed "with one-half its volume of ascitic fluid or the fluid from ovarian cysts." Plate cultures and streak cultures should be made. This excessive care in proving the presence of the gonococcus is imperatively necessary in

female children because of the medicolegal questions which may arise in such a case and also because of the danger there is of the case infecting other persons.

Surgeons are apt to be doubtful about the diagnosis in many supposed cases of gonorrhea in female children. The clinical picture may simply be that of catarrhal vulvovaginitis, it may be that of gonorrhea. The finding of the gonococcus is regarded as conclusive from a clinical standpoint, but not from the legal point of view. Again, as Taylor points out, in some cases in which the clinical and microscopical evidence seem to prove the existence of gonorrhea no proof can be obtained that the condition is of venereal origin, and that in some cases in which everything indicates that the disease began as a catarrhal vulvovaginitis, a condition seemingly identical with gonorrhea has arisen. Obtaining a culture of gonococci is conclusive. The treatment consists in taking every care to prevent diffusion of the infection to others and to the patient's own eyes. She is put to bed, given frequent baths, and fed upon milk, etc. Irrigations of bicarbonate of sodium are employed, followed by protargol (1 : 5000, according to White and Martin). Later astringent injections are indicated.

*Treatment of Gonorrheal Arthritis and Endocarditis.*—In a communication in the "Jour. Amer. Med. Assoc.," Jan. 27, 1906, pages 261–263, Messrs. Rogers and Torrey described the method of preparation of an antigonococcic serum for the treatment of gonorrheal arthritis. This serum in its present form, as manufactured by a good chemical company, seems possibly to be of some value in the treatment of gonorrheal infections of joints, tendon-sheaths, and allied structures. Dr. Thomas C. Stellwagen, of the Genito-urinary Department of Jefferson Hospital, has used the material in a series of 26 cases of acute and chronic gonorrheal arthritis with encouraging results ("Therapeutic Gazette," April 16, 1910, page 248).

*Preparation of the Serum.*—To quote from Messrs. Rogers and Torrey's reports (Loc. cit.): "Rabbits were at first used exclusively in producing the serum. Although a very potent serum may be obtained from these animals, it was found that the serum itself is decidedly toxic for some individuals and may produce a rather alarming reaction. In order to obviate this serious objection we have experimented with goats and sheep. Similar objectionable properties, although less in degree, were found to be present in goat serum, but from sheep serum they seem to be entirely absent. Accordingly in later work only these animals have been used. They should be full grown, uncastrated males. In immunizing these animals it has been found advantageous to pursue the following plan: The first inoculation may consist of the twenty-four-hour surface growth from 18 square inches of solid culture-medium, emulsified in about 30 c.c. of physiologic saline soution, and heated for one-half hour at 65° C."

In Stellwagen's studies and use of the serum it was found that the best results were obtained by giving the injections as close to the area of infection as possible, and, further, to give them every day. The only untoward results noticed were now and then an eruption of wheals or urticaria accompanied by slight headache and itching, with a trivial rise of temperature. The acute cases generally showed marked improvement after about the seventh injection. The serum also seemed to be of use in other complications, such as orchitis, prostatitis, and epididymitis. Stellwagen maintains that the serum is a valuable adjunct in treatment, especially where the older standard remedies are slow to produce a result. The great claim for serum, however, has been in arthritis and other synovial infections, in which it seems to be distinctly valuable.

The serum must not be confused with the vaccine, which is decidedly uncertain. When the vaccines were used in the venereal clinic of the Jefferson Hospital they did not produce the happy results that other clinicians

have claimed for them. Schäfer's gonorrheal phylacogen possibly furnishes a useful method of treatment for metastatic gonorrheal infections.

**Stricture of the urethra,** or narrowing of the urethral caliber, is divided into *inflammatory, spasmodic,* and *organic.* The so-called *inflammatory* or *congestive* stricture is not a stricture, but is an inflammatory swelling of the mucous membrane.

*Spasmodic* stricture does not exist alone, but complicates organic stricture, a hyperesthetic urethra, or an inflamed bladder.

*Organic* stricture is a fibrous narrowing of the urethra, due, as a rule, to chronic gonorrheal inflammation or to traumatism. True organic stricture is very rare in children, but can occur. Abbe reported a case of impassable stricture in the deep urethra of a male child two and one-half years of age, due to urethral gonorrhea. There were also two strictures of the anterior urethra. External urethrotomy was performed. Traumatic strictures occur in the bulbous or membranous urethra, and are due generally to force applied to the perineum, the urethra being squeezed between the subpubic ligament and the vulnerating body. Strictures resulting from gonorrheal inflammation occur in the penile, bulbous, or membranous urethra. Stricture never forms in the prostatic urethra except as a result of traumatism. Recent non-traumatic strictures are soft and are easily distended. Old strictures and traumatic strictures are very dense. A resilient stricture is one which contracts quickly after dilatation. The nearer a stricture is to the meatus, the more fibrous it is.

A *congenital* stricture is congenital narrowness of a portion of the urethra, usually the portion near the meatus. The more fibrous a stricture is, the more it narrows the urethra and the less dilatable it will prove. A stricture may be annular (forming a ring around the urethra), tubular (surrounding the urethra for a considerable distance), or bridle (when a band crosses the urethra from wall to wall). A stricture of large caliber will admit an instrument larger than a No. 15 French sound. A stricture of small caliber will not admit a No. 15 French sound. An impermeable stricture will not admit the passage of any instrument. "Impermeable" is more or less a relative term. A stricture may be impermeable when an anesthetic is not used, and permeable when the patient is anesthetized, or may be impermeable to one surgeon, but permeable to another. Impermeability is often a temporary condition due to spasm or to inflammatory edema about an organic stricture.

**Symptoms and Results of Stricture.**—There is usually a history of repeated attacks of urethritis. A chronic discharge may exist, the amount of which is variable. There is a feeling of weight in the perineum and soreness of the back, and frequency of micturition is complained of. Hypochondriacal tendencies are usual. In a deep stricture there is difficulty in starting the stream in micturition. In most cases the stream is small, twisted, and forked. There is often interruption or "stammering" of the stream, and it dribbles long after the conclusion of the act, so that the penis must be "milked" before it is returned within the clothing. The urethra back of the stricture dilates, a pouch forms, drops of urine collect and decompose, and a chronic inflammation results in the mucous membrane or the parts adjacent, which inflammation may go on to ulceration or to periurethral abscess. A urinary fistula results from the opening externally of a periurethral abscess. Retention of urine may occur, not from actual obliteration of the tube by the growth of the stricture, but by closure of the lumen of the urethra by muscular spasm and by edematous swelling in the neighborhood of the stricture. Edematous swelling may be due to cold, wet, venereal excitement, the use of alcohol, overexertion, etc. Spasm of the muscles results, and contact of the urine increases the spasm, and spasm plus edema of the mucous membrane closes

the urethra. Spasm may exist in the urethra itself and in the muscles of the neck of the bladder, but is only a temporary condition. In old strictures the bladder is hypertrophied and often fasciculated, and is very liable to cystitis. The diagnosis of stricture and of its location is made by the use of exploratory bougies. In this examination the author follows to a great extent the plan of Ramon Guitéras,[1] which is as follows: Have the patient pass urine into two glasses. Examine the urine for clap-shreds. Cloudiness in the first glass shows that urethral discharge exists. Cloudiness in the second glass points to cystitis. The patient is placed recumbent with his shoulders elevated, and the urethra is washed out with warm salt solution or boric acid. Bulbous sounds are inserted, beginning with No. 15 French. If this passes with ease, take a larger size and note where strictures are situated by the catch on withdrawal. If No. 15 does not pass, use a smaller size. Remember that the posterior layer of the triangular ligament catches a bulbous instrument on withdrawal. If the meatus is too small to permit of exploration, divide it by a curved bistoury, cutting from within outward. After cutting the meatus bleeding is arrested with styptic cotton, and a piece of absorbent cotton is tucked into the cut. After each act of micturition the patient inserts a fresh bit of cotton, and after three days the urethral examination may be proceeded with.

**Treatment.**—A stricture of large caliber in the deep urethra requires gradual dilatation. A steel bougie is introduced every fifth day, the size being gradually increased. Never anoint a bougie with vaselin, as it may become a nucleus for a stone in the bladder; use liquid cosmolin, glycerin, synol soap, or lubrichondrin. Before passing an instrument the patient urinates and his urethra is washed out with salt solution or boric acid solution. Glans, meatus, and urethra are cleansed as directed on page 1345. The sound is rendered sterile by boiling before using. Gradual dilatation can be effected by the use of the dilator of Oberlander, the tube being distended to the extent of 3 mm. every fifth day. If after dilatation there is urethral spasm, pain, or very frequent micturition, suspend the treatment for a number of days and order each night a hot hip-bath and a dose of paregoric. During gradual dilatation the patient should not use alcohol, should refrain from sexual excitement, should avoid cold and damp, and should take internally capsules containing boric acid and salol. It is rarely necessary to dilate above No. 32 French. After the surgeon finishes treatment he teaches the patient to use an instrument and directs him to pass it once a month, because gradual dilatation rarely cures a stricture, and if dilatation is permanently abandoned contraction will probably occur. Strictures in the pendulous urethra, if soft, are treated by gradual dilatation; if fibrous and contractile, by internal urethrotomy. For fibrous stricture in or near the bulb external urethrotomy should be combined with internal division. External urethrotomy secures drainage, and the doing of it greatly lowers mortality. In performing internal urethrotomy prepare the patient carefully; for several days before the operation give salol and boric acid by the mouth, and wash out the bladder repeatedly with boric acid solution. Be thoroughly aseptic. Anesthetize the patient. Before cutting irrigate the urethra with warm normal salt solution, and after cutting irrigate again, pass a rubber catheter into the bladder and tie it in. These precautions will prevent urethral fever. In cutting, insert Gross's urethrotome (Fig. 913) back of the stricture, spring out the blade, cut the stricture on the roof of the urethra, close the blade, withdraw the instrument, and pass a full-sized bougie.

Stricture of the meatus requires incision by a knife and the use of a meatus bougie until healing is complete. Strictures of small caliber in front of the

[1] "Med. Record," Nov. 14, 1896.

membranous urethra require gradual dilatation and, if this fails, internal urethrotomy or divulsion. Internal urethrotomy can be performed with the urethrotome of Maisonneuve (Fig. 914) or the Otis-Wyeth instrument (Fig. 916). The instrument of Maisonneuve is shaped like a sound, has a groove upon its surface, and into this groove a shaft carrying a triangular knife can be inserted. The staff is screwed to a guide, the guide is carried into the bladder and the staff

Fig. 913.—S. W. Gross's exploratory urethrotome.

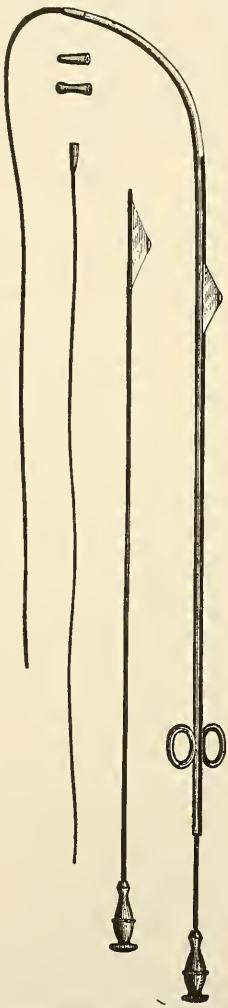


Fig. 914.—Maisonneuve's urethrotome.

Fig. 915.—Horwitz's modification of Maisonneuve's urethrotome.

follows it. The point of the staff is carried to the prostatic urethra and the guide curls up in the bladder. The penis is held upon the stretch, the blade is inserted and pushed down through the stricture. This instrument cuts the stricture, but not the healthy urethra. Stricture within 1 inch of the meatus is divided on the floor of the urethra. A deeper stricture is divided on the roof, except at or near the bulbomenbranous junction. In such a stricture the roof is divided by internal urethrotomy and the floor by external urethrotomy. In

using a urethrotome do not overdilate or cut deeply. To do so will cause deformity of the penis. For *divulsion* the patient is prepared as for internal urethrotomy. The dilator of Gross (Fig. 917), or divulsor of Sir Henry Thompson or of Gouley (Figs. 918, 919) is introduced, the blades are separated, the instrument is withdrawn, a large bougie is passed, and a catheter is tied in the bladder. Strictures of small caliber in the deep urethra are seldon permanently benefited by gradual dilatation. The best method is combined internal and external urethrotomy. In strictures of the deep urethra, if only a filiform bougie can be introduced, the bougie may be left in place, and in a day or two another can be slipped in beside it, until in a few days the channel becomes permeable to a metal bougie. A tunneled catheter can be slipped over the filiform bougie, both be withdrawn, and a metal bougie passed. A tunneled and grooved staff can be carried in over the bougie and external urethrotomy be performed. Thompson's dilator can be carried in over the filiform and the stricture be divulsed. What is known as *modified rapid dilatation* consists in first dilating as described above by the Thompson dilator, then introducing the powerful dilator of Gross (Fig. 917) and distending the urethra to the limit. This operation tears and lacerates rather than dilates and has been practically abandoned. In impassable stricture of the deep urethra perform external perineal urethrotomy without a guide (the operation of Wheelhouse).

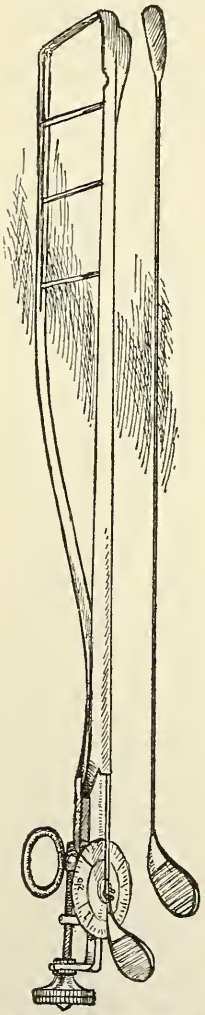


Fig. 916.—Otis's dilating urethrotome.

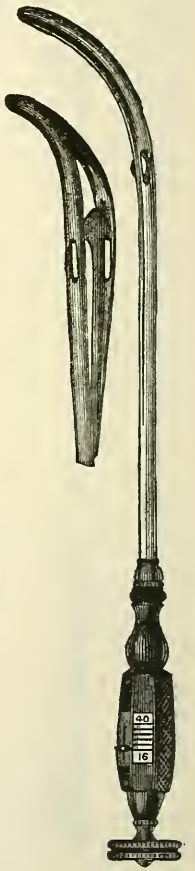



Fig. 917. — Gross's urethral dilator.

If a perineal fistula exists, dilate, divulse, or cut the stricture and retain a catheter in the bladder for forty-eight hours. After this period dilate the urethra every fourth or fifth day by a metal instrument. Every morning and evening draw the urine by a soft catheter, introduce boric acid solution into the bladder, remove the catheter, and let the man empty his bladder naturally. A part will flow from the fistula and a part from the meatus. Day by day the quantity which comes from the fistula lessens, and finally the abnormal opening heals.

**Urethral Fever.**—Any operation upon the urethra may be followed by a chill owing to shock (urethral shock), and this may be followed by a nervous fever. Urethral fever proper is sapremia following a urethral operation. The condition is due to absorption of toxic elements which may be in the urine, may have been in the urethra, or may have been introduced from without. It usually follows the first urinary act after operation. It begins with a violent chill and presents the characteristics of a septic fever. It is accompanied by a marked tendency to urinary suppression, and may eventuate

in septicemia or pyemia. Urethral fever can be prevented by rigid antisepsis. If this fever should arise, a catheter must be tied in the bladder, the bladder and urethra must be repeatedly irrigated with aseptic or antiseptic fluids, and the patient must be given urinary antiseptics and stimulants by the mouth.

**Urinary Fever.**—Sir Benjamin Brodie pointed out that the withdrawal of residual urine in a case of enlarged prostate may be followed by very serious symptoms. The condition is spoken of as urinary fever, and was said to be due to the sudden and complete emptying of a bladder which has become accustomed to retaining permanently a considerable quantity of urine. Modern studies prove that urinary fever is due to infection of the bladder and kidneys, and not simply to the sudden withdrawal of all of the urine from the bladder, although such a procedure leads to vesical con-

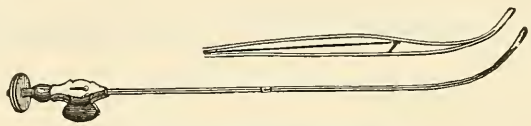


Fig. 918.—Thompson's divulsor.

gestion and probably favors infection. The bacteria most often found are pyogenic cocci, colon bacilli, and micro-organisms which cause putrefaction and decomposition of urea.

The condition does not arise promptly, suddenly, and violently, as does urethral fever, but begins rather insidiously after several days. Mr. C. Mansell Moullin[1] thus describes the condition:

"So far as the broader features are concerned, the symptoms that present themselves in these cases are remarkably uniform. They do not begin at once. Nearly always some few days elapse before there is anything to excite suspicion. Then the urine becomes cloudy, though it may still retain its acid reaction. A small quantity of albumin, more than can be accounted for by the amount of pus that is present, makes its appearance. Under the

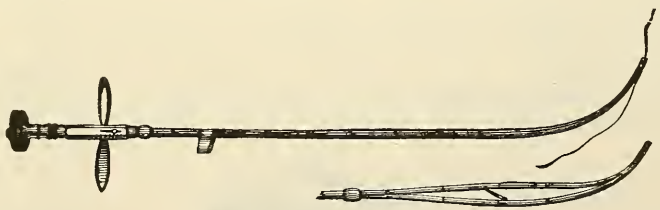


Fig. 919.—Gouley's divulsor.

microscope there are a few hyaline casts, perhaps a blood-corpuscle or two, numerous pus-corpuscles, and myriads of bacteria. The specific gravity is lower than it ought to be, and is lower than it was before the catheter was used. The total amount passed in the twenty-four hours may either increase until it is as much as 7 or 8 pints, or diminish until it scarcely reaches 20 oz. There is seldom any definite rigor, but there may be numerous slight chills. The pulse grows more rapid and feeble. The tongue becomes red and dry. There is complete anorexia. Delirium sets in at night, and in a considerable proportion of cases the symptoms rapidly grow worse and worse until, at the end of a few days, the patient sinks into a semicomatose condition from which he seldom rallies. Postmortem there are all the signs of recent acute cystitis and pyelonephritis. The mucous membrane lining the pelvis

[1] "Lancet," Sept. 10, 1898.

and calices of the kidney, the ureters, and the bladder is swollen and stained by old and recent hemorrhages, and here and there a thin layer of pus is adherent to it. The pelvis and the ureters are dilated, the apices of the pyramids are eaten away, the cortex is shrunken and hard, the capsule is adherent, and in places between the tubules are minute collections of pus differing in shape and outline according to the anatomical arrangement."

**Treatment.**—Aseptic catheterization is necessary if we would avoid urinary fever; and as the urethra contains some of the causative organisms, the

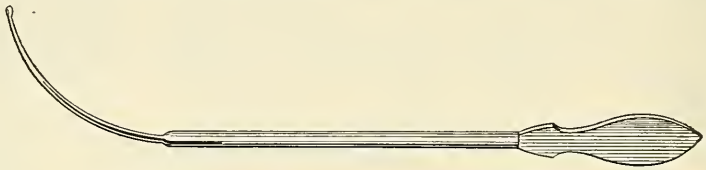


Fig. 920.—Syme's grooved staff.

prepuce, glans, and meatus should be washed with soap and water and irrigated with boric acid or permanganate of potassium solution, and the urethra be irrigated with boric acid solution or permanganate of potassium before the sterile catheter is introduced to draw the urine.

If urinary fever arises, it may be possible to control it by frequently irrigating the bladder with warm normal salt solution, solution of nitrate of silver (1 : 8000) or boric acid solution, and by administering stimulants,

Fig. 921.—Wheelhouse's staff.

diuretics, diaphoretics, saline cathartics, urotropin, salol or boric acid, quinin, and nutritious food. In severe cases perform suprapubic cystostomy for drainage.

**Perineal section** is external perineal urethrotomy. There are three methods—the operation of Syme, of Wheelhouse, and of Cock.

**Syme's Operation.**—This operation is employed if a stricture is very contractile, if dilatation fails to cure, or if urethral instrumentation invariably causes pronounced urethral fever. The patient is anesthetized, Syme's

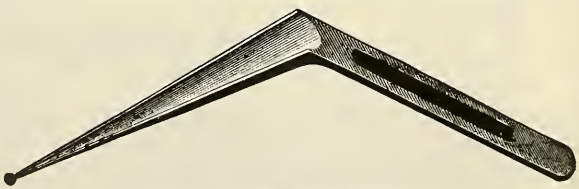


Fig. 922.—Teale's probe gorget.

staff (Fig. 920) is introduced, and the surgeon makes an incision in the midline of the perineum and exposes the staff just above the shoulder of the instrument. The knife is carried along the groove and divides the stricture. A catheter is passed into the bladder from the meatus and is retained for several days, and the wound is dressed antiseptically. After the catheter has been removed it must be used every six hours until the urine comes entirely by the meatus. During the rest of the patient's life a full-sized sound should be passed at monthly intervals.

**Wheelhouse's Operation.**—This operation is employed for the treament of impermeable stricture. Wheelhouse's staff (Fig. 921) is passed into the urethra until it blocks on the stricture. The perineum is incised down to the staff and in front of the stricture. The edges of the cut urethra are held apart by forceps, the surgeon seeks for the opening through the stricture, passes a fine probe through it, divides the stricture, carries into the bladder from the wound an instrument known as a probe gorget (Fig. 922) to dilate the canal and furnish a solid floor to facilitate the introduction of a catheter. With the gorget in place a metal catheter is carried from the meatus into the bladder. The gorget is removed and the catheter is tied in place. After three or four days the catheter is removed and is then passed frequently. The perineal wound is, of course, dressed antiseptically.

**Cock's Operation.**—This operation opens the urethra back of the stricture without the aid of a guide and relieves retention of urine. The surgeon introduces into the rectum the index-finger of the left hand, and the tip of the finger is rested upon the apex of the prostate gland. The surgeon incises the median line of the perineum, the back of the knife being toward the anus. When the point of the knife is felt to be near the finger the handle is lowered slightly, the blade is placed a little oblique, and the urethra is opened. A catheter is passed into the bladder from the wound and retained for a time, and the stricture is subsequently treated.

**Gibson's Operation for Impermeable Stricture** (C. L. Gibson, in "Med. Record," Aug. 6, 1910).—Open the posterior urethra by Cock's operation (see above). In most cases it is now possible to pass a small urethrotome from the urethral wound forward. If this can be done the deep stricture is at once divided. If it cannot be done a filiform is passed from the urethral wound out through the urinary meatus. This maneuver may require the aid of a special instrument (Gibson's retrograde filiform carrier). The straight staff of the Flührer urethrotome is threaded over the filiform bougie in front of the meatus. It is pushed back until it passes through the stricture, when the knife is inserted and "pushed home." This trivial cut permits the passage of a large urethrotome (for instance, Otis's). The stricture is now freely divided (up to No. 30 French). After division of the stricture a No. 30 French steel sound is carried from the meatus into the bladder. A tube to drain the bladder is introduced through the wound.

**Epispadias** is a congenital cleft in the corpora cavernosa, the roof of the urethra being completely or partly absent. In complete epispadias there are absence of the pubic arch and exstrophy of the bladder.

Partial epispadias may sometimes be remedied by a plastic operation.

**Hypospadias** is a congenital cleft on the floor of the urethra, the meatus opening on the floor at some point between the scrotum and the end of the glans penis, the channel in front of the meatus being a gutter and not a tube.

Hypospadias of the glans is the most common form. In this condition the urethra has no floor as it passes beneath the glans, the site of the urethra is indicated by a groove, and the foreskin is absent below. Partial hypospadias requires no treatment except possibly dilatation or incision of the meatus. People who suffer from it are very prone to develop chronic urethral inflammation. In hypospadias of the penis the ill-developed cord-like corpus spongiosum draws the penis to the scrotum. In this variety of the deformity the penis is very short.

In complete hypospadias the opening of the urethra is back of the scrotum in the perineum, the penis is dwarfed and bound down, and looks not unlike a clitoris, the scrotum is divided into two portions, a gap existing between them, and in many cases the testicles have not descended. Such individuals are occasionally mistaken for females. In the penile complete forms of

hypospadias a plastic operation should be performed between the eighth and tenth years of age. Such an operation, unfortunately, may fail. Hypospadias is rare in women, but it may occur. In such a case the urethra opens into the vagina. Figures 923–925 show the ingenious operation successfully practised by Carl Beck for penile hypospadias.

**Chancroid** (*soft chancre; the local venereal sore*) is an ulcer, usually of venereal origin. The name "chancroid" was introduced by Clerc, who believed that a soft sore resulted from inoculating a person already syphilitic with the products of a hard sore. He further held that when a soft sore arose the syphilitic poison lost its infective properties, and "could be transmitted as a soft sore to a healthy person, and not cause general infection."[1] The chancroidal ulcer is not connected with the syphilitic poison, but is developed by inoculation with the bacterium of Ducrey. Until recently it was believed that a chancroid was not produced by a special poison, but arose after in-

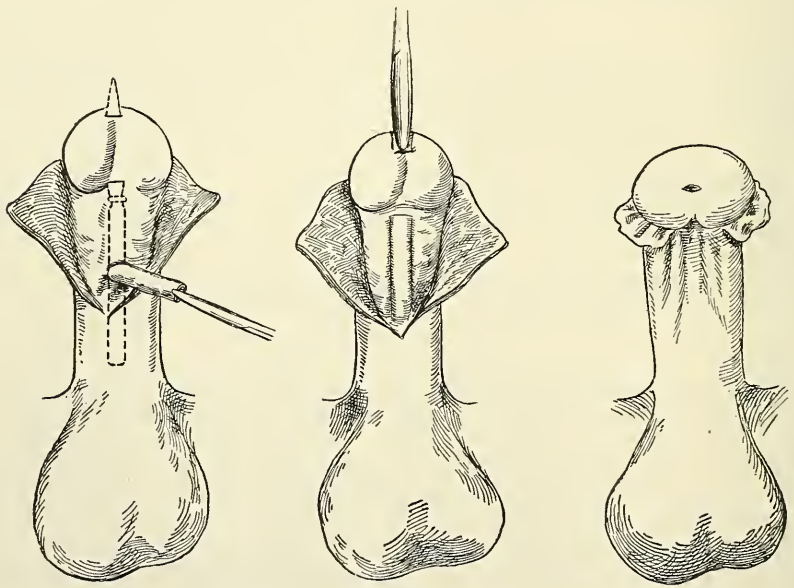


Fig. 923. Fig. 924. Fig. 925.

Figs. 923–925.—Beck's operation for hypospadias.

oculation with inflammatory products or irritating secretions. It seems to have been proved, however, by Krefting and Colombini that the organism discovered by Ducrey in 1889 is the real cause. This organism is grown on a medium of fresh blood and bouillon or in "unmixed human blood." (See Lincoln Davis, "Observations On the Distribution and Culture of the Chancroid Bacillus," "Report of Research Work," 1902–03; the Division of Surgery of the Medical School of Harvard University.) As a rule, chancroids are of venereal origin, and result from contact with other chancroids, pus, mucopus, or areas of ulceration. A chancroid appears soon after intercourse, usually within five days, always within ten days. It is first manifested by a pustule which ruptures and discloses an ulcer. This ulcer has sharply defined and undermined margins; it looks "punched out"; the base is gray and sloughy; the discharge is profuse, purulent, foul and auto-inoculable, and causes fresh chancroids by flowing over the parts. The area around a chan-

[1] "Syphilis," by Alfred Cooper.

croid is red and inflamed, and considerable pain is apt to be complained of. The original chancroid spreads and new sores appear. The edge of a chancroid is rarely indurated unless caustics have been used or there is mixed infection with syphilis. Inflammatory induration fades gradually into the tissues, but the induration of a hard chancre is sharply defined. Fournier says that a chancroid may have a hard base if the sore is located in the sulcus back of the glans, on a lip of the meatus, or on the lower border of the prepuce of a man with phimosis, or when the ulcer is inflamed. The surgeon should always ask if the sore has been cauterized and how it has been treated. When a chancroid after a time displays marked and sharply outlined induration it points to mixed infection with chancroidal and syphilitic organisms. Chancroids are not followed by constitutional symptoms, but are apt to be accompanied by painful inflammatory buboes which are prone to suppurate. In hospital practice about 30 per cent. of patients with chancroids develop buboes. The bubo may be either unilateral or bilateral. In the majority of cases the adenitis of chancroid is due to the absorption of toxins alone and the pus may be entirely free from bacteria. Cases have been reported in which non-indurated

Fig. 926.—Buttonhole perforation of the prepuce following phagedenic chancroid (Horwitz).

Fig. 927.—Buttonhole perforation of the prepuce following phagedenic chancroid (Horwitz).

sores were followed by syphilis. It is probable that a mixed infection existed, and that induration was overlooked, because a papular initial lesion was beneath the chancroidal ulcer. When inflammation in chancroids is high, a rapidly destructive ulceration known as *phagedena* may arise (Figs. 926 and 927), but this process is more common in syphilitic sores.

**Treatment.**—Ordinary cases of chancroid are treated by spraying with peroxid of hydrogen, drying with cotton, touching each sore first with pure carbolic acid and then with pure nitric acid, and dressing with black wash or dusting with iodoform or with calomel. Every few hours the patient soaks the penis in hot salt water (a teaspoonful of salt to a pint of water), sprays the sores with peroxid of hydrogen, dries with cotton, and dresses with black wash or dusts with iodoform or with calomel. As soon as granulation begins the sores should be dressed with 1 part of ointment of nitrate of mercury to 7 parts of cosmolin. Mild cases do well without cauterizing, peroxid of hydrogen being frequently used and a drying powder being employed. In chancroids with phimosis slit up the foreskin, smear the raw edges of the wound with pure carbolic acid, and treat the ulcers by cauterization. A regular circumcision usually fails because of infection of the stitch-holes. Phage-

dena requires the internal use of iron, quinin, and milk-punch, and the local use of powerful caustics (bromin or nitric acid or even the actual cautery). In some cases continuous antiseptic irrigation is valuable. When a bubo first begins, order rest, apply iodin or an ointment of belladonna or ichthyol, and make pressure by a spica bandage of the groin. Some surgeons advise the injection of 20 to 40 min. of a solution of carbolic acid (10 gr. to the ounce), but I have never seen any benefit from it. Some inject a 1 per cent. solution of bichlorid of mercury, but the proceeding causes intense pain. Welander recommends the injection of a 1 per cent. solution of benzoate of mercury. I have had no experience with this method. If the bubo persists though it does not suppurate, it should be completely excised. If pus forms, several methods of treatment are open to us: aspiration, injection with a solution of carbolic acid, squeezing out the acid and injecting 10 per cent. ointment of iodoform and glycerin, and sealing the opening with collodion (Scott Helms). Hayden makes a puncture, squeezes out the pus, washes out the cavity with peroxid of hydrogen, and then with corrosive sublimate solution, injects warm iodoform ointment, and dresses with cold, moist, corrosive sublimate gauze to set the ointment. Otis, Fontain, Perry, and others commend this plan. We have sometimes found it to succeed. If the above-mentioned plan fails, if it is not used, or if an ulcer or sinus exists, incise, curet, cauterize with pure carbolic acid, cut away hopelessly infiltrated skin, and pack the wound with iodoform gauze. In some cases it will be necessary to extirpate fragments of gland.

**Phimosis** is a condition of the prepuce that renders retraction over the glans impossible. It is usually congenital, but it may arise from inflammation. Congenital phimosis causes retention of sebaceous matter, which decomposes and lights up inflammation and the prepuce is apt to grow fast to the glans. Congenital phimosis may induce irritability of the bladder, incontinence of urine, prolapse of the rectum, and various nervous symptoms.

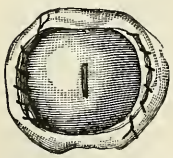


Fig. 928.—Circumcision completed (Esmarch and Kowalzig).

The **treatment** is *circumcision.* Asepticize the parts. Grasp the foreskin and the mucous membrane by two pairs of forceps, draw the prepuce forward, catch the skin (at the point it is desired to cut) horizontally between the arms of the handle of a pair of scissors, and cut off the redundant prepuce. Retrench the excess of mucous membrane by trimming with scissors $\frac{1}{4}$ inch from the glans, stitch the skin to the mucous membrane with catgut, and dress with sterile gauze (Fig. 928).

**Fracture of the penis,** which is a laceration of the cavernous bodies with extravasation of blood, occurs occasionally during coition.

The **treatment** consists of cold and bandaging to arrest bleeding, and in some cases incisions to let out clot.

**Gangrene of the penis** arises from phagedena, from tying constricting bands around the organ, from fracture with excessive hemorrhage, and from paraphimosis. If extensive, it requires amputation.

**Cancer of the penis** (Fig. 929) is commonest in persons with phimosis. In a limited epithelioma of the foreskin circumcision is performed and the glands of the groin are removed; if a well-developed cancer affects the glans, amputation of the penis is imperative and removal of the inguinal glands is absolutely necessary. Certain recent superficial cases without glandular involvement may apparently be cured by fulguration.

**Amputation of the Penis.**—Ricord advised cutting off the organ by a single stroke of the knife, making four slits in the mucous membrane of the urethra, and stitching each of these flaps to the skin. Treves splits the skin of the scrotum along the raphé, separates the halves of the scrotum

down to the corpus spongiosum, passes a metal catheter down to the triangular ligament, inserts a knife between the corpus spongiosum and the corpora cavernosa, withdraws the catheter, cuts the urethra across, detaches the urethra from the penis back to the triangular ligament, cuts around the root of the penis, divides the suspensory ligament, detaches each crus from the pubes, slits up the corpus spongiosum $\frac{1}{2}$ inch, stitches its edges to the rear end of the scrotal incision, introduces a drainage-tube, ligates the vessels, and sutures the wound.

**Seminal Vesiculitis.** — Inflammation of the seminal vesicles is due to the extension of a gonorrheal inflammation, to a pyogenic process, or to tuberculosis.

Fig. 929.—Cancer of penis (Horwitz).

**Acute vesiculitis** is made evident by frequent and painful micturition, pains in the anus, rectum, and perineum, and possibly the hip-joint, back, and thighs. Defecation and micturition are excessively painful. Persistent erections may take place, and in some cases bloody ejaculations occur. Rectal examination detects the enlarged and tender vesicles external to the lateral lobes of the prostate and on a higher level.

*Treatment.*—Abandon local urethral treatment, and treat the patient as for acute prostatitis.

**Chronic vesiculitis** may result from the acute form or may develop insidiously in an individual with gonorrhea. It is one of the possible causes of a chronic urethral discharge. The patient suffers from imperative and frequent demands to micturate, and he has a gleety discharge which becomes irregularly worse and better, but does not disappear. This chronic inflammation is believed to persist because of narrowing of the duct and consequent incomplete drainage of the vesicle. In chronic seminal vesiculitis there is usually sexual weakness, nocturnal emissions occur, and the semen may contain blood.

Fig. 930.—Dufaux's prostatic masseur.

*Treatment.*—Treat the posterior urethritis by ordinary methods. Use hot rectal enemata. Milk the ducts by Fuller's method once every seven days. During massage the patient's bladder should be full. He leans over a chair-back, the knees being straight and the body at a right angle to the thighs. The surgeon covers his finger with a rubber stall, anoints it with oil or synol soap, introduces it into the rectum, and makes pressure over the pubes with the fist of the other hand. The finger comes in contact with the lower half of the vesicle; it makes firm pressure for a moment and is then drawn slowly toward the duct. This stroking is repeated several times. The other vesicle is treated in the same manner. These maneuvers

empty the vesicles and hasten the resolution of inflammation. In many cases the finger reaches only the very lowest part of the vesicle. In such a case practice massage by means of Dufaux's rubber masseur (Fig. 930). After the completion of the stripping the patient should micturate, and the bladder and urethra should be irrigated.

**Tuberculosis of the Seminal Vesicles.**—Primary tuberculosis is very unusual. As a rule, there is evidence of antecedent tuberculosis of the testicle or prostate gland. About 50 per cent. of the cases occur in individuals under forty years of age. The diseased vesicle is at first nodular and indurated, but later undergoes caseation and softening. Finally the disease passes through the capsule and invades adjacent structures. Dreyer collected 36 cases and found that in 34 of them the lungs were involved.

Tuberculous vesiculitis may be unilateral or bilateral. In unilateral tuberculous epididymitis the corresponding vesicle is apt to become diseased. In bilateral disease of the testicles both vesicles are liable to become victims. Peritoneal tuberculosis may follow tuberculous vesiculitis. In very unusual cases spontaneous cure is obtained by fibrous-tissue formation. On palpation a tuberculous vesicle is found to contain here and there hard and but slightly tender nodules.

**Treatment.**—If tuberculous epididymitis is followed by tuberculous vesiculitis, it is justifiable to remove the vesicle after removing the epididymis or testicle, provided the prostate and other parts of the genito-urinary tract are free from disease and there is no distant lesion of tuberculosis. If both testicles or epididymes are removed, both vesicles can be extirpated. If a vesicle or both vesicles suffer from primary tuberculosis, operation is advised by some surgeons. Reported cases, however, do not seem to favor operation.

Kraske, Schede, and Rydygier have removed the vesicles after preliminary resection of the sacrum. Zuckerkandl, Diettl, and Schede have employed the perineal route. Villeneuve reached them by way of the inguinal region. The curved perineal incision of Zuckerkandl is the method usually preferred. H. H. Young makes a suprapubic incision, strips the peritoneum from the bladder, and reaches the vesicles from behind. He calls it the suprapubic-retrocystic-extraperitoneal method (H. H. Young, in "Annals of Surgery," Nov., 1901).

**Acute Prostatitis.**—Acute inflammation of the prostate gland may be caused by inflammation in adjacent structures, the use of instruments or irritant applications in the deep urethra, injury by a passing or impacted calculus, various infectious diseases, a stricture of the urethra, but particularly by gonorrhea. The gland enlarges greatly, the prostatic fluid exudes mixed with blood and pus, and the gland-ducts become distended with pus. A distinct abscess may form. The orifices of the ejaculatory ducts become distended and filled with pus, and the seminal vesicles or epididymes may also suffer. An abscess is liable to form in the cellular tissue outside of the prostate.

**Symptoms.**—There is a feeling of weight, fulness, or soreness in the perineum; a persistent pain at the neck of the bladder; frequent micturition, pain being present and becoming most severe as the last drops are voided; perineal tenderness; painful defecation; and bulging of the anal mucous membrane. If a finger is introduced into the rectum, it causes severe pain and palpates the enlarged and tender gland, unless the outlines are destroyed by periprostatitis, in which case there will be felt a large, boggy, tender mass. (See Henry Morris on "Injuries and Diseases of the Genital and Urinary Organs.") These symptoms are accompanied by distinct elevation of temperature. The inflammation may abate without suppuration, but, as a rule, pus forms, the temperature becomes characteristic, the pain becomes pulsatile, micturition

causes agony, the inflammatory mass is felt per rectum to be softening, and sometimes the swollen perineum becomes dusky red. Retention of urine is almost certain to occur. The abscess may rupture into the urethra or the rectum, or may diffuse in the periprostatic cellular tissue and subsequently may open in the perineum. Spontaneous evacuation may be followed by recovery or by the development of annoying or dangerous complications.

**Treatment.**—Keep a hot-water bag on the perineum and three or four times a day use rectal injections of hot water. Place the patient on a milk-diet. Leech the perineum. Give suppositories of opium and belladonna, and also suppositories of ichthyol, and administer urotropin by the mouth. Retention of urine is relieved by a soft catheter. When pus forms it may be possible in some cases to rupture the abscess into the urethra by the passage of a steel sound. If this can be accomplished it will be fortunate. Occasionally a specialist may succeed in opening the abscess through an operating cystoscope. Most cases require to be cut externally. What is known as the *boutonnière operation* is the method of choice. The patient is placed in the extreme lithotomy position. The index-finger of the left hand is introduced into the rectum and carried to the apex of the prostate. A straight bistoury is entered to the side of the median raphé and carried into the abscess cavity, and the opening is dilated by forceps. The urethra is not to be opened.

**Chronic prostatitis** may arise from stricture, venereal excess, chronic cystitis, or stone in the bladder, but gonorrhea is the common cause. The prostate is usually, but not always, enlarged, is somewhat softened, and the ducts contain pus and blood.

**Symptoms.**—There is usually a mucopurulent discharge or fluid can be obtained by massage of the prostate. There is a feeling of weight and fulness in the perineum, there is increased frequency of micturition, and the prostate is very sensitive to digital pressure. The patients are neurotic, frequently suffer from nocturnal emissions, and have but feeble power of erection. The prostatic urethra is extremely hyperesthetic. All the symptoms are aggravated by worry, sexual excitement, or violent exercise. An abscess may form and rupture into the urethra.

**Treatment.**—Tonics and nutritious food are essential. Intravesical irrigations with nitrate of silver solution (1 : 8000) do good. Massage of the prostate is useful. Some cases are benefited by touching the posterior urethra through a urethroscope tube with nitrate of silver (3 gr. to the ounce) or by injecting by means of Ultzman's syringe a few drops of silver nitrate solution (5 gr. to the ounce). Rectal suppositories of ichthyol may be ordered. Blistering the perineum at intervals may prove of service. At intervals of three or four days a full-sized cold steel sound should be gently introduced. If an abscess forms, open it through the perineum.

**Prostatorrhea.**—Just as overaction of the glands of the urethra constitutes urethrorrhea, so overaction of the glandular apparatus of the prostate gland constitutes prostatorrhea. Prostatorrhea is not inflammatory, although the prostate and posterior urethra are often congested, and the latter region is usually hyperesthetic. In some cases urethrorrhea exists with prostatorrhea. Prostatorrhea is produced by sexual excess, masturbation, ungratified sexual desire, riding a bicycle with an improper seat, and sometimes by riding horseback. The condition is usually accompanied by marked neurasthenia, and may be associated with spermatorrhea and impotence.

The patient notices a milky or gray discharge after straining at stool (*defecation spermatorrhea*), after violent exercise, sexual excitement, or a bicycle ride. The discharge also gathers in the urethra during sleep. Examination of the discharge shows it to be prostatic fluid, although spermatozoids are sometimes found. It is not purulent and contains amyloid cor-

puscles. The meatus is not glued up in the morning and the linen is very slightly stained. The urine is clear and contains small comma-shaped hooks. Sexual excitement and alcohol do not appreciably aggravate the condition. The bladder is irritable, and there is frequency of micturition and often some pain in the head of the penis at the termination of the act. Nocturnal emissions may occur.

**Treatment.**—The patient should correct bad habits. If there is urethral hyperesthesia or prostatic congestion, irrigate the bladder and urethra once a day with a solution of silver nitrate (1 : 4000), and every fourth or fifth day introduce a cold sound. In some cases the occasional instillation into the prostatic urethra of a few drops of a 1 per cent. solution of nitrate of silver does good.

For the irritable bladder give hot hip-baths at night. The following prescription is of service: 15 gr. of bromid of potassium, ½ dram of tincture of hyoscyamus in ½ oz. of cinnamon-water, three times a day. Hot enemata are of service.

After the hyperesthesia of the urethra has abated and nocturnal emissions have ceased, the neurasthenia is treated by cold sponging of the body night and morning, the continued use, at intervals of several days, of a large-sized cold sound, irrigation every second or third day with silver nitrate (1 : 4000), and the administration of strychnin and other tonics.

**Hypertrophy of the Prostate Gland.**—It was pointed out by Morgagni that in old men difficulty of micturition is due to obstruction by an enlarged prostate gland. Enlargement of the prostate gland may be brought about by different forms of growth. It is, as a general thing, a senile change, occurring only after the age of fifty, and being most likely to arise after the attainment of sixty years. It is very rare for enlargement of the prostate to cause symptoms long before the age of fifty or to begin after the age of seventy. Sir Henry Thompson maintained that 34 per cent. of men over sixty have prostatic hypertrophy, but that only half of them have troublesome symptoms. According to Freyer, 33 per cent. of all men past fifty-five years of age present some enlargement of the prostate.

There are some who oppose the view that prostatic enlargement is essentially a senile change. For instance, Dr. L. Bolton Bangs ("Jour. of Dermatol. and Gen.-urin. Dis.," March, 1901) maintains that the change is not senile; that it really begins early in life, but that its effects do not become manifest until during or after middle age. Lydston asserts that it begins during the third decade of life, but the gland does not attain sufficient size to cause symptoms till beyond middle life. Socin and Burckhardt, as a result of 300 postmortem examinations, reached the following conclusions: Between the ages of thirty-six and forty the gland is hypertrophied in 13 per cent. of cases, between forty and fifty in 25 per cent., between fifty and sixty in 31 per cent., between sixty and seventy in 56 per cent., between seventy and eighty in 50 per cent., and between eighty and ninety in 54 per cent. Undoubtedly, the enlargement begins long before it occasions sufficient obstruction to induce symptoms, and the growth progresses very slowly. Guyon and the French school in general maintain that hypertrophy of the prostate gland is always the result of arteriosclerosis, affecting not only the prostate, but also the entire urinary tract. The hypertrophy that ensues affects the bladder walls notably, as well as the prostate, because of distinct growth. Caspar has apparently demonstrated that Guyon's view is not correct. He has shown that in many cases there is no sclerosis of the prostatic arteries, and that frequently there are no sclerotic changes in other portions of the urinary tract. Another important point made by Caspar is that arteriosclerosis tends to cause degeneration, and not hypertrophy.

Some think that sexual excess is a cause of prostatic enlargement; some

assert that antecedent gonorrhea is the cause, but it seems very improbable that either is causal. Belfield blames altered testicular secretion; Hawley believes the cause to be altered prostatic secretion and the "chemical action of pathological proteins resulting from irregular metabolism or derived from disintegration of the secretion, or in the usual action of tissue enzymes" ("Annals of Surgery," Nov., 1903).

In the hypertrophied prostate there is an excessive production of fibrous tissue and of ill-formed glandular tissue, the mass constituting a fibro-adenoma. Fibro-adenoma is the common cause of enlargement (W. Bruce Clarke). Typical adenoma, according to Albarran and Hallé, is found in 14 per cent. of the cases ("Annales des Maladies des Organes Génito-Urinaires," Feb. and March, 1900). Again, in not a few prostates there is no real enlargement, but there is an indurated fibrous mass producing obstruction. Albarran and Hallé (Ibid., 1898, vol. xvi) point out that in an enlargement of the prostate different elements may usually be recognized: soft hypertrophy of the gland; indurated enlargement of the glandular elements; fibrous enlargement; circumscribed tumor-masses; distinct fibromata or myomata; or adenofibromyomata. The real cause of the various forms of prostatic enlargement is not known. Nearly 10 per cent. of cases are cancerous (Oraison), and adenoma is apt to be transformed into cancer.

All the lobes may be enlarged equally; all may be enlarged unequally; the enlarged gland may surround the prostatic urethra like a horse-collar; or one lobe only may be enlarged. Symmetrical enlargement of the entire gland is not so apt to produce symptoms as is non-symmetrical enlargement. In some cases the chief enlargement is into the bladder; in others, into the urethra. An enlarged prostate frequently shows a circular groove about it, due to the constriction exerted by the rectovesical fascia at the vesical neck.

The bridge of prostate which joins the two lateral lobes behind the urethra is known as the *lobe of Home* or the *middle lobe*, and a comparatively trivial enlargement of the middle lobe may cause obstruction. Prostatic hypertrophy causes narrowing and lengthening of the urethra, and gives this tube a tortuous course. The opening of the urethra into the bladder is usually pushed to a higher level, and there forms behind it a pouch in which urine collects. The urine that gathers in this pouch is known as *residual urine*. It cannot be voluntarily expelled. It may, therefore, collect in large quantity, and it is likely to decompose, producing cystitis. Residual urine strongly favors calculus formation. The mechanical resistance to the expulsion of the urine causes congestion of the neck of the bladder and the posterior urethra and also hypertrophy of the muscles of the bladder. In consequence of the hypertrophy the bladder enlarges, thickens, and becomes fasciculated. When this takes place, micturition becomes very difficult and sometimes impossible. Enlargement of the middle lobe inevitably blocks the flow of urine and causes great distention of the bladder. In hypertrophy of the prostate gland the ureters and the renal pelves and calyces may distend and surgical kidney may develop.

It is useful to divide persons with prostatic hypertrophy into three groups: (1) those in whom there is no obstruction or in whom the urinary symptoms are very trivial; (2) those in whom there are residual urine and disturbances of urinary function, who depend upon the catheter for relief, but who do very well by this method; and (3) those that suffer a complete breakdown during the period in which the catheter is depended upon (Orville Horwitz, in "Phila. Med. Jour.," Nov. 16, 1901).

**Symptoms.**—In 90 per cent. of the cases there is very trivial inconvenience, the patient merely being annoyed somewhat by episodes of nocturnal frequency of micturition. The stream of urine is slow to start and falls feebly

from the end of the penis. In some cases there is interruption of the stream (stammering). The last drops fall entirely without control. If the patient becomes sexually excited, chilled, or worried, or indulges inordinately in the pleasures of the table or in wine, beer, or alcoholic liquors, nocturnal frequency of micturition becomes for a short time most harassing. In 10 per cent. of all cases the bladder cannot be emptied entirely, and residual urine collects. Frequency of micturition comes on, particularly at night; the patient has to get up often; the bladder never feels empty; and cystitis is apt to arise. The urine, at first acid and clear, becomes neutral and cloudy, and finally ammoniacal and turbid, and contains bacteria, mucopus, precipitates of phosphates, and blood. Above the pubes there is aching pain, soon spreading to the perineum, which pain is increased when the bladder is distended and during micturition. The rectum becomes irritable, and piles form or prolapse of the mucous membrane occurs, because of straining in micturition. Attacks of retention of urine may occur. In about one-third of all cases we can make a diagnosis by rectal palpation. In enlargement of the middle lobe alone or in pure intravesical enlargement rectal touch will fail to make the diagnosis and the cystoscope must be relied upon. The bladder becomes thin and distended, or hypertrophied, rigid, and fasciculated. In rare cases true incontinence is caused by the median lobe growing toward the neck of the bladder and preventing closure. The health breaks down because of pain, restless nights, indigestion, and disorder of the bowels. The kidneys may become involved (inflammation of the pelves or calices, or surgical kidney) and suppression may occur. Septic fever may arise. Calculi may form in the bladder. Death is due to exhaustion, suppression of urine, or septic cystitis. A foul catheter is the usual cause of septic cystitis, but microorganisms sometimes enter by passing along the urethral mucous membrane.

A patient should be examined by rectal touch, by a sound, and by a cystoscope, if possible; the amount of residual urine must be determined, and the condition of the urine is to be carefully studied. The presence or absence of stone should always be determined. After an examination by instruments the patient must remain in bed for twenty-four hours.

**Treatment.**—There is no known method of preventing prostatic hypertrophy. Many cases of enlargement are treated by regular catheterization, and if this is conducted with careful cleanliness, and if the patient rigidly adheres to hygienic rules, he may be kept comfortable in this way for a considerable time. When a man must depend upon a catheter to empty his bladder he is said to be in *catheter life*. Alexander has formulated several sound rules as to when catheterization is the proper treatment. He says, if the patient is intelligent and dextrous, if cystitis is not severe, if the amount of residual urine is not very large, if obstruction is not great, if the bladder retains considerable expulsive power, and if catheterization is easy and painless, we are justified in relying upon this simple plan of treatment. Prevent cystitis by emptying the bladder each evening with a coudé catheter. If there is trouble in passing the catheter, strengthen the instrument by inserting a filiform bougie as a stylet. It is very seldom that a metal instrument is required, but if it is necessary, a catheter with a large curve is employed. If a soft or semisolid instrument can be passed, teach the patient how to clean it, how to use it, and how to keep it, but never permit the patient to use a metal instrument himself. A dirty instrument may cause fatal infection. It is true that some people use dirty instruments for long periods without trouble, but in most cases there will be trouble if it is attempted. It is absolutely necessary to use only perfectly aseptic instruments. Metal instruments are sterilized by boiling in water. Rubber catheters can be cleansed by washing with soap and running water, wrapping in gauze, and boiling. After

sterilization the instruments are kept ready for use in a glass cylinder which contains calcium chlorid.[1] The cleansing of catheters is discussed on page 1311. If there are 3 oz. of residual urine, use the catheter only at night. If there are 6 oz., use it night and morning. If there are more than 6 oz. of residual urine, add one more catheterization a day for every additional 2 oz. present until the catheter is used six times in the twenty-four hours. It should never be used oftener than this. Gradual dilatation with steel sounds is of benefit, but forcible dilatation is not advisable. The sound may be passed once a week. Tell the patient to avoid violent exercise, cold, damp, sexual excitement, and the use of alcoholic liquors; prevent constipation and indigestion, and direct him to drink milk and plenty of water. A hot hip-bath at night adds to his comfort. Hot enemata are of value. If a large quantity of residual urine exists, or if cystitis begins, wash out the bladder daily with boric acid solution, or normal salt solution, or nitrate of silver (from 1 : 10,000 to 1 : 4000), and give urotropin or salol and boric acid by the mouth (see Cystitis, page 1324). In some severe cases, if a large-sized rubber catheter be tied in the bladder for a few days, great relief is obtained. Retention of urine can usually be relieved by the introduction of a coudé catheter strengthened with a whalebone. In exceptional cases a silver instrument with a prostatic curve must be employed or aspiration must be practised. Many cases occurring among well-to-do people can be kept comfortable by catheterization. Some surgeons still think that only when this fails should an operation be performed. Unfortunately, sooner or later a man who regularly relies upon the catheter will develop cystitis. A poor man cannot give the necessary time and attention to make catheter life safe and operation must be thought of in him sooner than in others. If the symptoms grow constantly worse, if the suffering becomes severe, if the patient cannot urinate without the use of an instrument, if catheterization is painful or impossible, if the patient is too careless or ignorant to trust with a catheter, if only a catheter of very small size can be introduced, if attacks of obstinate retention occur, if there is persistent or recurring cystitis or hematuria, if there are signs of beginning infection of the kidney, if the residual urine gradually increases in amount, operation is called for. Do not postpone operation until the patient becomes really ill. Give palliative measures a reasonable trial, and if they fail, operate. Before determining upon any operation make a cystoscopic examination. This is particularly valuable before a Bottini operation and before a perineal operation. It shows us the condition of the bladder; the nature, size, and situation of the enlargement, the median lobe if present, and a calculus if one exists. This examination may determine the form of operation desirable. Prostatectomy is not to be regarded as a trivial affair certain to result in cure. It is a grave procedure, with a considerable mortality, which may be attended by disastrous complications and from which unfortunate consequences may arise. I agree with James E. Moore that "it is altogether too grave an operation to be resorted to as a routine treatment for every enlarged prostate, and is applicable only to properly selected cases." The operation is contra-indicated if there is advanced disease of the kidneys, and if it is performed in such a case, fatal uremia is to be expected. Age is not in itself a contra-indication if the kidneys and cardiovascular system are sound. An occasional sequel of prostatectomy is incontinence of urine due to injury of the neck of the bladder or to the nerves of the part. A possible sequel is sterility, but most of the subjects have been rendered practically sterile before operation by age.

In the majority of cases in which palliation fails the operative indication is to remove an obstructing mass and depress the level of the opening from the bladder into the prostatic urethra, so that the prostatic pouch is abol-

[1] R. W. Frank, in "Berliner klin. Woch.," No. 44, 1895.

ished and the bladder can be thoroughly drained. The surgeon chooses between prostatotomy and prostatectomy. Prostatotomy is usually performed by the galvanocautery (Bottini's operation). Prostatectomy may be suprapubic or perineal, and the latter may be by enucleation without the aid of sight (as in the operations of Nicoll and Alexander) or by open dissection (as in Young's operation). No one routine plan is suitable for all cases. The patient should be studied, and the operation chosen which is safest and best for that individual case. The surgeon who only uses one method must wrong many patients, and he retains consistency at the expense of humanity. It was formerly believed that any operation of total prostatectomy must of necessity produce impotence. This we now know need not be the case. The suprapubic operation is proabaly less likely to be followed by this than is the perineal, as it usually spares the ejaculatory ducts. Young's perineal operation spares the ejaculatory ducts. Destruction of the ejaculatory ducts certainly produces sterility and may, but does not of necessity, produce impotence. Willy Meyer ("Med. Record.," Oct. 7, 1905) points out that impotence may be caused by damaging important nerves or blood-vessels in advancing through the perineum, and also by the operation producing relaxation of the verumontanum and prostatic urethra, parts necessary in the reflex for erection.

The *perineal operation* is as safe as the suprapubic or safer, and can be rapidly performed. It is the desirable route when the gland can be palpated per rectum, and does not mount high up when we are dealing with the early stages of soft hypertrophy (Willy Meyer, Ibid.) and when prolonged drainage is required. According to Francis S. Watson ("Annals of Surgery," June, 1904), the mortality in 203 cases was only 2.9 per cent. In 563 cases of removal through the perineum by dissection the mortality was 5.5 per cent. Young's cases in this group number 150 and his mortality was 4.6 per cent.; the mortality of Hartman was 9 per cent.; of Albarran, 4 per cent., and of Murphy, 3.9 per cent. (Schachner, in "Annals of Surgery," August, 1908). In 190 cases of blind enucleation through the perineum the mortality was 4.7 per cent. (Ibid.).

After the performance of the perineal operation the drainage is at the lowest part of the bladder. In a perineal operation every effort should be made to do as little damage as possible to the urethra. If we destroy the entire prostatic urethra the operation becomes easy and rapid and Nature rapidly repairs it, but a traumatic stricture may follow and may make the patient's condition worse than at first. As Moore says, we must destroy a portion of the floor of the urethra, but we can preserve the roof and the side walls. Another point in the perineal operation is to avoid injuring the rectum. A tear may enter the rectum, or, even if the gut was not torn, sloughing of the rectum resulting in recto-urethral fistula may occur. The rectum may be opened because the surgeon fails to stick close to the urethra in his dissection, and sloughing may be due to an injudicious use of the retractors. If the rectum is opened, it should be at once sutured with catgut. In most cases it takes about three weeks for the wound in the perineum to heal, and in some few cases a perineal urinary fistula is established. Urinary incontinence may follow this operation. By simply incising the prostate gland the floor of the urethra may be lowered to the level of the floor of the bladder. Simple incision of the prostate in this manner, or by Bottini's method, is known as *prostatotomy*. The mortality is small and the relief is often great. Prostatotomy is performed on old and exhausted patients with damaged kidneys. A large tube should be worn during the healing of the wound.

The *suprapubic operation* is easier than the perineal; it is less safe; it gives excellent results if temporary drainage only is needed. According to Watson ("Annals of Surgery," June, 1904), the mortality in 69 cases was 8.6 per cent. P. J. Freyer reports 600 cases varying in age from forty-eight to

eighty-nine years. There were 47 between the ages of eighty and eighty-nine, and 7 were seventy-nine; most of the cases had been entirely dependent on the catheter for periods up to twenty-four years. "Nearly all were in broken health and many apparently dying before operation. Few were free from one or more grave complications, such as cystitis, stone in the bladder, pyelitis, kidney disease, diabetes, heart disease, chronic bronchitis, paralysis, hernia; and in a few instances there was malignant disease of some other organ than the prostate" ("Archives Internationales de Chirurgie," vol. iv, Fascic. 4, 1909). In these 600 cases there were 37 deaths in periods of from six hours to thirty-seven days after operation, a mortality of 6.15 per cent. Suprapubic prostatectomy is indicated in rather young subjects in whom we greatly fear impotence; in cases in which the gland is placed high; in cases in which the gland is not palpable per rectum, but is causing serious symptoms, and in which the hypertrophy is recognized by the cystoscope (Meyer); in cases in which there is a middle lobe; in cases in which cancer exists, or in which calculus complicates the case. It is the most useful operation when the gland is very large and intravesical. It is not a suitable method if the bladder is markedly contracted or if the belly walls are very thick. If prolonged drainage (short of permanent drainage) is required, as it is sure to be in cases with advanced cystitis, the opening is better placed in the perineal operation. If when a suprapubic operation has been performed it is found that prolonged drainage is indicated, a siphon drain (Fig. 886) may be used. If permanent drainage is required in a case, the suprapubic method is the best. After a suprapubic cystostomy has been performed for drainage, the opening may be kept permanently patent by the retention of a tube (Hunter McGuire's operation). It is only in very advanced cases or in cancer that permanent suprapubic drainage is employed. After making a suprapubic incision the floor of the urethra cannot be brought level with the floor of the bladder by a simple incision of the prostate through this incision; it can be brought level only by the performance of prostatectomy. In the suprapubic operation the structures divided are less important, the hemorrhage is less, and the drainage is less conveniently placed but better than in the perineal operation. Suprapubic prostatectomy inflicts injury upon the bladder, it may gravely damage the sphincter of the bladder, and is sometimes followed by incontinence or by inability to expel urine (John B. Murphy, "Jour. Amer. Med. Assoc.," March 29, 1902), but disturbance of control is less common than after the perineal operation. The bladder wall may be seriously torn, and if such a wound should be inflicted, it ought to be sutured with catgut. In this operation if the bladder is contracted, the surgeon must exercise great care to avoid injuring the peritoneum. The ureters may be damaged and subsequently become obstructed from contraction. If death occurs after prostatectomy it is due to shock, uremia, sepsis, or postoperative complications, usually pulmonary. Schachner ("Annals of Surgery," August, 1908) uses Watson's figures to present the special dangers of each operation. The figures give the percentage of the deaths due to shock, uremia, hemorrhage, and pulmonary complications. The table is as follows:

| | Per cent. | |
|---|---|---|
| Bottini | 2.7 | Uremia (or renal insufficiency). |
| Perineal operations | 35.0 | |
| Suprapubic operations | 34.0 | |
| Bottini | 52.0 | Sepsis. |
| Perineal operations | 17.8 | |
| Suprapubic operations | 8.6 | |
| Bottini | 5.0 | Shock. |
| Perineal operations | 21.4 | |
| Suprapubic operations | 30.0 | |
| Bottini | 8.0 | Postoperative pulmonary complications. |
| Perineal operations | 17.8 | |
| Suprapubic operations | 22.0 | |

At the International Urological Congress held in London in 1911 Young presented the records of 484 cases of perineal prostatectomy. The mortality in simple hypertrophy (450 cases) was 3.77 per cent.; 34 cases were cancerous, and of these 2 died. Zuckerkandl's mortality from perineal operations was 9.5 per cent., from suprapubic operations, 18.7 per cent. ("Ann. des Mal. des Org. Gén.-Urin.," 1911, ii). Freyer ("Amer. Jour. of Dermatology and Genito-Urinary Diseases," 1912) reports 1000 cases of suprapubic prostatectomy since 1901. The average age of these cases was sixty-nine. The youngest patient was forty-nine, the oldest was ninety. There were 55 deaths (a mortality of 5.5 per cent.). Freyer's mortality in his first 100 cases was 10 per cent., in his last 100 cases it was 3 per cent. This proves the great value of experience. The less training a man has had, the higher will be his mortality. Prostatectomy is distinctly not an operation for a surgical juvenile.

**Suprapubic Prostatectomy.—Freyer's Method.**—The bladder is washed out through a catheter with warm boric acid solution and is then filled with the solution. The nozzle of a large syringe filled with fluid is inserted into the end of the catheter. This keeps the bladder fluid from running out and enables the surgeon to quickly distend the viscus more if occasion shall arise. The bladder is exposed and opened in the midline and in an area free from veins (see Suprapubic Cystotomy, page 1337). The incision is vertical, is made toward the symphysis, and is about 1 inch in length (it can be enlarged later if necessary). If any calculi are found they are at once removed. A finger is placed in the rectum to raise up the prostate and keep it steady. The finger of the other hand is introduced into the bladder, and by means of the finger-nail the mucous membrane is scratched through over "the most prominent portion of one lateral lobe, or over the so-called middle lobe, if there be but one prominence" (Freyer, in "Archiv. Internat. de Chir.," Fascic. 4, 1909). This portion of the gland is only covered by mucous membrane, and when this is scratched through the true prostatic capsule is reached. In doing Freyer's operation the author passes the finger to the extreme anterior limit of the trigone, so that the finger is really within the urethra. At this point he splits the capsule and begins the enucleation, being careful to injure the trigone as little as possible. This precaution greatly lessens the danger of incontinence. The finger is kept in close contact with the true capsule and enucleates the gland by passing first posterior, next outside, and finally in front of one lateral lobe.

"The finger is then swept in a circular fashion from without inward, in front of and to the inner side of the lobe, detaching this from the urethra, which is felt covering the catheter, and pushed forward toward the symphysis between the lateral lobes, which will, as a rule, have separated along their anterior commissure in the course of the manipulations. The other lobe is attacked and treated in the same manner. The finger is next pushed well downward behind the prostate and the inferior surface of the gland is peeled off the triangular ligament. When the prostate is felt free within its sheath and separated from the urethra, with the finger in the rectum, aided by that in the bladder, it is pushed into the bladder through the opening in the mucous membrane, which, during the manipulations, will have become considerably enlarged" (Freyer, Ibid.). The prostate is removed from the bladder by forceps. If the lobes come away separately, Freyer believes that the ejaculatory ducts are uninjured and remain attached to the urethra. There has been an active controversy as to whether Freyer's operation does or does not destroy the prostatic urethra. It seems certain that even if it is left it must slough for want of blood-supply. In my operations by this method it has come away with the prostate.

In Freyer's earlier operations he sought to leave the urethra and accom-

panying structures behind, but he is now convinced that the prostatic urethra may be torn or removed without ill results. Figure 931 shows the drainage as I employ it after suprapubic prostatectomy. Figure 932 shows the drain.

**McGill's Operation.**—The bladder is opened by a suprapubic incision, the edges of the cut bladder are sutured to the abdominal wound with catgut, and the interior of the viscus is carefully explored by the finger and by sight, an electric light being used for illumination. If a sessile growth exists, the mucous membrane is incised and the growth enucleated by finger or a curet. A pedunculated growth is cut away by sharp-edged forceps. If a mass projects into the bladder, an incision is made to divide it into two portions and each half is enucleated. Hemorrhage is arrested by irrigation with hot salt solution and by compression with gauze pads. In some cases a tampon must be inserted. The bladder is drained for several days or a number of days. As a matter of fact, a dense fibrous prostate cannot be enucleated and can be removed only by scissors or cutting forceps.

**Fuller's Operation.**—Open the bladder above the pubes; have an assistant push the gland up by means of a fist in the perineum. The gland can be

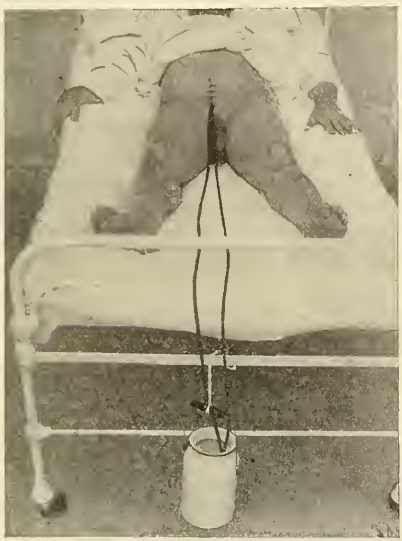


Fig. 931.

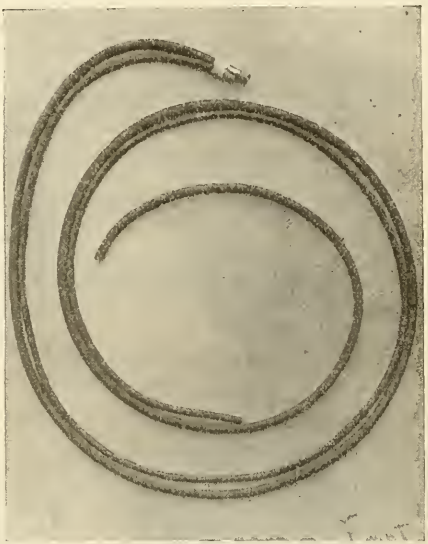


Fig. 932.

Figs. 931 and 932.—Showing double-tube drainage for suprapubic operation. Fig. 931 was taken from an elevation to show the termination of the drainage-tubes. Fig. 932 shows tubes enlarged in separate view.

lifted by two fingers in the rectum. The surgeon makes a small incision through the mucous membrane over the prostate, enucleates the gland by means of the finger, and drains through an incision in the membranous urethra, as well as through the suprapubic opening.

**Belfield's Operation.**—Belfield performs suprapubic cystotomy, makes a perineal cut to enable the finger to approach the prostate, pushes the prostate up toward the belly, and enucleates it from within the bladder.

**Perineal prostatectomy** is less bloody than suprapubic prostatectomy. The sphincter of the bladder is not damaged, the entire prostate can be brought into view and removed, and perfect drainage is obtainable after operation.

**Nicoll's Operation.**—Perform suprapubic cystotomy. Then incise the perineum down to the prostate, split the capsule of the prostate, insert two fingers of the left hand into the bladder, and push the prostate into the perineum so

as to bring it within reach. Enucletae the gland from the perineal wound without damaging the mucous membrane of the floor of the bladder.

**Alexander's Operation.**—Alexander makes a suprapubic incision and uses it for the same purpose as does Nicoll, but he also opens the membranous urethra on a grooved staff. After enucleating the gland he inserts a drainage-tube through the incision in the membranous urethra. In a very thin subject it may not be necessary to perform suprapubic cystotomy. Alexander has brought the gland into an accessible position in the perineal wound by suprapubic pressure, and Guitéras has done so by making an incision in the linea alba and inserting two fingers into the prevesical space. Syms advocates opening into the peritoneal cavity, inserting the hand, and pressing the prostate into the perineum without opening the bladder above the pubes.

**Bryson's Operation.**—This is a satisfactory method in some cases. The bladder is irrigated and filled with warm salt solution. A grooved staff is introduced and a median perineal section is made to open the urethra just in front of the apex of the prostate gland. The knife is pushed back in the groove of the staff sufficiently far to incise the ring at the apex of the prostate; the forefinger is passed into the prostatic urethra and the staff is withdrawn. Then a short tear is made by means of a blunt instrument into the mass of the left lobe and the finger is introduced and enucleates the lobe. The same procedure is carried out on the right lobe, and, finally, if necessary, on the middle lobe. If the middle lobe requires removal, but cannot be reached, a suprapubic cut is made into the cave of Retzius, two fingers are inserted, and the lobe is pushed within reach of the finger below. A large perineal tube is introduced for drainage, a catheter is introduced and tied in place, and bleeding is arrested by packing.

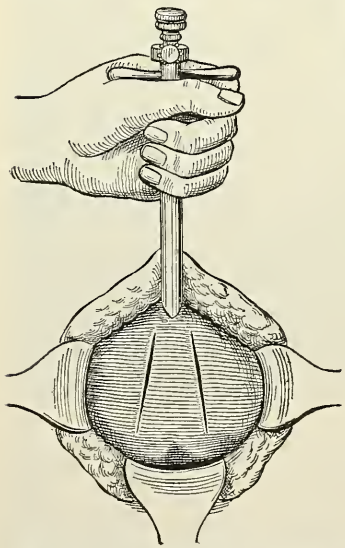


Fig. 933.—Tractor introduced; blades separated; traction made, exposing posterior surface of prostate. Incisions in capsular on each side of ejaculatory ducts (Young).

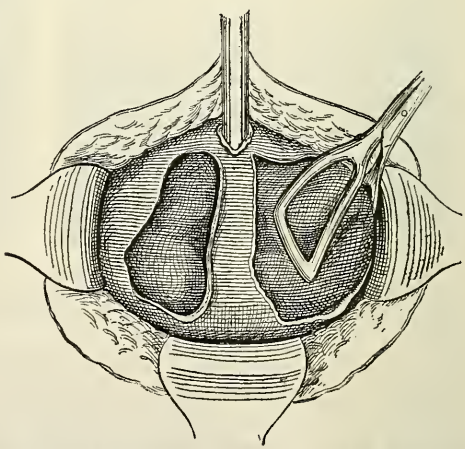


Fig. 934.—Enucleation of lobes. Forceps in position (Young).

**Young's Operation.**—This surgeon frequently operates under spinal anesthesia. He places the patient in an exaggerated lithotomy position and introduces a sound. In thin subjects the incision is in the raphé and is carried close to the anus; in short individuals the incision is an inverted V. He incises the recto-urethralis muscle transversely, exposes the membranous urethra, opens it, and inserts his tractor into the opening in the urethra (Fig. 933). The tractor is turned 180 degrees, the blades are opened, and traction is made.

The capsule is incised on each side of the ejaculatory ducts and the gland is removed by blunt dissection, the forceps grasping each lobe during enucleation (Fig. 934). Every effort is made to save the urethra. After removing the lateral lobes the tractor is used to bring a middle lobe, if one exists, into the wound, and it is also enucleated. The bladder is drained for about one week.

**Young's punch operation** was devised for the removal of small prostatic bars and contractures of the vesical neck involving the prostate. Young reports 200 cases with no mortality. It can be done under local urethral anesthesia; 4 per cent. novocain is employed. The operation is performed by means of a special prostatic punch devised by Young. By means of the punch a portion of the offending bar is removed.

**Bottini's Galvanocaustic Prostatotomy.**—Bottini, of Padula, in 1874 suggested cauterizing the prostate by means of a special instrument. He sought to burn away a portion of the gland in hope that the contraction of the scar would cause the remainder of the gland to shrink. The instrument of Bottini is shaped like a catheter, and carries a platinum blade which is heated by an electric current. Bottini's early instrument was not satisfactory and the operation never became popular until Freudenberg improved the tools in 1897 (Fig. 935).

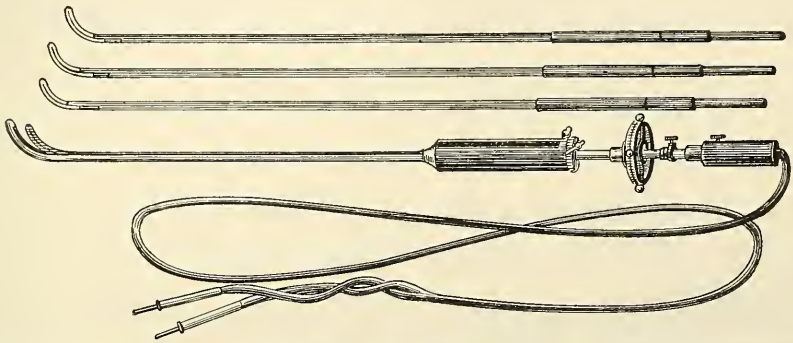


Fig. 935.—Young's modification of Freudenberg's instrument for prostatotomy by galvanocautery.

Bottini's galvanocaustic operation is performed as follows: The bladder should be emptied, irrigated, and distended with air, and the posterior urethra must be anesthetized by instillation of cocain or eucain. The current is tried to see how many seconds it requires to heat the blade sufficiently. The current is broken, the instrument is introduced, the cooling current is set in motion, and one assistant watches this and nothing else. The current is turned on and the surgeon waits the required number of seconds for the blade to become red hot (twelve to fifteen seconds), and then turns the screw at the handle, and burns a groove in the prostate. A groove should be burned toward the rectum, one to the side, and, if it is thought desirable, one to the opposite side. No groove should be burned toward the pubes. When a groove has been burned, the blade is returned into its sheath, the current being increased while doing so in order to keep the blade from adhering to the tissue, then the current is shut off. After withdrawing the instrument it is not necessary to introduce and retain a catheter. The patient is confined to bed only twenty-four hours, there is rarely bleeding or fever, and the results are fairly good. The scars contract and the gland atrophies. During the period of healing a steel sound should be passed from time to time (Bangs). It is alleged that fibrous stricture of the neck of the bladder may follow in some cases.[1]

[1] For description of this operation see Freudenberg, in "Berliner klin. Woch.," No. 46, 1897; and Willy Meyer, in "Med. Record" of March 5, 1898, and May 12, 1900.

Bottini's operation is the procedure to be selected for a sclerotic prostate and for hypertrophy in a feeble and aged individual with damaged kidneys. It is not probable that the cautery operation will ever replace prostatectomy. The best instrument is Young's modification of Freudenberg's instrument (Fig.

Fig. 936.—Incisions of the middle lobe (Young).

935). Figures 936 and 937 show various methods of making the cuts as advised by Hugh H. Young. When there is a distinct and pedunculated median lobe the ordinary plan of burning fails entirely; but, as Young shows (Figs. 936, 938), if an oblique cut is made on each side across the base, this lobe will drop

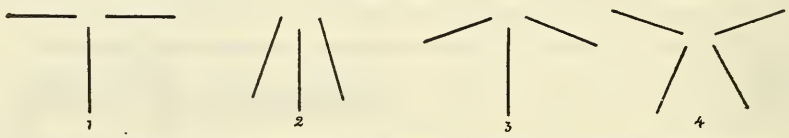



Fig. 937.—Different incisions of prostate gland in Bottini's operation (after Young).

out of the way and quickly atrophy. Bottini's operation does not gain in public confidence.

**Castration and Vasectomy.**—In 1886 Sanitzin demonstrated clinically the shrinking of a large prostate after double castration (Hawley, in "Annals of Surgery," Nov., 1903). In 1893 Ramm, of Norway, performed double castration in order to cause shrinking of an enlarged prostate. In 1893, after a long series of careful experiments, J. William White recommended the operation of bilateral orchidectomy for the treatment of prostatic hypertrophy. He proved that removal of the testicles causes a rapid shrinking in an enlarged prostate. Much of this shrinking may be due to diminution of congestion and edema, but true atrophy undoubtedly occurs in the glandular elements. Very remarkable results have been recorded. In some cases the patients become absolutely comfortable and dispense entirely with the catheter. Cystitis ceases, and desire to urinate frequently becomes less marked. Unilateral orchidectomy has been employed, but it is not satisfactory. In 1894 Mears suggested ligation of the spermatic cord. In 1895 Lauenstein suggested division of the spermatic cord. In 1896 Tilden Brown suggested ligation of the

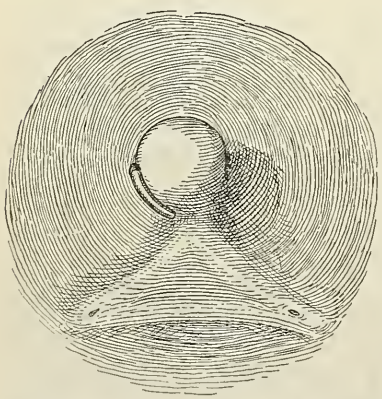


Fig. 938.—Incising the middle lobe (Young).

vas. Reginald Harrison in 1896 advised section of the vas. Lennander in 1897 proposed exsection of the vas deferens (*vasectomy*). It is slower in its results, but just as certain as castration. In spite of the great simplicity of orchidectomy the mortality has been considerable (from 11 to 18 per cent., according to some authors. Socin and Burckhardt say 16.2 per cent.). In several instances mental disturbance has followed the operation. Castration is now very seldom performed, as vasectomy is just as useful and is safer. Vasectomy is valueless in cases of fibroid prostate, does some good in adenoma, but is most valuable when the prostate is generally hypertrophied and prone to great congestion, causing violent symptoms. The testicle does not atrophy after vasectomy, mental disturbance does not occur because the internal secretion of the testicle is still furnished to the organism, and impotence may not develop, though sterility must.

*Other Methods.*—Among other operations which have been suggested are: ligation of the vascular elements of the cord; resection of all the cord elements except the vas and its artery and vein (*angioneurectomy*, proposed by Albarran in 1897); parenchymatous injections of cocain into the testicles; and ligation of both internal iliac arteries. Angioneurectomy has a mortality of 5.5 per cent. (Socin and Burckhardt).

*Selection of Operation and Results.*—The relative merits of these various operations alluded to above are in dispute. It is certain that many cases of prostatic hypertrophy can be kept comfortable by aseptic catheterism. If this procedure fails, or for other reasons must be abandoned, or if the surgeon decides not to employ it, a careful study of the case should be made before selecting a special operation. The Bottini operation for a time had somewhat extensive use. Some applied it to almost any sort of case, claiming that the operation is practically free from danger. Meyer used it for any case of uncomplicated hypertrophy, but if the prostate was very large, ligated the vasa deferentia some weeks before cauterizing the prostate, in order to lessen the danger of thromboses.

A more conservative view is that of Eugene Fuller, who doubts the permanence of the results of the Bottini operation, fears that stenosis of the vesical neck may follow, and would restrict the operation to uncomplicated cases not of a grave character, and in which the bladder has not been seriously damaged. It is the operation of choice if the prostate is fibrous. It is the preferable operation if the patient is old, debilitated, or the victim of kidney disease. Some residual urine usually remains after a Bottini operation. In over 10 per cent. of cases no benefit follows. Vasectomy is used for an engorged and generally enlarged prostate. It may do great good or may fail completely. If the urine is extremely foul, some operation permitting drainage is advisable. In an adenomatous prostate in which enucleation is easy we should prefer the perineal method. In other cases in which it is probable enucleation will be hard; in cases of uncertain diagnosis; in cases in which a calculus may exist; and in cases in which the middle lobe is at fault, do a suprapubic operation, although sometimes a perineal incision may be made, and a cut be made in the prostate to bring the floor of the urethra level with the trigone.

In old men with great obstruction and with serious disease of the bladder and involvement of the kidneys, and in individuals with prostatic cancer, permanent suprapubic drainage is sometimes the most useful procedure.

The mortality from Bottini's operation is over 5 per cent. Young had 3 deaths in 41 operations.

Vasectomy done early gives a mortality of from 3 to 5 per cent. If performed later, the mortality is 10 to 15 per cent. Socin and Burckhardt estimate the mortality of bilateral vasectomy as 8.3 per cent. The mortality of bilateral orchidectomy is 16.2 per cent.

The mortality of perineal and suprapubic prostatectomy has been considered on page 1382.

**Malignant Disease of the Prostate Gland.**—Primary malignant growths of the prostate are not infrequently encountered, but secondary growths are much more rare than primary growths. When malignant disease does occur, it is usually cancerous. Secondary cancer of the prostate finds its most usual antecedent in cancer of the rectum. Epithelioma does not occur. Scirrhus occasionally occurs; but the most frequent form is encephaloid. Sarcoma is rare, although probably not quite so rare as has been thought. Some cases of prostatic tumor obviously inoperable when first seen by the surgeon are probably sarcomatous. Round-celled, spindle-celled, or mixed-celled sarcoma may develop. Powers says there have been but 31 authenticated cases of primary sarcoma reported ("Annals of Surgery," Jan., 1908). According to Gibson ("Jour. Amer. Med. Assoc.," April 23, 1910) there are on record 36 absolutely authenticated cases of sarcoma of the prostate. Sarcoma is most frequent in childhood. It grows rapidly, is usually soft, and causes pain in the rectum and perineum or pubic region, but early in the case, at least, there is seldom residual urine (Powers, Loc. cit.). No real cure of sarcoma has yet been reported.

Carcinoma of the prostate may occur at an earlier age than ordinary hypertrophy of the prostate. The latter does not become evident until after the age of fifty; but carcinoma of the prostate may begin at any time after the age of forty, and sarcoma of the prostate may commence in early youth.

At first the carcinomatous growth enlarges slowly; but it soon begins to grow with rapidity. It breaks through the capsule and fungates into the bladder or into the urethra. The pelvic, the inguinal, and the femoral glands become involved early in the course of the disease. It is unusual to find great obstruction to urination or to the passage of a catheter at an early period, but later both these conditions are noted. Early in the case there is pain only when obstruction to urination occurs; later, the pain in the neck of the bladder may be severe, and there may also be pain in the loin and in the sciatic nerves. Hemorrhage usually occurs. In the beginning the hemorrhage is trivial and intermittent, but when fungation exists, large hemorrhages generally take place. The blood is usually mixed with urine, but there is sometimes a large hemorrhage unassociated with micturition. The urine is not likely to contain pus or any large quantity of mucus unless the bladder is involved in the growth.

When the prostate gland is felt by means of a finger in the patient's rectum, it is found to be of stony hardness and to be firmly anchored in place. Reginald Harrison points out that an ordinary hypertrophied gland is not so firmly anchored as a carcinomatous gland; that the bowel moves over it with freedom; and that, although it is firm to the touch, it is not of stony hardness. The patient with carcinoma of the prostate loses flesh rapidly and develops distinct cachexia, and metastatic deposits are likely to form in the vertebral column, in the kidneys, and in other organs and structures.

In making a diagnosis Harrison insists upon the value of the cystoscope. He says that in cancer one does not find much intravesical projection, and that what projection there is is uneven and irregular. In an ordinary adenomatous prostate, on the contrary, the surface is smooth and rounded and projects into the bladder.

**Treatment.**—Radical operation is out of the question in these cases. Permanent suprapubic drainage is made in most instances, and usually gives the patient great relief. (See "Remarks on Cancer of the Prostate," by Reginald Harrison, in "Brit. Med. Jour.," July 4, 1903.)

**Tuberculosis of the prostate gland** is rarely primary. It is usually secondary to tuberculosis of the kidney. It may follow tuberculosis of the epididymis. In the majority of cases of tuberculosis of the prostate the lungs are involved in a tuberculous process when the patient is first seen by the surgeon. The disease appears particularly between the ages of twenty and thirty years, but it may attack elderly men and even the aged. It begins by the formation of a number of tuberculous nodules in the immediate neighborhood of the prostatic tubules. These nodules caseate and run together, forming cavities and, eventually, tuberculous abscesses, which are prone to rupture into the urethra. In very rare instances a large tuberculous abscess ruptures through the perineum, into the rectum or into the peritoneum.

The disease occasionally undergoes spontaneous cure through fibrous tissue formation or calcification. The tuberculous process is liable to spread to the seminal vesicles, the bladder, the ureters, and possibly the peritoneum; and in some cases it inaugurates thrombophlebitis and pyemia.

**Symptoms.**—The patient suffers from pain during micturition; there is frequent micturition, and from time to time the urine contains blood. Attacks of cystitis take place, and weakness and a loss of flesh are greater than is commensurate with any ordinary inflammation. Tuberculosis of the prostate alone is said not to cause marked hectic fever, but when adjacent structures become involved the temperature is definitely elevated and becomes characteristic. When the disease has advanced there is not unusually urinary incontinence, on account of the involvement of the circular muscular fibers about the neck of the bladder. Commonly, there is a mucopurulent discharge, or mucopurulent matter may be obtained by massaging the prostate. This matter may contain tubercle bacilli, and in some cases the urine also contains bacilli. Early in the course of the case rectal examination detects some enlargement of the gland, many nodules, and tenderness; later in the disease it finds marked enlargement and areas of softening.

**Treatment.**—Early in the case Senn recommends parenchymatous injections of iodoform emulsion, the punctures being made through the perineum. If these fail, operation must be considered. When one takes into account how rare primary tuberculosis of the prostate is, one is impressed with the infrequency with which a radical operation should be attempted. If there is absolutely no evidence that any adjacent organ is involved or that any distant focus of disease exists, it is justifiable to perform perineal prostatectomy. As a rule, however, the only surgical operation performed consists in making a curvilinear incision in front of the rectum, which exposes the prostate, and permits the surgeon to open and curet caseous foci. If an abscess forms, it should be evacuated by means of a perineal incision and cavities should be curetted and packed with iodoform gauze.

When a patient is convalescent after an operation or if it is determined that no operation is advisable, full antituberculous treatment is employed (see page 230). One should look to the patient's general health, administer urotropin, and avoid using instruments as much as possible; because, as Sir Henry Thompson has shown, instrumentation irritates the prostate, causes a great deal of pain, and makes the disease worse in every case.

**Retained and Malplaced Testicle.**—The normally descended testicle is entirely within the scrotum. In 1 person of 1000 there is either undescended or ectopic testis. In 80 per cent. of individuals the testicles have descended at birth; most often it is the right testicle which fails to descend. Sometimes a testicle descends after being retained for months or even years. In Keyes's case it descended in the thirtieth year. Late descent usually causes hernia, and in 90 per cent. of all cases hernia exists. The testicle may be arrested in its passage to the scrotum (*cryptorchism*, single or double); it may remain in

the lumbar region; it may reach the internal abdominal ring; it may lodge in the inguinal canal; it may emerge from the external ring, but fail to enter the scrotum; or it may pass into an unnatural position, as into the perineum or the crural canal (*ectopia of the testis*). The failure of descent may be unilateral or bilateral, but when bilateral the degree of descent is seldom the same on the two sides. The gland may be, but seldom is, functionally active. When retained in the abdomen it never has the power of spermatogenesis. Before puberty the testicle is usually normal, but after puberty there is practically always more or less atrophy. In about 1 case out of 5 there is spermatogenesis for a time. A retained testicle is liable to attacks of orchitis and may become tuberculous, carcinomatous, endotheliomatous, or sarcomatous. In most cases there is neither pain nor tenderness, and the patient presents himself for treatment because a lump has appeared in the groin. In some cases there are sudden attacks of violent pain, accompanied by nausea (Rawling, on "Incompletely Descended Testicle," in "Practitioner," August, 1908). A testicle in the inguinal or crural canal or in the perineum is far more apt to become sarcomatous than one retained within the abdomen. Over 10 per cent. of cases of sarcoma of the testicle are in undescended glands. Russell Howard ("Practitioner," Dec., 1907), out of 57 cases of undescended testicle, found 15.7 per cent. with malignant disease. In double cryptorchism, in which the testicular function has been abolished, there is delayed union of the bony epiphyses and epiphyseal fractures are common, and there may be excessive growth of long bones. The same liability is noted in those subjected to castration in infancy. When such a subject reaches manhood, he may develop some disease of the skeleton which is usually seen only in children (Gross and Sencert, "Rev. de Chir.," No. 11, 1905). In operating on these cases we usually find a well-developed gubernaculum and a vaginal tunic extending below the testicle.

**Treatment.**—If one testicle is undescended at or after the eighth year of life and before puberty, if it lies in the canal, and if the other testicle is sound, the former should be removed if it is found impossible to draw the gland into the scrotum. Both testicles should not be removed from a child: one should be placed within the abdomen. I would save one testicle in order to have the child certainly enter and remain in the masculine groove. Removal of both testicles is permissible in an adult, because he has definitely become and will remain masculine, and undescended or ectopic testicles in an adult are or will surely become functionally useless and menaces. If a testicle is retained in the abdomen it should not be operated upon unless it causes trouble. Always try to get a retained gland into the scrotum before the age of puberty. If it is retained after puberty, it will be almost certain to be or to become functionally useless. An ectopic testicle should be restored to the scrotum if possible; if not, it should be removed. Even when operation is performed to replace the testicle, success is seldom enduring. In Rawling's 29 cases only 4 were permanently successful. Other operators, however, claim better results. For instance, Broca reports 79 cases traced from one to six years, and in 31 of them the result was perfect and permanent. The method I employ is that advocated by Coley, viz., Bassini's incision through the aponeurosis, separation of the cord from the peritoneum (it is usually in the posterior sac wall); removal of the vaginal process, the cremaster and fibrous adhesions around the cord; the making of a scrotal pouch by the fingers, closure of the canal as in hernia; suturing of the cord to the pillars of the external ring, as advocated by Dowd. This is practically Broca's method. Bevan operates for a testicle within the canal by separating and dividing the vaginal process, removing all the coverings of the cord, and leaving the testicle suspended by the vas and spermatic vessels only. This proceeding gives the testicle a very wide range of mobility. A pocket is made in the scrotum by

means of the finger, the testicle is placed in the pocket, and remains there without suturing. The canal is now sutured as in a Bassini operation without cord transplantation. There is a certain though small risk of gangrene of the testicle after Bevan's operation. I believe Bevan's method should be employed in the more severe cases. If there is no hernia, operation should not be performed until between the eighth and twelfth years of life.

**Orchitis** is inflammation of the testicle. *Acute orchitis* may follow cold, wet, traumatism or epididymitis, and may follow or develop during gout, mumps, rheumatism, or a specific fever. The testicle is round, swollen, tender, and very painful, the scrotum is red and swollen, the tunica vaginalis is filled with fluid, and there is fever. *Chronic orchitis* results from the acute form or from a chronic urethral inflammation, and is almost always combined with epididymitis. *Gonorrheal orchitis* is rare and almost certainly results in sloughing of the testicle.

The **treatment** of the *acute* form consists of rest in bed and applications as for epididymitis (see page 1397). The *chronic* form requires the removal of the causative lesion, if possible, the wearing of a suspensory bandage, applications of ichthyol or mercurial ointment, and the administration of iodid of potassium by the mouth. Strapping with zinc oxid adhesive plaster may do good. Castration may be required.

**Tuberculosis of the testicle** may perhaps be primary, but in the vast majority of cases is secondary to tuberculosis of the kidney, prostate, bladder, or seminal vesicles. As Keyes ("Annals of Surgery," June, 1907) says, careful examination will show one of three conditions—tubercle bacilli in urine, indurations in the prostate, and vesicles or "a distinct haze in the urine due to prostatic catarrh." Tuberculosis of the prostate or vesicles exists in probably one-half the cases (Barney, in "Boston Med. and Surg. Jour.," March 14, 1912). In about one-third of the cases there is evidence of tuberculosis distant from the genito-urinary organs (especially in the lungs or bones). Patients with tuberculosis of the testicle are nearly always sterile. The disease may be preceded by pulmonary tuberculosis, lymphatic tuberculosis, peritoneal tuberculosis, anal fistula, renal tuberculosis, or tuberculous disease of bones or joints; and primary tuberculosis of the testicle may be followed by near or distant tuberculous lesions. In some cases involvement of the prostate exists, but cannot be detected (*latent tuberculosis of the prostate*); in other cases the prostate is in a state of subacute inflammation. The epididymis is usually involved before the testicle and early chronic lesions are localized there for some time. In most cases the bacilli reach the prostate and vesicles by way of the blood, and reach the epididymis by way of the vas deferens, the lesions of the prostate and vesicles developing first or remaining latent. In some cases tuberculosis of the kidney or bladder is followed by tuberculosis of the testicle. The spread from the prostate, vas, or bladder is by epithelial infection. There is no evidence confirmatory of the idea of ascending infection from the urethra. In a child with an open vaginal process, tuberculous peritonitis may directly cause tuberculous epididymitis. The disease begins in one testicle, but in the vast majority of cases the other testicle becomes involved after a few weeks or months. If the other testicle remains free for three years its chance of remaining free is good. If but one epididymis is involved the testicle may not be affected for weeks or months. Von Bruns says that in 18 per cent. of such cases the testicle is not involved for six months; in 40 per cent., for over two months ("Archiv. f. klin. Chir.," Bd. 63, H. 4). It usually comes on gradually, but it may begin acutely, as I have seen in two instances during the progress of tuberculous peritonitis. An acute onset or an acute exacerbation of a chronic case usually means mixed infection. The disease may follow a slight injury or inflammation, and is most common in young men, but may arise at

any age. The causal influence of antecedent or existing gonorrhea is doubtful. Some maintain that sexual excess predisposes. There is often a family history of tuberculosis. A chronic case begins by swelling of the epididymis. Palpation detects one or two or several rounded nodules, or a diffuse hardening. In the latter case the epididymis is much enlarged, and there is usually a slight hydrocele. Rectal examination commonly detects nodules in the prostate and vesicles. In a few cases there are frequency of micturition, tenesmus, hematuria, and seropurulent fluid can he massaged from the vesicles and prostate and milked from the urethra. In some cases bacilli are found in the urine. In others the urine is hazy. In about 80 per cent. of cases the guinea-pig test will prove the presence of bacilli. In some cases the urine is normal. Sooner or later nodules appear in the testicle. The vas is always swollen and may or may not be palpable. In an acute case one testicle is involved. The testicle is very painful and the epididymis is greatly swollen and smooth. The testicle quickly swells, there is always a hydrocele, and the scrotal skin becomes

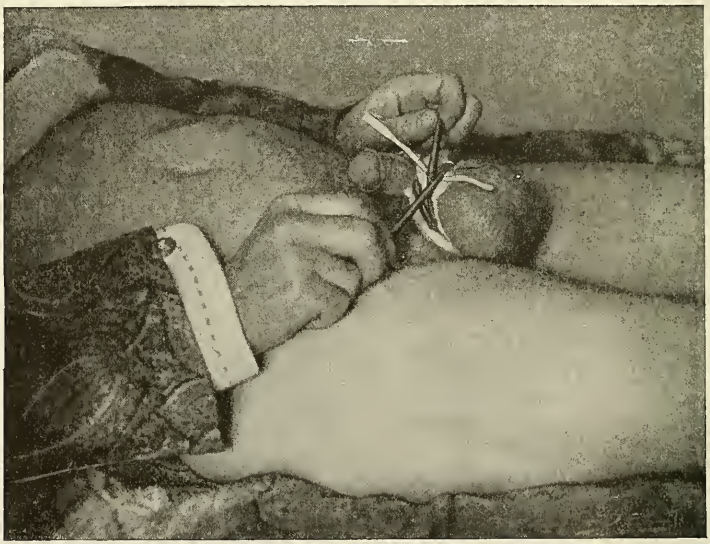


Fig. 939.—Obstructive hyperemia for the testicles. The ends of the elastic tube are held by the patient, crossed. A piece of tape is placed beneath to be tied by an attendant (Meyer and Schmieden).

reddened. In a few days the acuteness of the symptoms subsides, but suppuration occurs soon. In any case of tuberculosis of the testicle nodules tend to soften and run together. After a time the skin may become red and adherent, give way, and expose a caseous breaking-down epididymis or testicle. Caseation can occur without mixed infection, but in many cases in which softening and sinus formation occur there is mixed infection. The duration of the disease is uncertain; 10 per cent. of the 100 cases carefully studied by Keyes were known to be alive ten years after the beginning of the trouble, and 4 (and not one of them had been operated upon) seemed free from tuberculous lesions anywhere. One-third of the cases that suppurated were apparently well after three years ("Annals of Surgery," June, 1907). Except in the acute cases the testicle is only slightly, if at all, painful, and tenderness is trivial. In one-sixth of the cases a small hydrocele forms. If a hydrocele exists the fluid should be withdrawn by tapping in order that cultures may be made from it.

**Treatment.**—Before attempting any operation try Bier's method. The

patient is placed recumbent, the diseased testicle is lifted upward, cotton batting is placed around the neck of the scrotum, and a rubber drainage-tube of a caliber of 25 of the French scale is wound twice around and fastened with a string or clamp. If both testicles are diseased, both are held up and the neck of the scrotum is embraced by the tube with the required firmness (see Fig. 939). The treatment is applied for two or three hours a day or longer. The patient had best be recumbent during the application. In the intervals he wears a suspensory and gets about. During the use of Bier's method full antituberculous treatment is required (see page 230). If a cold abscess forms, open it and dress the part antiseptically. If Bier's treatment fails, consider the advisability of operation. An acute case requires unilateral castration. If, in a chronic case the disease is limited to the epididymis or to the epididymis and vas, resect the epididymis (*epididymectomy*) and the vas deferens. If the testicle is diseased, *orchidectomy* is performed. It was long believed that orchidectomy was useless if the vesicles and prostate were involved, but Koenig and others maintain that vesicular and prostatic tuberculosis improves after removing the diseased testicle or epididymis. If the epididymis of each testicle is involved, bilateral epididymectomy should be performed. When both testicles are diseased and other organs and structures are not extensively involved, bilateral orchidectomy is performed or, better, the testicle which is most diseased is removed and the *diseased portion* of the other is extirpated. Cumston points out that when the testicle is diseased the disease may not be detectable even on operative exposure. Hence in doing epididymectomy he splits open the testicle to see if it is diseased. If it is not diseased he sutures it with catgut and removes the epididymis. If it is diseased he considers the advisability of unilateral orchidectomy (Charles Greene Cumston, in "Annals of Surgery," June, 1909).

In many cases after epididymectomy sinuses form. They may remain open for months. In association with and after operation employ antituberculous remedies, order a nourishing diet, send the patient to a good climate, and insist on an open-air life. Tuberculin may prove useful. A considerable percentage of unilateral cases are cured by operation (over 40 per cent.). Some few bilateral cases are cured.

**Cysts and Tumors of the Testicle.**—Innocent tumors are very rare; in fact, some dispute their existence. The elements of a testicular growth are very complex. A majority of growths are unquestionably malignant. Some are wholly malignant. Many show a mingling of benign and malignant elements. Even growths which are not malignant in the beginning have an irresistible tendency to become so. It is believed by many surgeons that fibroma and adenoma can arise and perhaps remain for some time benign. Embryomata are at first benign, but tend, perhaps after years, to become malignant.

**Embryomata.**—H. Morriston Davies ("Lancet," Feb. 17, 1912) considers embryomata to be "composite tumors containing elements derived from epiblast, mesoblast, and hypoblast." They are "developed in the body of the testicle" and not in the epididymis, though that structure may eventually be involved. Early in their development these growths are benign, but any or all the layers may become malignant (Nicholson, in "Guy's Hospital Reports," vol. lxi, 1907).

When two or more layers become malignant "a mixed tumor is the result; when hypoblast alone, the tumor assumes the character of a columnar-celled carcinoma; when mesoblast alone, of a sarcoma or myxosarcoma" (Davies, Loc. cit.). When a dermoid-like growth from the epiblast becomes malignant it resembles chorionepithelioma. Nicholson (Loc. cit.) divides embryomata into solid embryomata (which often contain cysts and are common) and cystic embryomata or dermoids (which are very rare). Many surgeons regard

sarcoma, others carcinoma, as the commonest form of malignant disease. Endothelioma may occur.

**Malignant Disease of the Testicle.**—It may arise from an embryoma or may be malignant from the start. If a tumor which has long existed, perhaps years, begins to grow rapidly, we may assume that there is malignant change in an embryoma. It is seldom possible to diagnosticate the form of a malignant tumor. Sarcoma is usually of the small round-celled variety (the type known as lymphosarcoma). The other testicle soon becomes involved in malignancy. The growths disseminate rapidly. In 10 per cent. of cases the testicle is undescended. Any sarcoma is liable to sudden increase in size because of hemorrhage.

Nicholson ("Guy's Hospital Reports," vol. lxi, 1907) divides carcinomata into encephaloid (which is commonest) and scirrhus (which is rare), and regards so-called columnar carcinomata as teratomata. Davies ("Lancet," Feb. 17, 1912) admirably sums up the clinical picture of malignant disease of the testicle. I have utilized his article extensively below. The tumor may attain a great size (that of a cocoanut). It is usually oval, but may exhibit a few rounded projections "due to the presence of degeneration in the tumor or of fluid in the tunica vaginalis." Early in the development the growth is smooth (except for the projections mentioned), later it breaks through the tunica albuginea and so comes to have an irregular surface. The epididymis is at first free, but sooner or later becomes part of the tumor. The consistence of malignant tumors is very variable (may be hard, elastic, soft, or fluctuating), and the consistence of a tumor may vary in different parts. In most cases there is an associated hydrocele, in which the fluid may be limited or may be distributed throughout the entire sac. The hydrocele may be translucent, the tumor never is. Pain is seldom severe and tenderness seldom acute in the testicle. I have seen terrible agony when the lumbar glands are involved. The tumor when raised on the hand feels very heavy. The cord becomes enlarged because of involvement.

The growth finally adheres to the scrotum, and the skin reddens and gives way and fungation occurs. When this happens there is severe pain. Sarcoma of the testicle, unlike sarcoma in most other regions, causes early glandular involvement. Carcinoma, of course, involves glands. The inguinal glands are not involved unless the scrotum is attacked. The lumbar glands receive the testicular lymphatics. Secondary growths are early and widespread and are common in the skin.

H. M. Davies emphasizes the great fatality of malignant disease of the testicle; quotes Chevassu's collection of 100 cases treated by castration, of which 81 died of malignant disease and only 19 were cured, and insists that "early diagnosis and the removal of the testicle and surrounding fascia and the glands in the lumbar region offer the only hope of decreasing so appalling a mortality" ("Lancet," Feb. 17, 1912).

**Orchidectomy, or Castration** (*Excision of a Testicle*).—Bilateral castration should never be performed without deliberate consideration. It often produces grave mental disorder. This is in part the result of the mental depression attendant on knowing that the highly prized glands are gone for ever, and in part the loss to the organism of the internal secretion of the testicles. A boy castrated before puberty never becomes potent. A man may retain potency for a considerable time after castration. I removed a tuberculous kidney from a man who had been castrated by my colleague, Dr. Horwitz, two years before, yet he was still able to have intercourse. Unilateral orchidectomy does not make a man either sterile or impotent and does not produce mental disturbance. In orchidectomy for

benign disease an incision is made over the cord, commencing just outside the external ring and running down over the base of the tumor. Clamp the cord and divide it near the ring, remove the testicle, ligate the spermatic artery alone, and then ligate the entire thickness of the cord. The cord is ligated with chromic gut. The skin is sutured with silkworm-gut. Drainage is not required. It is often advisable to remove a considerable amount of scrotal skin. Orchidectomy for malignant disease must be a much more radical procedure. After the cord has been divided and the testicle removed, as in the ordinary operation, the incision is prolonged along and through the roof of the inguinal canal, and is continued in the same direction to a point a little above the anterior superior spine of the ilium. The incision is then curved and carried up to "the costal margin at the level of the tenth rib" (Davies, in "Lancet," Feb. 17, 1912). The peritoneum is exposed. The cord is traced well into the true pelvis, is tied, and divided. The fascia over the iliacus and psoas muscles is dissected, "together with the contained spermatic vessels and lymphatics," and the glands upon "the inferior vena cava and aorta" are removed (Davies, Ibid.). This operation was first performed by Grégoire in 1905. Davies did the thirteenth operation on record (Ibid.).

**Epididymitis,** or *inflammation of the epididymis*, is usually due to inflammation of the urethra. It is apt to occur in the stage of decline of a gonorrhea, and in such a case is announced by a notable diminution or a complete cessation of discharge. It may result from the passage of a urethral instrument, the voiding of urine which contains fragments of calculi, or as a complication of prostatic hypertrophy. *Acute epididymitis* is characterized by swelling of the epididymis, severe pain in the groin, and tenderness over the posterior part of the testicle. The pain becomes acute, swelling rapidly increases, and the constitution sympathizes. The swelling is due partly to engorgement of the epididymis and partly to fluid in the tunica vaginalis (*acute hydrocele*). *Chronic epididymitis* is usually linked with orchitis, and it follows an actue exacerbation of a chronic urethral inflammation.

**Treatment** by aseptic puncture with a tenotome, if fluctuation is marked, will relieve tension and pain. Hagner makes multiple punctures in the epididymis. Leeching over the external abdominal ring, the use of an ice-bag early in the case, elevation, application of guaiacol, and administration of laxatives and opium constitute the usual treatment in the acute stage. Applications of guaiacol over the cord, epididymis, and testicle seem to relieve pain and distinctly lessen swelling. Two applications a day should be made for one week. At each application paint the scrotum and over the external ring with 15 drops of guaiacol in 1 dram of glycerin or olive oil. Strapping is employed as the inflammation subsides. The treatment of the chronic form is the same as that for chronic orchitis.

**Strangulation of the Cord by Axial Rotation.**—In nearly one-half of the cases the testicle is undescended or only partly descended. In every case there is a long mesorchium, and if a normal testicle is normally placed torsion of the cord will hardly occur (Chas. L. Scudder, "Annals of Surgery," Aug., 1901). The twisting may be toward the right or toward the left. The symptoms arise suddenly, and usually during exertion. In some cases a hernia also exists. When the rotation occurs, the testicle swells, hemorrhages take place into it, and gangrene may develop. If the cord of an undescended or partially descended testicle twists, swelling and tenderness are noted in the abdomen or groin. If the swollen testicle is in the scrotum, the gland feels nodular and the epididymis is found to be anterior instead of posterior, as it is in a normally placed gland. The symptoms are sudden pain, vomiting, moderate shock, and a swelling in the groin or a swollen testicle in the scrotum. The swelling receives no impulse on coughing. The symptoms resemble those of

strangulated hernia, but are less violent, and the bowels, though often much constipated, are not obstructed.

**Treatment.**—An incision should be made, and if the twisting was recent and the testicle is not gangrenous, the cord may be untwisted and the testicle fastened to the scrotum by a catgut stitch. If the testicle is gangrenous it should, of course, be removed. Scudder tells us that in 88 per cent. of cases the testicle is found to be gangrenous. According to Scudder, there are 32 cases on record: 31 were operated upon and 1 was not, but all recovered; in 3 the testicle sloughed and in 2 it atrophied ("Annals of Surgery," Aug., 1901).

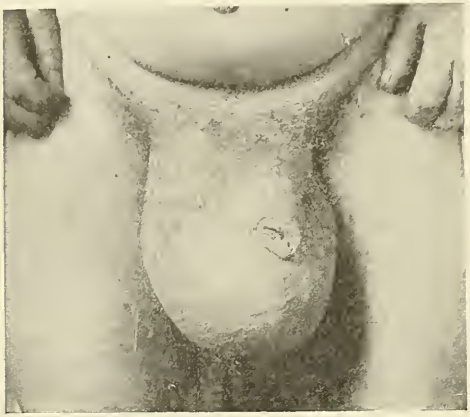


Fig. 940.—Hydrocele of tunica vaginalis (Horwitz).

**Vaginal hydrocele** (*chronic hydrocele*) (Figs. 940 and 942, *e*) is a collection of fluid in the tunica vaginalis testis. An enlargement of the testis may cause it (*secondary hydrocele*), but in most instances the cause is unknown and no signs of inflammation exist (*primary hydrocele*). The fluid is albuminous, but it does not coagulate spontaneously; it is thin, straw colored, and may contain crystals of cholesterin. The testicle is at the lower and back part of the sac. The pyriform mass fluctuates, is translucent, grows from below upward, and the introduction of an exploring needle permits the yellow fluid to flow out. Sometimes a hydrocele has an hour-glass shape. This is the *hydrocele "en bissac"* of the French. In this condition (Fig. 941) two cavities exist, usually but not invariably communicating. The constriction between the cavities is due to inflammatory thickening.

Fig. 941.—Hydrocele "en bissac." This hydrocele extends up the cord into the inguinal canal and to the internal abdominal ring (Horwitz).

**Treatment.** — In secondary hydrocele the treatment of the diseased testicle is the essential plan. We discuss here the treatment of primary hydrocele. Simply tapping the sac with a trocar is only palliative; air must run in as fluid runs out, and suppuration may occur, which will be dangerous without drainage. Never tap a rigid sac. The injection of irritants should be abandoned, as it exposes the patient to serious danger because of inflammation occurring without provision for drainage. A good plan is to incise the sac, dry its interior by bits of gauze, swab it out with pure carbolic acid, pack it with iodoform gauze, and dress it antiseptically. The packing is removed in

twenty-four hours and the wound is allowed to close. In most cases I prefer this method. If the sac is rigid and will not collapse, either stitch it to the skin and pack it or excise a large portion of its parietal layer and insert a drainage-tube (*Volkmann's operation*). Another plan is to tap the sac with a trocar and cannula, leaving the cannula in place as a drain for some days, and dressing antiseptically.

Longuet's operation is easy and is advocated by many surgeons. It is called *extraserous transposition of the testicle.* It was introduced by Longuet in 1898 ("Progrès Méd.," Sept. 21, 1901). Doyen and Winklemann do a similar operation. Jaboulay, too, advocates splitting the sac and turning it inside out. He folds it around both the testicle and cord and stitches it so that the smooth endothelial surface of the tunic will be in contact with the raw scrotal tissue. It will adhere to the scrotal tissue ("Keen's Surgery," vol. iv). A local anesthetic is injected and an incision 2 inches in length is made. The testicle is lifted from the scrotum. The serous and all the other coats except the skin fall together behind and make a sheath for the cord. One catgut suture will hold them behind the cord. A bed is made for the testicle beneath the inner edge of the skin wound by tearing with the fingers. The testicle is rotated on its long axis and inserted into this cavity. The testicle rests against the scrotal septum, and in front of the gland is the cord covered by the tunic. The skin is sutured and the wound is dressed. E. Wylly Andrews devised *the*

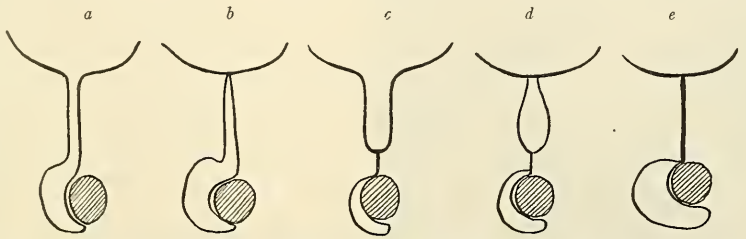



Fig. 942.—Varieties of hydrocele: *a*, Congenital; *b*, infantile; *c*, funicular; *d*, encysted; *e*, vaginal.

*bottle operation* ("Keen's Surgery," vol. iv). The unopened tunic (with the testicle) is separated from the scrotum and dislocated through the scrotal wound. A small incision is made near the summit of the funnel-like prolongation of the sac upon the cord. The sac empties and then resembles a bottle or bag with a small opening at the top. The testicle is squeezed through the opening. When this is done the sac is inside out and the edges of the small opening lie closely about the cord. The skin is closed without drainage. In some cases after doing any one of the operations which turn the sac inside out a large tender mass forms, composed of swollen testicle and thickened sac. I have had this experience several times. In such a case it is necessary to excise the thickened sac.

**Congenital hydrocele** (Fig. 942, *a*) is hydrocele through an unclosed funicular process into the tunica vaginalis. If the pelvis is raised the fluid runs back into the peritoneal cavity, from which it originally came.

The **treatment** is the application of a truss to obliterate the funicular process, and when that occurs, if the counterpart of an infantile hydrocele persists, puncture and scarification of the walls of the sac.

**Infantile hydrocele** (Fig. 942, *b*) is a collection of fluid in a funicular process and the tunica vaginalis, the funicular process being closed above, but not below.

The **treatment** is to puncture the sac and to scarify the sac wall with a needle.

**Funicular Hydrocele** (Fig. 942, *c*).—The funicular process is closed below, but is open above. Raising the pelvis causes the fluid to trickle back into the peritoneal cavity.

The **treatment** is the application of a truss.

**Encysted Hydrocele of the Cord** (Fig. 942, *d*).—In this variety the funicular process is obliterated above and below, but it is patent between these two points and fluid collects.

The **treatment** is the same as that for infantile hydrocele. If this fails, incise and pack.

*Encysted hydrocele of the testicle and of the epididymis* may occur. *Diffused hydrocele* of the cord is simply edema of the cord. *Hydrocele of a hernia* is the distention of a hernial sac by peritoneal fluid.

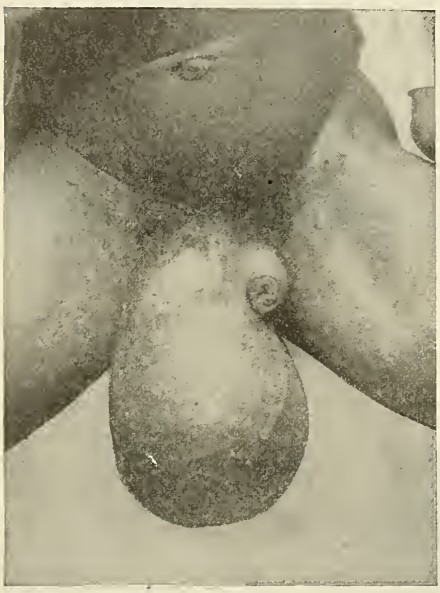


Fig. 943.—Acute hematocele of tunica vaginalis the result of traumatism (Horwitz).

**Hematocele** (Fig. 943).—*Vaginal hematocele* is blood in the tunica vaginalis, the result of traumatism, a tumor, or the tapping of a hydrocele. There is a pyriform swelling which fluctuates, but which gradually becomes firmer; the scrotum is livid and the testicle is below and posterior to the tumor. The *encysted* form of *hematocele of the cord* is a hydrocele of the cord into which bleeding has occurred. The *diffused* form is due to extravasation of blood into the cellular substance of the cord. *Encysted hematocele of the testicle* is due to effusion of blood into an encysted hydrocele of the testicle. *Parenchymatous hematocele* is extravasation of blood into the substance of the testicle.

The **treatment** of a recent case of vaginal hematocele is to put the patient to bed, support the scrotum, and apply an ice-bag over the testicle. If the swelling does not soon abate, incise, irrigate, and pack.

**Varicocele** is varicose enlargement of the veins of the venous plexus of the spermatic cord. The veins are thickened, lengthened, dilated, and convoluted. The assigned causes are straining, cough, constipation, and an occupation requiring prolonged standing. Some believe ungratified sexual desire is a cause. Hereditary predisposition is probable. There are more left-sided than right-sided varicoceles, because the right spermatic vein has valves and empties into the vena cava at an acute angle, but the left spermatic vein has no valves and empties into the left renal vein at a right angle. Varicocele is a very common condition. The elder Senn found it in 21 per cent. of 10,000 recruits. An irregular swelling exists in the scrotum and extends up the cord. This swelling feels like "a bag of earth-worms"; it exhibits a slight impulse on coughing; the scrotal skin and cremaster muscle are attenuated; the testicle lies at the bottom of the swelling and is softer and smaller than normal; the swelling diminishes on lying down and increases on standing or on making pressure over the external ring. The scrotum is pendulous and the scrotal skin frequently contains varicose veins. The testicle may be soft and shrunken. There is usually some discomfort, aching,

or dragging in the testicle and the groin, and often neuralgic pain in the cord. There may be no discomfort of any sort. A large varicocele may be free from discomfort and a small varicocele may produce much annoyance, or vice versa. There are sometimes mental depression and hypochondriasis. As a man reaches middle age a varicocele usually ceases to give trouble.

**Treatment.**—In treating varicocele, reassure the patient: tell him there is no real danger of impotence; order cold shower-baths, correct constipation and indigestion, give occasional tonics, and order the patient to wear a suspensory bandage. If the testicle is undergoing atrophy, if the pain and the dragging are annoying, or if the mind is much depressed, operate.

*Operation for Varicocele.*—Subcutaneous ligation is no longer practised. The open operation is universally employed.

The patient is placed in a recumbent position. Local anesthesia is very satisfactory. A fold of skin is pinched up over the external ring, and the surgeon transfixes it on the line of the cord, so that he will have an incision about 1½ inches long. The skin and fascia are cut by a scalpel, the veins are well exposed, and the cord is located and held aside. A double ligature of strong catgut or chromicized gut is passed under the veins by an aneurysm needle. The threads are separated 1 inch, tied tightly, and the ends are left long. The veins between the ligatures are excised. The two gut ligatures are tied together and cut. This shortens the cord. The wound is sewed up with silkworm-gut.

Bloodgood points out that it is well to avoid dividing the genital branch of the genitocrural nerve which supplies the cremaster muscle. If this nerve should be divided, the cremaster will become lax and return of the varicocele will be favored. Bloodgood makes the incision over the external ring, draws the veins up, and resects them. A wound so placed heals more certainly and promptly than does a wound of the scrotum. Of late years I have always followed this plan.

## XXXVIII. AMPUTATIONS

An amputation is the cutting off of a limb or a portion of a limb. Removal of a limb or a portion of a limb at a joint is known as "disarticulation." Amputation may be necessary because of the existence of severe injury, of gangrene, of tumor, of intractable disease of bones or joints, of ulcer which will not heal, of traumatic aneurysm, etc. A re-amputation may be required because of the existence of a defective stump or disease in the stump.

**Classification.**—Amputations are classified as follows: (1) As to time of operation after the injury: a *primary* amputation is performed soon after the occurrence of the accident—as soon as the sufferer reacts from shock, and before he develops fever; a *secondary* amputation is performed some time after the accident, suppuration having supervened; and an *intermediate* amputation is performed during the existence of fever, but before the development of suppuration. (2) As to the situation, where the bone is divided or according to which joint is cut through. (3) As to the form and situation of the flap.

In performing an amputation maintain rigid asepsis; completely remove the hopelessly damaged portion; sacrifice as little of the sound tissue as possible; prevent hemorrhage during the amputation, and carefully arrest it after the operation; have enough sound tissue in the flap to *cover* the bone, and enough skin to *cover* the muscles; and secure drainage at a dependent point.

*Hemorrhage* may be prevented by the elastic bandage of Esmarch (Fig. 944). Ordinarily we can apply this bandage from the periphery to well above the line of prospective incision, encircle the limb with an elastic band (not

the thin tube shown in the cut), and remove the bandage. The bandage and band, asepticized before using, are applied to the limb, which has been carefully sterilized. After the band has been applied the limb should not be freely or forcibly moved, because of the danger of tearing muscles which are firmly fixed by the compressing band. When elastic compression has been used in an operation the surgeon, after removal of the band, should be very careful to tie *every visible vessel*. The paralysis of the small vessels induced by pressure often prevents bleeding, and unless their mouths be found and the vessels be tied reactionary hemorrhage will occur. Reactionary hemorrhage is the great danger after the use of the Esmarch bandage, and paralysis or sloughing may also follow its employment. If there be an area of suppuration or of gangrene or an extra-osseous malignant growth, do not apply the bandage as directed above. One bandage can be applied from the periphery to near the lower border of the area of growth or infection, and another, from near the upper border of this area, up the limb. If the bandages are applied in this manner the contents of the diseased area (tumor-cells and fluid or septic prod-

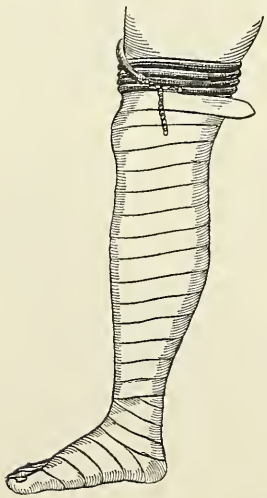


Fig. 944.—Esmarch's elastic bandage.

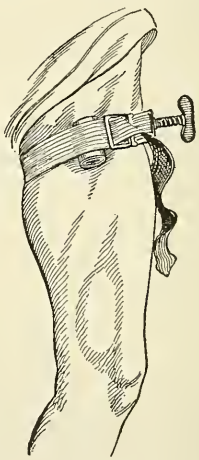


Fig. 945.—Application of tourniquet.

ucts) are not squeezed into the circulation. In cases like the above the best plan is to hold the extremity in a vertical position for five minutes, lightly stroking it toward the body with the hand, and then apply the constricting band. As a matter of fact, this plan satisfactorily empties the limb of blood, and it is not necessary in any case to force the blood out by elastic compression. Some surgeons prefer the tourniquet. Figures 946 and 947 show two forms of tourniquet. To apply Petit's tourniquet, place the plates in contact, apply a small, firm compress over the artery and a broad thick compress over the outer surface of the limb, buckle the tapes around the limb so that the plate is over the broad pad, and tighten the tourniquet by separating the plates by the screw (Fig. 945). When a tourniquet is applied to arrest bleeding during transportation, bandage the limb, sew the compress pad to a bandage, and place the plates of the instrument over the pad. Signorini's horseshoe tourniquet may be used upon the brachial artery. In hip-joint and shoulder-joint disarticulations Wyeth's pins may be passed, and after the limb is emptied of blood the band can be fastened above them. These pins prevent the band from slipping.

The instruments and appliances required for amputation are Esmarch's apparatus or tourniquet, amputating knives (Fig. 948), a bone-knife, scalpels, saws (Fig. 948), a lion-jaw forceps, bone-cutting forceps, a periosteum-elevator, retractors of linen, dissecting, hemostatic, and toothed forceps, a tenaculum, an aneurysm-needle, a probe, scissors, needles, ligatures, sutures of silkworm-gut, dressings, bandages, and solutions. A retractor has two tails for the thigh and arm and three tails for the leg and forearm: it is made by taking a piece of muslin 8 inches wide and 12 inches long and cutting tails on one side 8 inches in length.

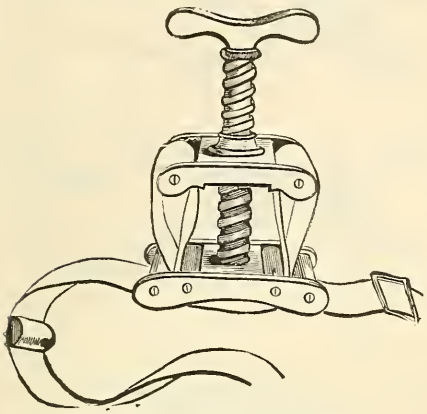


Fig. 946.—Petit's spiral tourniquet.

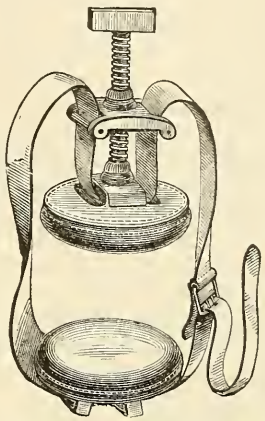


Fig. 947.—Charriére's tourniquet.

**Methods of Amputating.**—The *transverse circular* is the oldest method of amputating. The common circular incision is at a right angle to the axis of the limb. Kocher considers also as a circular incision an oblique cut around the limb if the line of incision "continues in one direction" (Kocher's "Text-Book of Operative Surgery," translated by Harold J. Stiles). This method is called the *oblique circular amputation.* A *racket incision* is formed by adding a longitudinal cut to a transverse circular cut. If the edges are rounded, the *lanceolate incision* is formed. *Rectangular* flaps are

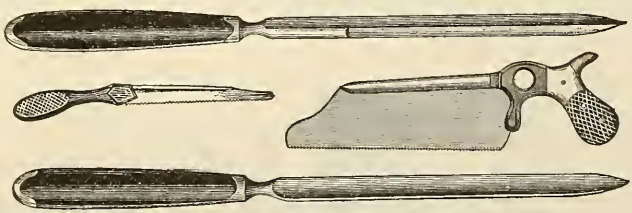


Fig. 948.—Catlin, long knife, and saws for amputation.

formed when two longitudinal incisions are added to a transverse circular cut. If the corners of a rectangular flap are trimmed, *rounded flaps* are formed. The three last-mentioned plans are considered under the head of the Modified Circular Amputation (see page 1404).

**Transverse Circular Method** (Figs. 949–952).—The surgeon should stand to the right of the limb and use a long amputating knife which cuts from heel to point (Fig. 949). After an assistant has retracted the skin the operator divides the soft parts by a series of circular cuts. He does not cut at once to the bone, but divides the skin and subcutaneous tissues. At the retracted edge of the

first cut the superficial muscles are divided, and after these muscles retract the deep muscles are divided. The periosteum is incised by a bone-knife and pushed up by an elevator, and after the application of the retractors the bone is then sawed, the saw starting from heel to point. A periosteal flap can be made to cover the end of the bone, but it is unnecessary. In this amputation is formed a cone whose apex is the bone and whose base is the skin edge. Figs. 950–952, from Kocher, show the steps of the operation and the shape of the resulting stump. In one form of circular amputation (*amputation a la manchette*) the retracted skin is cut by a circular sweep of the knife, a cuff of skin and subcutaneous tissue is freed and turned up, and the muscles are cut circularly at the edge of the turned-up cuff (Fig. 953). The pure circular amputation is performed on the arm and the thigh; the amputation *a la manchette* is performed chiefly through the wrist and the lower forearm.

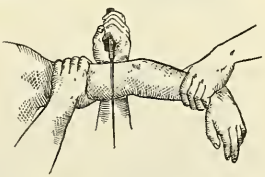


Fig. 949.—Amputation of arm by the circular method (Druitt).

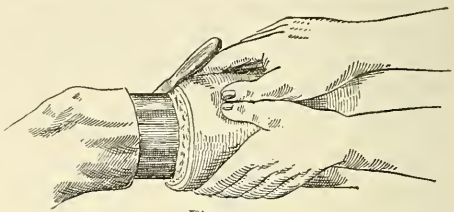


Fig. 950.

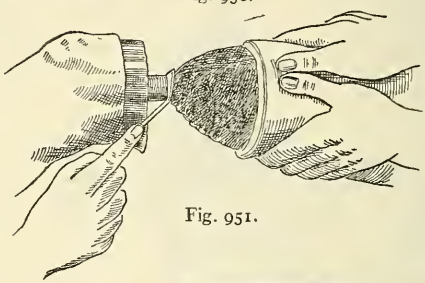


Fig. 951.

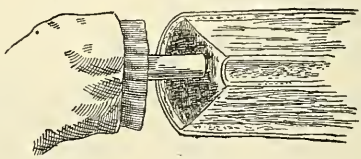


Fig. 952.

Figs. 950–952.—The steps of a transverse circular amputation (Kocher).

If there is more sound skin upon one side of the extremity than upon the other, the transverse circular incision sacrifices more of the limb than is necessary and the oblique circular is preferable. An objection to the transverse

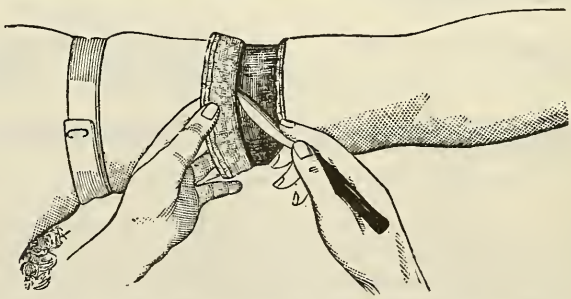


Fig. 953.—Circular amputation: Dissecting up the skin-flap (Esmarch).

circular incision is that the cicatrix lies directly at the end of the stump and is liable to cause pain when subjected to pressure.

**Modified Circular Method.**—In this operation the circular skin-cut may be modified by making a vertical incision to join the first wound, the muscles

being cut by a circular sweep (racket incision) or by making two vertical skin incisions (rectangular flaps). The lanceolate incision is made by round-

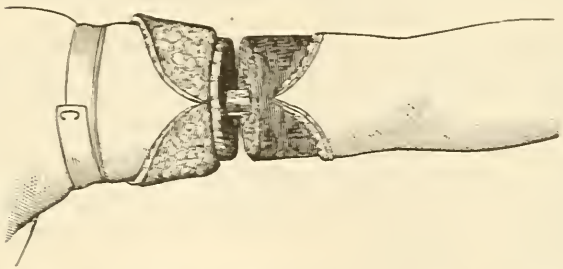


Fig. 954.—Modified circular amputation: Skin-flaps and circular cut through muscles (Esmarch).

ing the edges of the flaps which result from a racket incision. Liston's modification consists in dissecting up two short semilunar integumentary flaps and in dividing the muscles circularly (Fig. 954). This is known as the "mixed method." The modified circular method can be used upon the thigh, the leg, the arm, and the forearm.

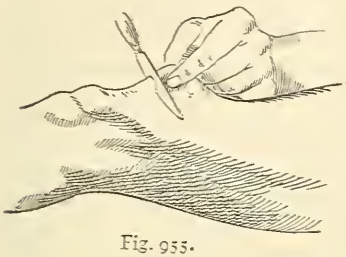


Fig. 955.

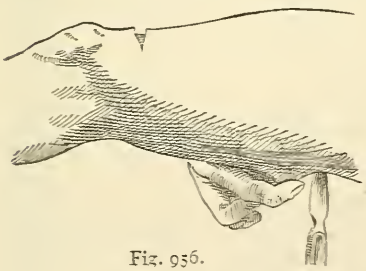


Fig. 956.

**Oblique Circular Method (Elliptical Method).**—Mark the upper and lower ends of the incision as shown in Figs. 955–957. The lowest incision is at a right angle to the cutaneous surface; the highest incision is parallel to the cutaneous surface (Kocher). The skin and fascia are divided so that an oblique incision to the muscles surrounds the limb. The distal elliptical portion of skin is picked up and drawn toward the body and the muscles are divided to the bone, the knife being held transversely (Figs. 955–957). Kocher points out that this flap increases in thickness

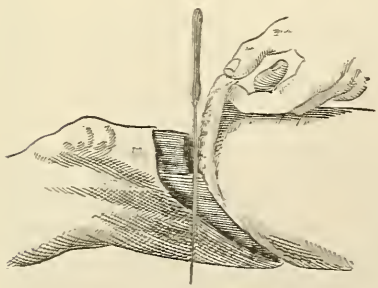


Fig. 957.

Figs. 955–957.—The early steps of an oblique circular amputation (Kocher).

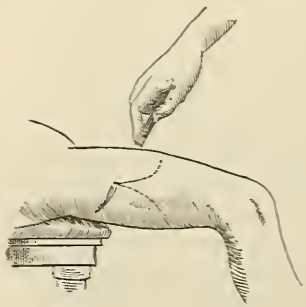


Fig. 958.—Amputation of the thigh by transfixion (Gross).

toward the bone. The rest of the muscles are divided on a level with and in the direction of the skin edge. The periosteum is cut transversely and is

treated as in the transverse circular operation. The flap of muscle and integument is brought over the wound. This method stands midway between the circular operation and the operation by a single flap, and is employed particularly in certain disarticulations.

**Flap Method.**—A flap may be composed of *skin* only or of both *skin* and *muscle*, but the skin-flap must always be longer than the muscle-flap, so that the latter will be covered by it. A flap containing much muscle heals badly, but the best flap has a moderate amount of muscle (enough skin to cover the muscle and enough muscle to cover the bone). Flaps may be *single* or *double*. Double flaps may be *lateral* or *anteroposterior*, *square* or U-*shaped*, *equal* or *unequal*, and they may be cut by *transfixion* (Fig. 958), by cutting from without inward, by dissection, or by cutting the skin from without inward and the muscles by transfixion.

**Racket Method** (if flaps are rounded, is known as the "oval" or "lanceolate" incision).—In an *oval* amputation the incision through the skin and subcutaneous tissue is an oval with a pointed end or a triangle; and the other parts down to the bone are cut from without inward. When a longitudinal incision down to the bone (see Fig. 967, *a–b*) extends from the point of the oval, the operation is called the "racket" amputation. If the longitudinal cut joins a circular cut, the operation is known as a T-amputation. The oval or racket operation is performed at the metacarpophalangeal, metatarsophalangeal, and shoulder-joints; the T-operation may be performed at the hip-joint.

**Completion of An Amputation.**—When an amputation has been completed, tie the main vessels, pull down the nerves and cut them high up, smooth the flaps, take off the constricting band, and after arresting hemorrhage apply sutures. In some cases the deep parts are stitched with a continusous catgut suture and the superficial parts are closed with silkworm-gut; in other cases the deep parts are not stitched at all, the skin alone being sutured with silkworm-gut. Drainage-tubes should be used except in amputations of the fingers and toes.

## Special Amputations

**Fingers and Hand.**—In amputating the thumb and index-finger save every possible scrap of tissue. If it is necessary to amputate a finger above the middle of the middle phalanx, the attachment of the flexor tendons will be cut off and the finger will be liable to project directly backward, so that it is better either to disarticulate at the metacarpal joints or to stitch the flexor tendons to the periosteum. The flexor tendons have fibrous sheaths extending from the proximal end of the distal phalanx to the metacarpophalangeal articulations, these sheaths being thin and collapsible opposite the joints, but being thick and rigid opposite the shafts of the bone. The fibrous sheath is known as the *theca*, and when it is cut in an amputation it should be closed, otherwise it may carry infection to the palm of the hand. The theca does not exist over the distal phalanx, and it is not distinctly visible over the joint between the distal and middle phalanges. To effect closure over the shaft of a bone, strip up the periosteum and pass catgut sutures vertically through the theca and the periosteum (Treves). In amputation of the fingers and the thumb an Esmarch bandage is unnecessary, though pressure may be made upon the arteries at the wrist. Only two or three ligatures are necessary. Close with a very few sutures, so as to favor drainage between the threads.

The distal phalanx is best removed by a long palmar flap (Fig. 959, A). The plamar flap (A) is marked out by cutting through the skin and subcutaneous tissue. The incisions are next carried to the bone, the flap is dissected from the bone, the finger is strongly flexed, a transverse incision (B) is

carried across the dorsum on a level with the base of the third phalanx, the soft parts are pushed back, the joint is opened, the lateral ligaments are cut from within outward, the third phalanx is forcibly extended, and the remaining structures are cut from below upward. Fig. 960 shows the lines of the joints when the finger is flexed. The middle phalanx can be removed by the same method (Fig. 959, C). The proximal phalanx can be removed by a long palmar flap or by a long palmar and a short dorsal flap (Fig. 959, D, E).

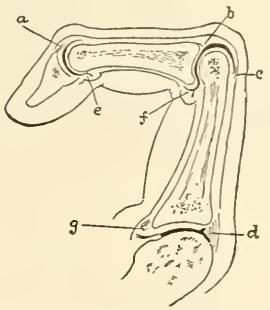



Fig. 960.—The line of the joints in the flexed position of the finger: *a*, Extensor longus digitorum; *b*, interossei and lumbricals; *c*, extensor longus digitorum and interossei; *g*, interossei and lumbricals; *f*, flexor sublimis; *e*, flexor profundus (Kocher).

Fig. 959.—Amputation of the finger.

**Disarticulation at a metacarpophalangeal joint** is best performed by the oval method. The incision upon the dorsum (A) is begun just above the head of the metacarpal bone, is carried down to beyond the base of the phalanx, and involves the skin only (Figs. 961 and 962). One incision sweeps around the finger at the level of the web, going only through the skin (B); the finger is extended and the palmar cut is carried to the bone; each lateral incision is carried to the bone while the finger is bent in the opposite direction, the flaps are dissected back to the joint, the finger is strongly extended, the joint is opened from the palmar side, and disarticulation is effected. Cutting off the head of the metacarpal bone improves the appearance

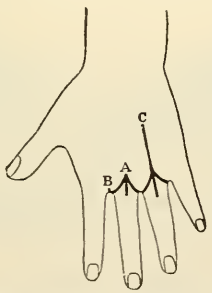



Fig. 961.—A, Disarticulation of a metacarpophalangeal joint; C, amputation of a finger with the metacarpal bone.

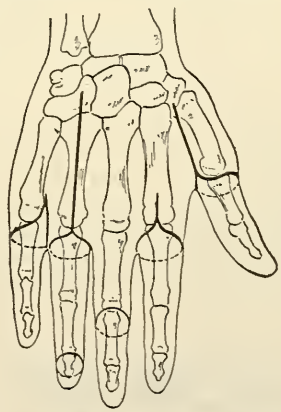


Fig. 962.—Disarticulation of the little finger and index-finger. Disarticulation of the ring finger with its metacarpal bone. Disarticulation of the thumb with its metacarpal bone (Kocher).

of the stump, but weakens the hand, hence in a workingman it must not be done unnecessarily. If it is necessary to remove a metacarpal bone, the incision (C) is made from the carpometacarpal joint.

**Amputation of the thumb** through its distal or proximal phalanx is performed in the identical way employed in amputation of a finger. Amputation of the thumb, with a portion or the whole of its metacarpal bone, is performed by the oval or racket incision (Fig. 962).

**Disarticulation at the wrist-joint** can be done by the oblique circular method (Fig. 963) or by a double flap. In the double-flap amputation a dorsal flap is made by carrying a semilunar skin incision between the styloid processes; the skin is lifted, the wrist is forcibly flexed, the joint is opened by a transverse cut, and a long semilunar palmar flap which includes only the skin and fascia is made by dissection. Kocher prefers to amputate by an oblique incision. The lower end of this incision is about the middle of the palm and the upper end is in the line of the wrist-joint (Fig. 963). The hand is strongly flexed, the extensor tendons are divided, the posterior ligament of

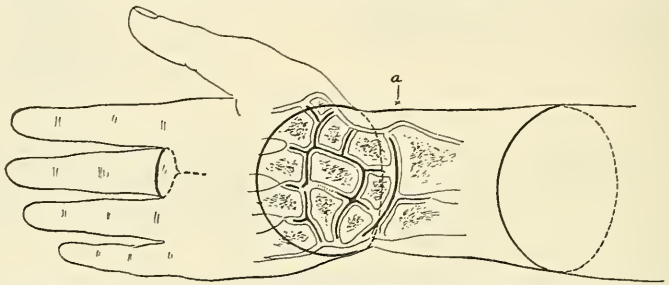



Fig. 963.—Disarticulation of the middle finger. Disarticulation at the wrist-joint. Amputation through the forearm by the oblique circular method (Kocher).

the joint is incised, and incisions below the styloid processes divide the lateral ligaments and certain tendons. The flexor tendons are separated from the bone and are divided so as to remain in the palmar flap.

**Amputation through the forearm** may be effected by the oblique circular method (Fig. 963), the circular, the modified circular, or the flap operation. The modified circular is an excellent plan. A semilunar dorsal skin-flap and a semilunar skin-flap on the flexor surface are made. The flaps are raised, the muscles are cut circularly (Fig. 964), the interosseous space is cleared by the knife, a three-tailed retractor is applied, the periosteum is pushed up, and the bones are sawn $\frac{1}{2}$ inch above the flap. In sawing the bones, start the saw upon the radius, draw it from heel to point, make a furrow on the radius and ulna, and saw both bones at the same time. After sawing, cut away any irregular edge

Fig. 964.—Modified circular amputation of the forearm (Bryant).

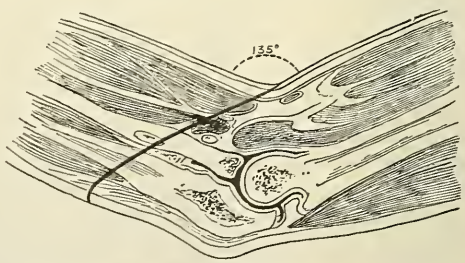



Fig. 965.—Disarticulation of the elbow-joint by the oblique circular method (Kocher).

with bone-pliers. In the lower third Teale's amputation may be done, the dorsal flap being the long one. In Teale's amputation rectangular flaps are made. The long flap is equal in width and length to one-half the circumference of the limb at the point where it is to be sawn. The short flap is equal in width to the long flap, but is only one-fourth its length. The two longitudinal cuts are at first taken only through the skin, but the two transverse cuts go at once to the bone. The flaps are dissected up from the interosseous membrane and the bone. In the middle or the upper third of a fleshy arm two semilunar

skin-flaps can be cut from without inward, and the muscles can be cut by transfixion.

**Disarticulation at the elbow-joint** can be done by the elliptical method or by a long anterior and short posterior flap. In Kocher's oblique operation the incision begins anteriorly over the joint-line and ends posteriorly a hand's breadth below the summit of the olecranon (Fig. 965). A posterior flap which contains the integument, the insertion of the triceps, the anconeus, and the periosteum is dissected up until the posterior surface of the humerus is reached. The joint is opened anteriorly by a transverse incision, and the radiohumeral articulation is opened from without inward (Kocher). In the double flap operation the forearm is partly flexed and a skin-cut marks out a long anterior flap, the knife being entered opposite the external condyle and being withdrawn 1 inch below the internal condyle. The muscles, which are bunched forward, are cut by transfixion. A posterior semilunar flap is made, which separates the attachments of the radius, the ulna is cleared, and the triceps is cut at its insertion (Bell). Gross advocated sawing through the olecranon and the inner trochlear surface.

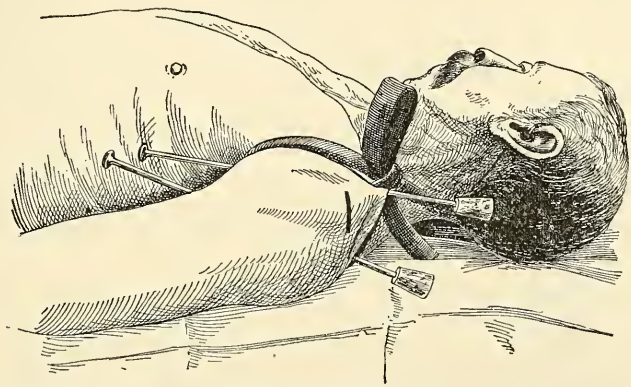


Fig. 966.—Use of Wyeth's pins in amputation at the shoulder-joint. The acromion is marked by a black line (Keen).

**Amputation of the arm** is best performed by marking out with a knife two equal semilunar anteroposterior flaps, the first cut being carried through the skin alone, the muscles being then transfixed with a long knife. Teale's method is shown in Figs. 438 and 439. The circular or the modified circular amputation may be performed.

**Disarticulation at the Shoulder-joint.**—In this operation some surgeons use Wyeth's pins to hold the Esmarch band in place. The anterior pin is entered at the middle of the lower margin of the anterior axillary fold, and emerges 1 inch within the tip of the acromion. The posterior pin is entered at a corresponding point on the posterior axillary fold, and emerges more posteriorly than the first pin and an inch within the tip of the acromion. After the extremity has been drained of blood by the Esmarch bandage or by stroking and a vertical position, the Esmarch band is applied above the pins (Fig. 966). With a competent assistant, however, the pins are not necessary, the surgeon divides his main vessels as the last step of the operation, and the assistant controls them, before they are cut and until they are tied, with his thumbs slipped back of the bone.

**Larrey's Operation.**—In this method of shoulder-joint disarticulation the limb is held from the side and an incision is made down to the bone, the incision beginning just below and in front of the acromion and running vertically for

4 inches down the outer surface of the arm (Fig. 967, *a–b*). From the center of this incision an oval incision (*c–d*, *c–e*) is carried around the arm, the inner aspect of the oval reaching as low as the lower end of the vertical cut. The oval incision at first involves only the skin and subcutaneous tissues. The anterior structures are divided close to the bone, and the posterior structures are next cut. To disarticulate, cut the capsule transversely upon the head of the bone; while the arm is rotated outward cut the subscapularis, and while the arm is rotated inward cut the supraspinatus and infraspinatus and the teres minor. Cut away any tissue holding the humerus to the body, hanging nerves, capsule-fragments and tissue-shreds, insert a tube, and sew up the wound vertically. Bell advises an oval incision with a racket handle. Spence used an anterior racket incision.

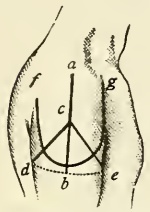



Fig. 967.—Amputation at the shoulder-joint: *a–b*, *c–d*, *c–e*, Larrey's operation; *f–g*, Dupuytren's operation.

**Kocher's Operation.**—Kocher makes an anterior lanceolate incision (Fig. 968). The incision begins over the clavicle just external to the coracoid process of the scapula, and is carried downward, dividing, as it advances, the anterior fibers of the deltoid muscle. "Bleeding vessels and the cephalic vein are ligatured. In the upper part of the wound the acromial branches of the acromiothoracic artery are also ligatured. The knife is carried down to the bone at the edge of the deltoid (only the upper fibers of which have been divided). The capsule is divided over the lesser tuberosity and the bicipital groove. The periosteum, the insertions of the subscapularis, pectoralis major, latissimus dorsi, and teres major are detached along with the capsule. The capsule, along with the insertions of the supraspinatus, infraspinatus, and teres minor muscles, is also detached from the upper part of the head

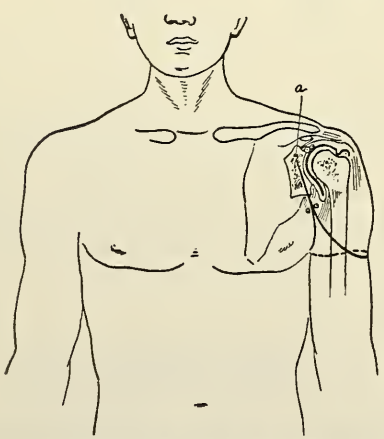



Fig. 968.—Disarticulation at the shoulder-joint by Kocher's method (Kocher).

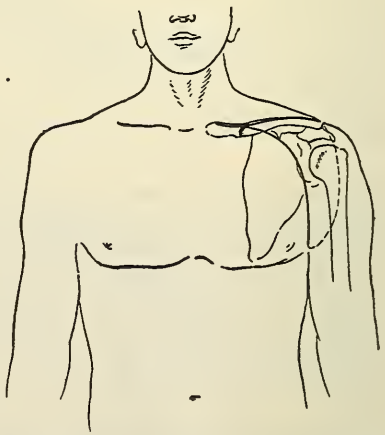


Fig. 969.—Removal of the entire upper extremity (Kocher).

and from the great tuberosity. The head of the humerus can now be protruded from the wound. In cutting down over the surgical neck it may be necessary to ligature the circumflex arteries; in any case the anterior vessel must be tied. The racket incision is now completed by dividing the skin circularly at the level of the axillary folds. The vessels and nerves are then easily isolated, the former being ligatured and the latter divided" (Kocher's "Text-Book of Operative Surgery," translated by Harold J. Stiles). Kocher

cautions us to avoid the circumflex nerve which supplies the deltoid, as the deltoid is the muscle of the stump.

**Dupuytren's Operation.**—In Dupuytren's shoulder-joint disarticulation a U-shaped flap is marked out by a skin-incision (Fig. 967, *f–g*). If the amputation is to be at the right shoulder, the arm is carried across the chest; the knife is entered at the root of the acromion, follows the margin of the deltoid, and is withdrawn at the coracoid process, the arm being gradually abducted and pulled off from the chest. If the left shoulder is to be amputated the procedure is reversed (Treves). The knife next cuts through the deltoid and raises a flap composed of this muscle, the shoulder-joint is exposed, and disarticulation is effected as in Larrey's method. The knife is passed down back of the bone and a short internal flap is cut.

**Lisfranc's amputation** is by transfixion with the formation of an anterior and a posterior flap, and can be performed very rapidly by a skilful surgeon.

**Amputation of the Entire Upper Extremity.—Berger's Operation.**—*The Interscapulothoracic Amputation.*—This operation, which is an amputation above the shoulder-joint, was described by Berger in 1887. By it are removed the arm, the scapula, and a portion of or the entire clavicle. It is occasionally employed in cases of malignant disease and of severe injury. The operation is attended with profuse hemorrhage, and as a preliminary the subclavian vessels should be ligated. The incisions must be varied according to the necessities of the case. In this operation Berger divides the clavicle at the junction of its outer and middle thirds, and resects the middle third of the bone; ligates and divides the subclavian vessels; cuts the anterior flap; divides the brachial plexus; marks out the posterior flap; and completes the operation by dividing the structures which hold the shoulder-blade to the chest. It is in this last step that bleeding is profuse. Figure 970 shows Berger's incisions for the operation. Figure 969 shows Kocher's incisions.

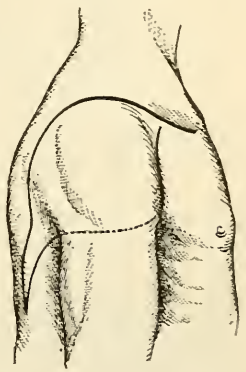


Fig. 970.—Removal of the whole upper extremity.

The usual procedure of tying the third part of the subclavian artery as a preliminary measure possesses certain disadvantages. The artery is very deeply situated at this point, is in close relation with the pleura, and is covered to a considerable extent by the vein, and the phrenic nerve is very near. Le Conte resects the entire clavicle before tying the vessels. He maintains that then one of two courses may be taken: The veins may be severed first, and afterward the artery may be exposed and tied. When this is done, the amount of blood remaining in the arm is lost. The procedure that he selects as the best, however, is to expose the axillary artery as high up as possible, and place a temporary ligature around it; then elevate the arm, empty it of blood, place a permanent ligature around the third part of the subclavian artery, and divide the artery in this portion of its course (Robert G. LeConte, "Annals of Surgery," Oct., 1902). If the scapula is involved in the tumor, the mortality is something over 23 per cent. (Berger, "Revue de Chir.," Aug., 1905). I have twice performed the operation successfully and in each case followed LeConte's plan.

**Amputation of the Toes and the Foot.**—Only through the great toe is *partial* amputation performed, and it is effected by the formation of a long plantar flap, just as a long palmar flap is formed from a finger. Amputation at a metatarsophalangeal joint is performed by an oval or racket incision (Fig. 971, *c*, *c*). Amputation of a toe with removal of its metatarsal bone is shown in Fig. 971, *a–b* and *d–e*.

**Disarticulation at the Tarsometatarsal Articulation.—Lisfranc's Operation** (*after Treves*).—In order to amputate the right foot by this method begin an incision on the outer border of the foot, behind the tubercle of the fifth metatarsal bone; carry the incision forward 1 inch and sweep it across the foot $\frac{1}{2}$ inch below the tarsometatarsal articulations; bring the incision to the inner edge of the foot, $\frac{1}{2}$ inch in front of the articulation of the tarsus with the first metatarsal bone, and carry the cut straight back along the inner margin of the foot until it reaches a point $\frac{3}{4}$ inch above the articulation of

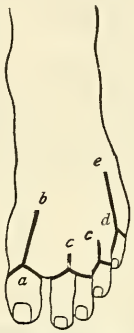



Fig. 971.—Amputation of the toes with and without the metatarsal bones.

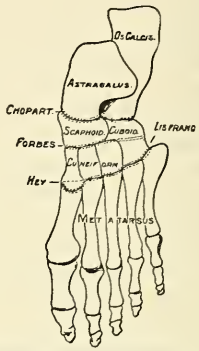



Fig. 972.—Lines in amputations of the foot (Gross).

the metatarsal bone of the great toe. A very short semilunar dorsal skin-flap is thus formed. Figure 977 shows the flaps as cut by Kocher. After the skin-flap has been dissected back for $\frac{1}{4}$ inch the tendons are divided, and the flap, which now contains all the soft parts, is dissected back to *above* the joint. A long plantar flap is cut, reaching from the origin of the first flap to the necks of the metatarsal bones. The skin-flap is dissected up until the hollow behind the heads of the metatarsal bones is reached, when, with the toes in extension, the tendons are cut across and a flap composed of all the soft parts is dissected up to above the tarsometatarsal joint. Figures 972 and 977 show the line of

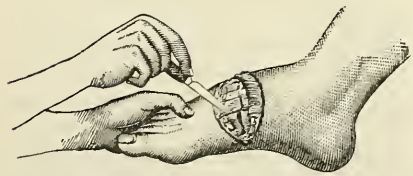


Fig. 973.—Lisfranc's amputation: First step in disarticulating the second metatarsal bone (Guérin).

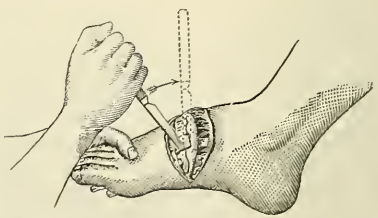


Fig. 974.—Lisfranc's amputation: Second step in disarticulating the second metatarsal bone (Guérin).

Lisfranc at the tarsometatarsal articulation. The joint is opened from the outer side according to the following rule: in separating the fifth metatarsal direct the edge of the knife toward the distal end of the first metatarsal; in separating the fourth metatarsal direct the knife toward the middle of the first metatarsal; in separating the third metatarsal carry the knife almost directly across. The separation is facilitated by bending down the front of the foot, and at the same time the tendons of the peroneus brevis and tertius are divided. Open the joint between the first metatarsal and the inner cuneiform

bone, turning the knife toward the middle of the shaft of the fifth metatarsal, and at the same time divide the tibialis anticus muscle. Treves says that in disarticulation of the second metatarsal the knife is to be held as a trocar, it is to be thrust between the base of the first and second metatarsal bone until the point strikes bone (Fig. 973), and is then to be raised to a perpendicular and the cut is to be made toward the external malleolus to sever the ligament of Lisfranc (Fig. 974). Divide any remaining ligaments, and also the tendon

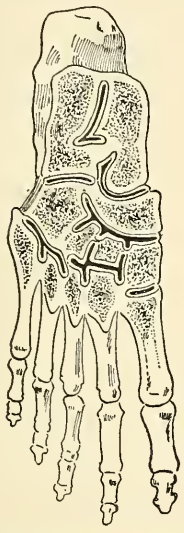


Fig. 975.—Anterior intertarsal disarticulation (Kocher).

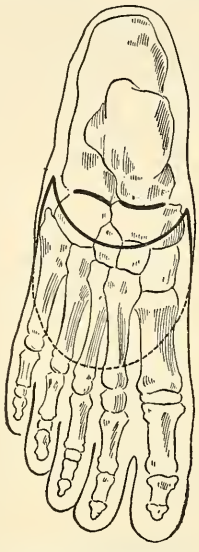


Fig. 976.—Chopart's amputation (Kocher).

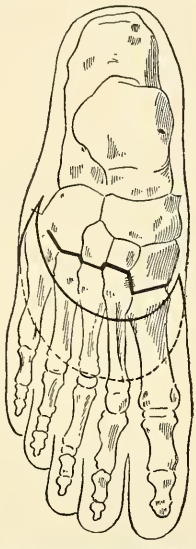


Fig. 977.—Lisfranc's amputation (Kocher).

of the peroneus longus muscle. The skin-incisions in the *left* foot are begun on the inner side, and in disarticulating the tarsal joint of the great toe is first opened. Figure 978 shows the parts after disarticulation at the line of Lisfranc.

**Hey's Operation.**—In Hey's method the incision is practically the same as that for Lisfranc's amputation. The four external metatarsal bones are

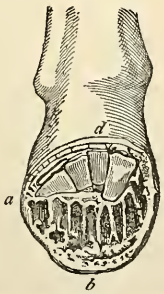



Fig. 978.—The parts after Lisfranc's amputation (Bernard and Huette).

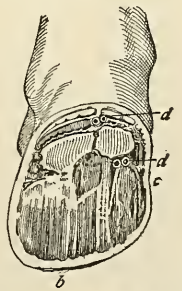



Fig. 979.—The parts after amputation by Chopart's method (Bernard and Huette).

disarticulated, but the first metatarsal is removed by sawing a portion of the internal cuneiform bone. Guérin advised sawing all the bones across. Skey advised the division of the head of the second metatarsal. Figure 972 shows the line of Hey.

**Anterior Intertarsal Disarticulation (Amputation of Forbes, of Toledo).**—The disarticulation is effected between the three cuneiform bones in front and the scaphoid behind, and the cuboid is sawn across. Figure 972 shows the line of Forbes. The incision of the soft parts is as for Lisfranc's amputation (Fig. 975).

**Disarticulation Through the Middle Tarsal Joint.—Chopart's Operation** (*Posterior Intertarsal Disarticulation*).—Make a transverse incision through the skin of the instep, 2 inches below the ankle-joint; cut the tendons and muscles, expose the tarsus, and make on each side a small longitudinal incision reaching to below and in front of the corresponding malleolus. The flap thus formed is retracted. The plantar flap is made as in Lisfranc's amputation. The flaps as made by Kocher are shown in Fig. 976. Open the astragaloscaphoid joint, then the calcaneocuboid joint, and disarticulate. Figures 972 and 976 show the line of Chopart. Figure 979 shows the parts after Chopart's disarticulation.

**Subastragaloid Disarticulation.**—A circular incision is carried around the foot at the level of the middle tarsal joint and a racket incision is added to it running below and posterior to the tip of the external malleolus (Fig. 980). "The joint between the astragalus and scaphoid is opened upon the dorsum, without opening the calcaneocuboid joint. A narrow knife is then passed backward and slightly upward beneath the head of the astragalus so as to divide the strong interosseous ligament between it and the os calcis. The soft parts are then dissected off the os calcis, first from its upper surface, then from its outer and under surfaces, and lastly from its inner and posterior surfaces. The greatest difficulty is met with at the inner side in clearing the projecting sustentaculum tali" (Kocher's "Text-Book of Operative Surgery," translated by Harold J. Stiles).

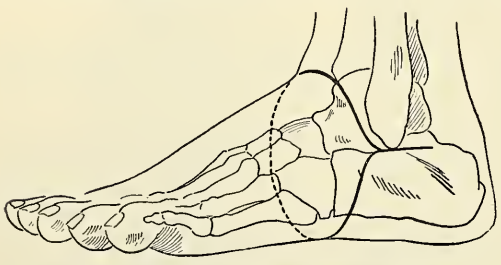


Fig. 980.—Subastragaloid disarticulation (Kocher).

**Disarticulation At the Ankle-joint.—Syme's Method.**—The foot is held at a right angle to the leg, and a skin-incision is carried, from just below the external malleolus, straight across or a little backward across the sole to a corresponding point on the opposite side. Do not take this incision near to the inner malleolus, as to do so will endanger the posterior tibial artery. The incision is carried to the bone, the flap being pushed back and separated from the bone by means of a strong knife and the thumb-nail until the tuberosity of the os calcis has been reached. The foot is now extended and a transverse cut is made across the dorsum, joining the two ends of the first incision; the ankle-joint is opened, the lateral ligaments are cut, disarticulation is effected, and the foot is finally completely removed by severing the tendo Achillis. A thin piece of bone including both malleoli is sawn from the tibia and fibula. The flap is perforated posteriorly to secure drainage (Fig. 438).

**Pirogoff's Method.**—Flex the foot to a right angle with the leg. "Make an incision from the tip of the internal malleolus across the sole, a little in front of the long axis of the tibia, to a point in front of the apex of the external malleolus down upon the bone."[1] Dissect the flap backward from the calcaneum for $\frac{1}{4}$ inch, but do not dissect the flap from the posterior portion of the

[1] "Operative Surgery," by Joseph D. Bryant.

os calcis. Join the extremities of the first incision by another cut which reaches to the bone, and which is "½ inch in front of the lower extremity of the tibia" (Bryant); but saw off this bony projection obliquely and leave it adherent to the tissues. The saw is used after disarticulation of the ankle-joint; it is passed behind the astragalus, cutting downward and forward, sawing the os calcis obliquely, and leaving a considerable portion in place in the flap. The lower ends of the tibia and fibula are well exposed by raising the anterior flap slightly; the sawing is begun anteriorly just above the articular surface, and is completed ½ inch above the articular surface posteriorly. The lines *a* and *b* (Fig. 981) show the sections made by the saw. The sawn surface of the os calcis is brought into contact with the sawn surfaces of the tibia and fibula, and the flaps are sutured.

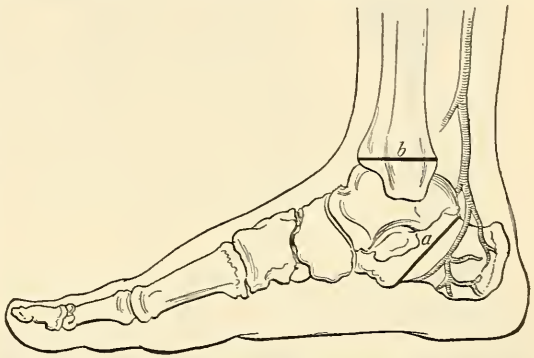


Fig. 981.—Lines of section of the os calcis and the bones of the leg in Pirogoff's amputation.

**Amputations of the Leg.**—The so-called "point of election" is at the upper part of the middle third of the leg. Seventy-five years ago Liston advised surgeons not to amputate in the lower third of the leg because of the scantiness of the soft parts, because the stump is apt to ulcerate, and because it is uncomfortable in an artificial leg. These views have been much modified. The amputation near the ankle is safer than the amputation near the knee, and artificial legs are now made which may be worn with comfort. In amputations of the leg by the *long anterior flap*, cut through the skin, dissect up the anterior muscles with the flap, and cut all the posterior tissues with a single transverse sweep. Amputation by the *rectangular flap*, Teale's method, is very useful (see page 1408). The long flap is anterior, and is in length and breadth equal to one-half the circumference of the limb. The short flap is one-fourth the length of the long flap. The flaps are dissected up, the bones are sawn, the long flap is turned upon itself, and its edges are sutured to the edges of the short flap.

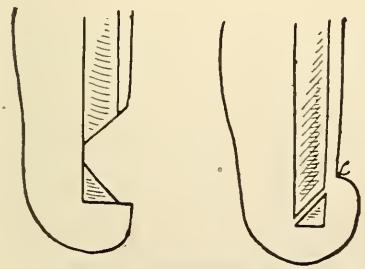


Fig. 982.—Diagrammatic representation of amputation of the leg after the method of Bier.

Bier suggests a plan (Fig. 982) to increase the supporting power of the stump after a leg amputation. After the wound has healed, a wedge-shaped piece of bone is removed above the level of the stump. The lower extremity is turned forward and upward through an arc of 90 degrees, and unites in this position (Zuckerkandl's "Operative Surgery"). Thus the medullary cavity is closed and the skin which must bear pressure is healthy and free from cicatrices; and as the muscles are still attached to the bone, they do not undergo atrophy.

**Sédillot's leg amputation** (Fig. 983) is by a long external flap. A longitudinal incision is made along the inner edge of the tibia, the tissues are drawn toward the fibula, a knife is introduced and passed to the outer edge of the tibia, just touching the fibula, and is brought out posteriorly, thus transfixing the calf-muscles and cutting an external flap. A convex incision is made on

the inner side, the bones are cleared and are sawn 1 inch above the flaps, ½ inch more being taken from the fibula than from the tibia, and the tibia being bevelled anteriorly.

**Modified Circular Amputation of the Leg.**—Cut semilunar skin-flaps, lay them back, and cut circularly to the bone at the edge of the turned-up flap. Another method of modified circular amputation is by adding to the circular cut a vertical incision down the front of the leg. In sawing the bones of the leg the surgeon, who stands to the outer side of the right leg or to the inner side of the left leg, divides the fibula first, and at a higher level than the tibia, and bevels the anterior surface of the tibia. In sawing the left fibula the saw points to the floor; in sawing the right fibula it points to the ceiling.

Fig. 983.—Sédillot's amputation of the leg (Wyeth).

**Amputation of the Leg by a Long Posterior and a Short Anterior Flap.**—In this operation a posterior U-shaped flap is made equal in length and breadth to the diameter of the limb. The skin-incision is begun 1 inch below the point where the bone is to be sawn, and behind the inner edge of the tibia, and is carried to a point posterior to the peronei muscles. The gastrocnemius muscle is divided transversely at the level of the flap, the soft parts on either side in the line of the flap being cut to the bone. Through these vertical cuts the muscles are lifted from the bones and are divided through their lower part by cutting from within outward. The anterior flap is formed by making a semilunar skin-flap and by cutting the muscles across at its retracted edge (Fig. 984).

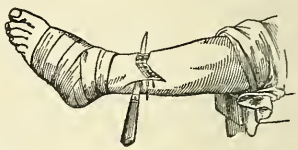


Fig. 984.—Amputation of the leg by a long posterior flap (Gross).

*Amputation of the leg by lateral flaps* is not a popular operation, as it offers too much encouragement to subsequent protrusion of the bone.

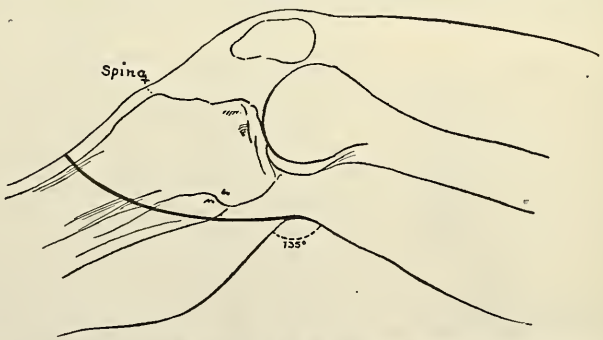



Fig. 985.—Kocher's oblique incision for disarticulation at the knee-joint (Kocher).

**Amputation Just Below the Knee.**—The seat of election is 1 inch below the tuberosities. No muscle is needed in the flap. Cut two flaps of skin, equal in size and of semilunar shape, these flaps beginning anteriorly 2 inches below the tuberosity of the tibia. One flap is antero-external and the other is postero-internal. The flaps are pulled up, the anterior muscles are cut as high up as possible, and the posterior muscles are cut through the middle of the portion exposed (Bell). The bone is sawn 1 inch below the tuberosity.

**Disarticulation At the Knee.**—In disarticulation by the long anterior flap, make a long anterior skin-flap, incise the ligament of the patella, turn up a flap containing the patella, open the joint, and complete the disarticulation by cutting from within outward and downward. The knee may be disarticulated by means of a long anterior and a short posterior flap. Kocher prefers the oblique incision (Fig. 985). This secures an anterior flap. The leg is so held that it makes an angle with the thigh of 135 degrees and "the incision falls in the continuation of the long axis of the thigh" (Kocher's "Text-book of Operative Surgery," translated by Harold J. Stiles). The posterior part of the incision is opposite the line of the joint and the anterior part of the incision ends four fingers-breadth below the tibial tubercle.

**Amputation Through the Femoral Condyles.**—*Syme's Method by a Long Posterior Flap.*—Carry a skin-incision, with a very slight downward curve from one condyle to the other, across the middle of the patella. Cut down to the bone, retract the flap, and cut the quadriceps above the patella. Insert a long knife at one angle of the wound, pass it back of the femur, and make it emerge at the opposite angle, cutting a posterior flap 8 inches long. Retract the posterior flap, clear for sawing, and section the condyles horizontally. Carden made a curved section of the condyles at their widest part. In children Buchanan showed that we can easily separate the lower femoral epiphysis.

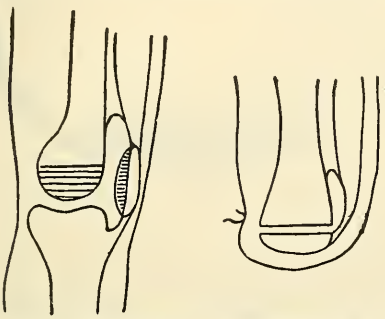


Fig. 986.—Diagrammatic representation of Gritti's operation.

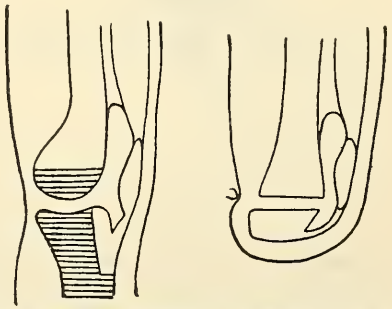


Fig. 987.—Diagrammatic representation of Sabanejeff's operation.

In *Gritti's supracondyloid amputation* an oblique incision is made. The upper end of the incision is posterior and just above the condyles. Its lower end is anterior and two fingers-breadth below the patella (Kocher). The ligament of the patella is cut, the flap is turned up, the femur is sawn at the base of the condyles, the articular face of the patella is sawn off, and the sawn patella is fastened to the sawn femur and the flaps are sutured (Fig. 986). Sabanejeff makes an anterior flap, opens the knee-joint from behind, saws the condyles at their broadest part, takes a bone-flap from the anterior portion of the tibia, and fastens it to the femur (Fig. 987).

**Amputation of the Thigh.**—In high amputation in the *lower third* either a flap or a circular operation may be performed. In a double-flap operation a semilunar skin-incision should be made from without inward, and the muscles should be cut by transfixion (Fig. 988). In the lower third Teale's flap or the long anterior flap may be employed. The amputation by a long anterior flap consists in making a lengthy skin-flap, reflecting it, cutting the anterior structures to the bone, again entering the long knife at one angle of the incision, pushing it back of the femur, bringing it out at the outer angle, and cutting the structures behind the bone directly backward. Bell amputates by a long anterior semilunar flap and a short posterior flap. In amputations in the *upper two-thirds* of the thigh the best plan is to mark out equal anterior and posterior

semilunar skin-flaps, divide the skin with a scalpel, enter the long knife at one angle of the anterior flap, bring it out at the other angle, and cut the muscles by transfixion. Cut the posterior flap in the same manner. Some surgeons prefer a long anterior semilunar flap and a short posterior semilunar flap. The pure circular amputation is not adapted to the thigh.

**Disarticulation At the Hip-joint.**—Various methods have been employed to prevent or limit hemorrhage during this formidable operation. Abernethy used digital compression of the external iliac artery or of the femoral artery. This is an extremely tiresome procedure; the finger is liable to slip; and, in any case, compression so situated fails to intercept the blood-current in a number of large vessels.

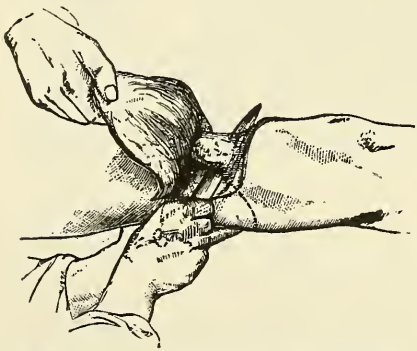


Fig. 988.—Amputation of the thigh (Bryant).

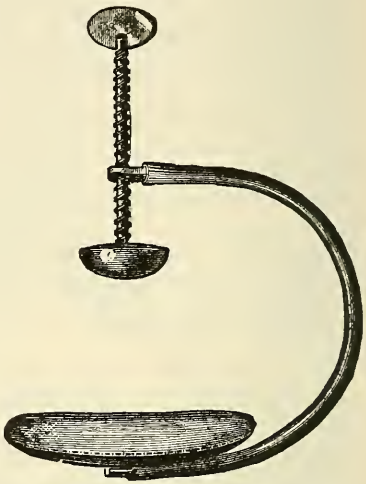


Fig. 989.—Pancoast's aorta tourniquet.

Various other methods have been employed. It was formerly the custom to compress the aorta by means of an abdominal compressor (Figs. 989, 990). A tourniquet is very likely to be displaced during the operation. The intention is to compress the artery against the spine, but in effecting this the circulation in a portion of the intestine may be impaired. In any case, as Senn says, the

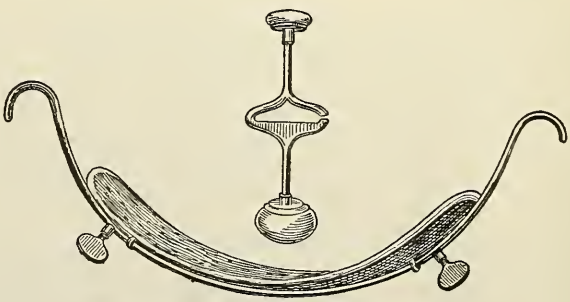


Fig. 990.—Von Esmarch's aorta tourniquet.

circulation is cut off from half the body, and the patient is exposed to grave danger from "sudden vascular engorgement of important internal organs" (Senn). Again, an abdominal compressor of this sort does not arrest venous bleeding. A number of years ago Davy suggested that a suitable cylindrical piece of wood, about 25 inches long, and shaped like a cone at the end, might be introduced into the rectum and used to compress the common iliac artery

upon the pelvic brim. This appliance is known as *Davy's lever*. It is apt to slip, and may do serious damage to the rectum.

Some surgeons have practised preliminary ligation of the common femoral artery or of the external iliac artery, and others have tied the vessels while making the flaps. I employed preliminary ligation of the common femoral with perfect satisfaction in 2 of my 4 cases of amputation at the hip-joint for sarcoma of the femur. If any form of compression is used, that recommended by Macewen, of Glasgow, is the most successful and satisfactory (Fig. 991). The weight of the assistant's body is thrown upon the patient's aorta by the right fist, placed slightly to the left of the umbilicus. McBurney has suggested the prevention of bleeding by making a small abdominal incision and having an assistant make direct digital pressure upon the iliac artery. I employed McBurney's method in one case and found it most satisfactory. In this case a sarcoma of the thigh reached up so far that no band could be applied above

Fig. 991.—Macewen's method for compression of the abdominal aorta ("American Text-Book of Surgery").

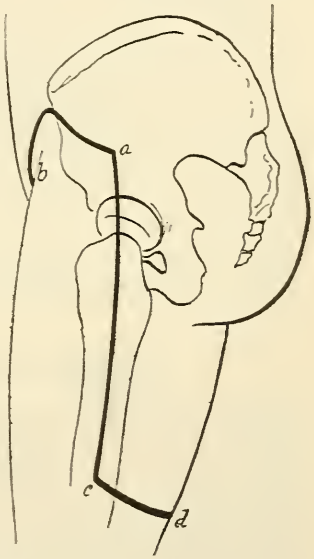



Fig. 992.—Posterior flap in author's unusual case requiring hip-joint amputation: *a–b*, The anterior incision; *a–c–d*, the external incision and the beginning of the posterior cut.

it and I was obliged to make the posterior flap shown in Fig. 992. If the constricting band of Esmarch is applied by the ordinary method, it is certain to slip. It may remain in place if applied as a figure-of-8 of the thigh and the pelvis, but even then it is uncertain.

A satisfactory method in many cases is Wyeth's, in which the constrictor is held in place by the preliminary passage of two steel pins (Fig. 993). Trendelenburg's method consisted in passing one pin and winding an elastic tube about it. Wyeth applied the principle and greatly improved the method. The outer pin is inserted 1½ inches below and a little internal to the anterior superior spine of the ilium, and is brought out just back of the great trochanter. The inner pin is entered 1 inch below the level of the crotch and internal to the saphenous opening, and it emerges 1½ inches in front of the tuberosity of the ischium. A sterile cork may be pushed on the end of each pin, to save the surgeon from wounding himself upon the sharp points. A cork is

apt to come off during the course of the operation. Because of the insecurity of the cork I have had made pins with removable points. After a pin has been passed, the point is unscrewed and a knob is screwed on in its place. After the limb has been emptied of blood by holding it in a vertical position for five minutes and stroking it from the periphery toward the body, the constricting band is fastened about the limb above the pins.

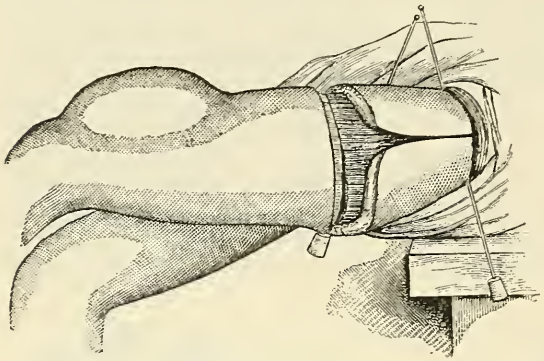


Fig. 993.—Amputation at the hip-joint: Wyeth's bloodless method.

In the *bloodless method of Wyeth* (Figs. 993, 994), after passing the pins, draining the limb of blood, and applying the band of the Esmarch apparatus, the amputation is proceeded with. The hip is brought well over the edge of the table, a circular incision is made down to the deep fascia, 6 inches below the constricting band, and is joined by a longitudinal skin-cut reaching from the band to the level of the circular incision, and the cuff is reflected to the level of the lesser trochanter. The muscles are cut by a circular sweep at the

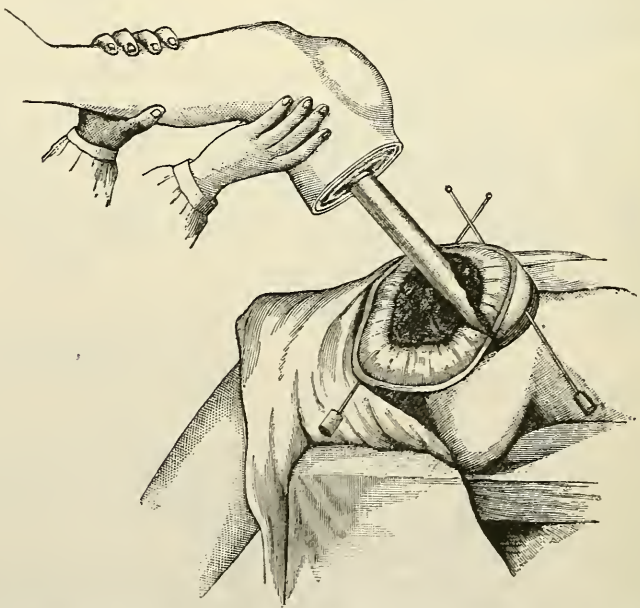


Fig. 994.—Wyeth's bloodless amputation at the hip-joint: Cuff of skin and subcutaneous fat turned back, muscles divided at level of small trochanter, bone partly stripped, and large vessels exposed for deligation.

level of the retracted cuff, the capsule of the hip-joint is opened freely, the cotyloid ligament is cut posteriorly, the thigh is bent upward, forward, and inward to dislocate the head of the bone, and, using the thigh as a handle, the round ligament is incised and the limb removed. After ligating the vessels

and introducing drainage-tubes the flaps are sewn together vertically. The old transfixion operation is practically extinct. A T-*amputation* may be

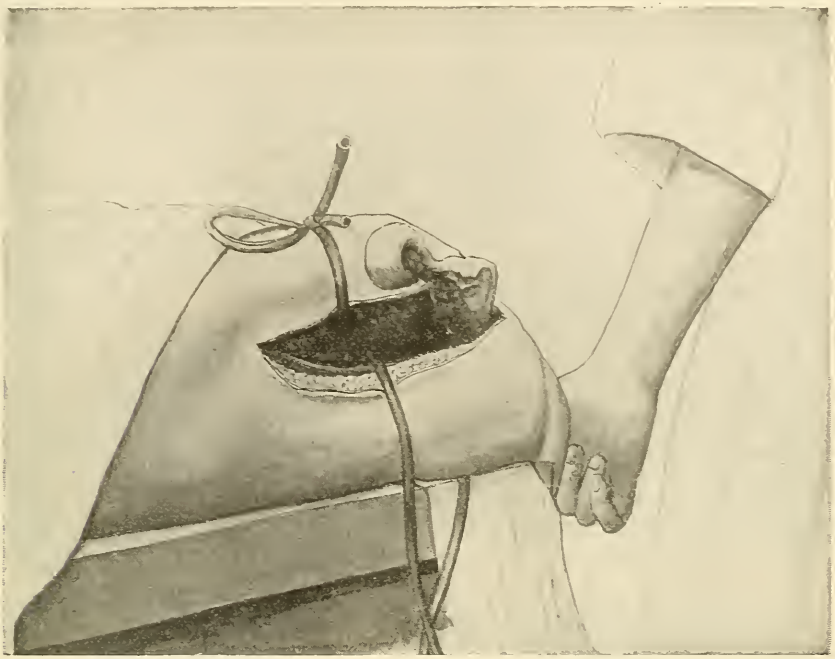


Fig. 995.—Senn's method of performing bloodless amputation at the hip-joint. Dislocation of head of femur and upper portion of shaft through straight external incision. Elastic constrictors in place, the anterior one tied (Senn).

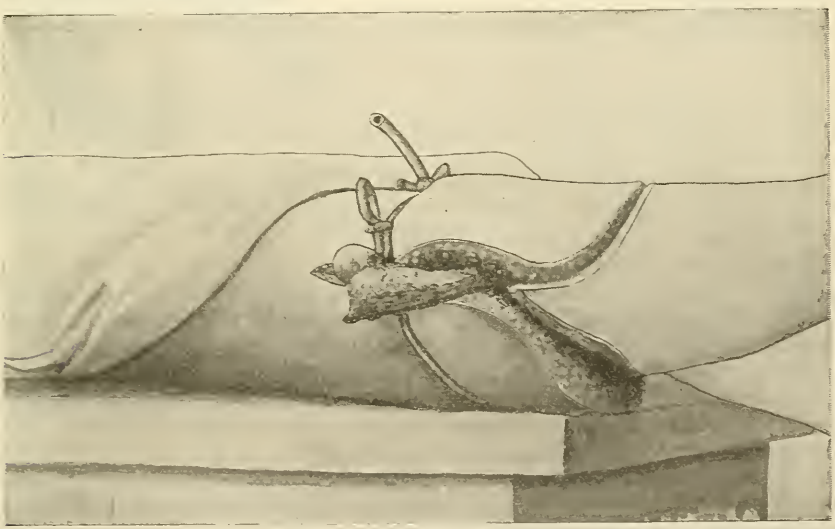


Fig. 996.—Elastic constriction completed by constricting the posterior segment of the thigh. Flaps formed, including all the tissues down to the muscles (Senn).

employed. It consists of an external straight incision down to the bone, starting over the great trochanter, down the outer side of the limb, and a

circular incision through the skin 5 inches below the constricting band, the muscles being cut by a circular sweep at the level of the retracted skin. This method affords easy access to the joint. The bloodless method of Wyeth, as applied to the hip-joint and shoulder-joint, is a notable advance in the art of surgery.

*Senn's Bloodless Method.*—The elder Senn has devised a method for preventing hemorrhage during amputations of the hip-joint. He makes a straight incision, about 8 inches in length, in the direction of the long axis of the femur and directly over the center of the great trochanter. This incision reaches about 3 inches above the upper margin of the great trochanter. The muscular insertions are divided close to the bone, and the thigh is flexed, strongly adducted, and rotated inward. The capsular ligament is divided at its upper and posterior aspect. While the thigh is brought into a position of slight flexion, the remaining portion of the capsular ligament is cut. Then the thigh is dislocated outward, and the ligamentum teres is cut. If this cannot be accomplished, the head of the bone is forcibly dislocated upon the dorsum of the ilium. After dislocating, the lesser trochanter and the upper part of the femoral shaft are cleared. The limb is now brought down in a straight line with the body, the thigh is slightly flexed, a long and stout pair of forceps is inserted into the wound behind the femur and on a level with the normal situation of the lesser trochanter, and the instrument is pushed downward and inward, 2 inches below the ramus of the ischium and just behind the adductor muscles. As soon as the point can be felt under the skin, an incision 2 inches in length is made upon it, and the instrument is forced through the opening. The tunnel in the tissues is enlarged by opening the forceps. A piece of rubber tubing $\frac{3}{4}$ inch in diameter and 4 feet in length is caught about the middle with the forceps and is withdrawn. The rubber tube is cut in two at about the point at which the forceps have held it, and half of the tube is used to constrict the anterior segment of the thigh (Fig. 995) and the other half to constrict the remaining portion of the thigh (Fig. 996). Before the constricting bands are tied the limb is held vertically for a sufficient length of time to make it practically bloodless; the amputation is then completed (Senn's "Practical Surgery").

Fig. 997.—Keen and DaCosta's method of interilio-abdominal amputation ("International Clinics," vol. iv, 13th series).

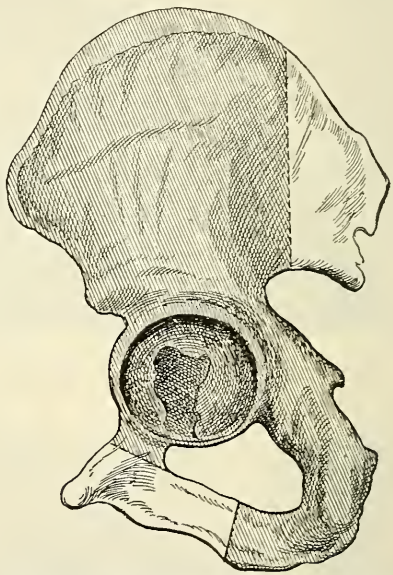


Fig. 998.—Keen and DaCosta's case of interilio-abdominal amputation. The shaded portion of the bone was removed ("International Clinics," vol. iv, 13th series).

*Other Methods.*—John G. Sheldon ("Amer. Med.," April 19, 1902) has modified Senn's method as follows: He disarticulates the head of the femur and frees the upper part of the femur from its attachments. He then introduces a pair of long, stout artery-forceps behind the femur and clamps the femoral vessels. He forms the flap, removes the limb, and ligates the vessels. In this operation the surgeon can work rapidly and can make a flap of any size or shape, and is not hindered by a constriction apparatus; but this method does not cut off the bleeding from the obturator and the sciatic arteries.

Larrey amputated by lateral flaps, and Liston by anteroposterior flaps. Forneaux Jordan's method consists in dividing the soft parts low down, tying the blood-vessels on the face of the stump, shelling out the femur from the soft parts, and disarticulating.

**Interilio-abdominal Amputation.**—This very formidable operation is occasionally performed for sarcoma of the ilium. The operation was first performed by Billroth in 1891, and the patient died. Prof. Keen and I collected 19 cases, including 1 of our own. Five of these cases recovered (W. W. Keen and J. Chalmers DaCosta, in "International Clinics," vol. iv, 13th series). Our patient perished in thirty-three hours from suppression of urine and with gangrene of the parts supplied by the internal iliac artery. In some cases the entire innominate bone has been removed, in others portions of it have been left. In our cases we made the flap shown in Fig. 997, tied the internal iliac artery after rolling up the peritoneum, but spared the external iliac, kept the femoral in the flap, and sawed through the bones as indicated in Fig. 998, leaving in place the portions shown in white.

## XXXIX. DISEASES OF THE MAMMARY GLAND

**Hypertrophy of the Breast** (Fig. 999).—This is a rare condition. It may affect one breast or both. It is most apt to appear at the age of puberty, but it may appear in childhood, adult life, or old age. The breast may attain enormous size. In Porter's case the breasts of a woman of thirty-seven were so very large that they were carried hung upon a frame ("Boston Med. and Surg. Jour.," March 3, 1892).

These very large breasts are not composed of true gland tissue, but rather of fat and connective tissue ("Diseases of the Breast," by A. Marmaduke Sheild). Hypertrophy may also occur in the male breast. In some cases hypertrophy occurs so rapidly as to merit the name *acute*. Such cases may perhaps be sarcomatous.

**Treatment.**—Be sure it is hypertrophy and not sarcoma, adenoma, or lipoma. Try recumbent posture, dry diet, pressure, and iodid of potash (Sheild, Ibid.). If these means fail, amputation is the only resource.

**Mammillitis and Fissure.**—The nipple may inflame as a result of injury, but the condition is rarely encountered except in a woman who is nursing a baby. It is most common after a first pregnancy, when the nipple is deformed or when the skin is delicate. The nipple is slightly injured during nursing, and the epithelium is macerated by the milk and saliva. If the inflammation is not arrested, a spot excoriates or an irritable ulcer forms (a fissure). A fissure is often surrounded by an area of acute inflammation, and nursing causes intense agony. Because of the pain the mother is apt to extend the intervals between nursing, and as a consequence the breasts become swollen with retained milk. The ulcer not unusually bleeds when the breast is taken by the child. Besides the fact that a fissure causes pain to the mother, it often leads to grave trouble. It is a suppurating area, and as such may lead to abscess of the mother's breast, or may impair the health of the nursing child.

**Prevention of Fissure.**—During pregnancy the nipples should be carefully attended to. They should be washed often in sterile water and bathed in alcohol, and if retracted, ought to be drawn out repeatedly. During the period of lactation the nipples are washed in sterile water, dried, and dusted with borated talc powder as soon as an act of nursing is completed. Washing the nipples regularly with the following solution tends to prevent the formation of a fissure: iodid of mercury, 2 gr.; alcohol, 1½ oz.; glycerin and distilled water, of each a pint (Lepage). If a small abrasion appears, order the woman to wear a nipple-shield during nursing, and after each act of nursing to wash the part with hot sterile water, dry, and dust borated talc over the surface. If a fissure forms, wean the child at once, and dry up the milk in both breasts. It is useless to try and dry it up in one breast. Milk may be dried up by applying ointment of belladonna locally, and administering iodid of potassium internally; by strapping the breasts with adhesive plaster (Parker); or by applying to the nipples six times a day a 5 per cent. solution of cocain in equal parts of glycerin and water (Joise). The fissure is not treated by ointments. These preparations are septic, prevent drainage, and aggravate maceration. Wash the fissure twice a day with peroxid of hydrogen, dress it with gauze wet in boric acid solution (10 gr. to 1 dram of water), and cover the dressing with waxed paper. If the fissure resists treatment, touch it with lunar caustic.

Fig. 999.—Hypertrophy of breast.

**Acute Mastitis and Abscess.**—Acute inflammation of the breast, as a result of injury of the breast or nipple, may occur in either sex at any time of life. Very commonly in both sexes a few days after birth the breast becomes distended with a material which in reality is milk. The fluid is usually small in quantity. The process is physiological, and, as a rule, ceases spontaneously (Guelliot). If it lingers, the application of belladonna ointment will stop secretion. If the nurse meddles with the glands and tries to squeeze out the fluid, acute mastitis is apt to arise in one gland, or occasionally in both. The skin of the breast reddens, the gland swells and becomes tender and painful, the child loses its appetite and becomes feverish, restless, and sleepless. Such a condition is treated by the local use of alcohol. If pus forms, the local signs and constitutional symptoms are aggravated. Evacuate the pus, dress with hot antiseptic fomentations, and be sure that the child is well nourished. Tonics and stimulants are indicated.

A condition identical with the secretory activity of the glands of the newborn may occur in either sex at puberty. The methods of treatment are the same in both cases. As a matter of fact, at this time of life rarely more than one lobule inflames, and suppuration is most unusual.

Acute mastitis is most usually met with in a woman who is nursing a child, and is due to bacterial infection. Primipara are particularly liable to develop mastitis. So are women with deformed nipples. In most cases an abrasion of the nipple exists, and through this breach of continuity bacteria gain entrance to the breast-tissue. The abrasion may be so slight that it can only be detected when the nipple is examined through a magnifying-glass (Marmaduke Sheild). Streptococcic infections are very generally due to inoculation of a fissure of the nipple. Bacteria may pass up the milk-ducts, coagulating the milk and penetrating through the walls of the acini. Staphylococci not unusually pursue this route in reaching the breast-tissue. Occasionally causative bacteria reach the breast through the arteries (in septicemia and in septic wounds of the genital organs).

**Symptoms.**—There are pain, swelling, and tenderness in the breast, and in most cases a fissure or abrasion exists. There is a febrile condition. Occasionally a chill ushers in the attack.

If a case supposed to be acute mastitis proves utterly rebellious to treatment the suspicion should arise in the surgeon's mind that the condition is acute or inflamed cancer (carcinoma mastitoides, see page 1436). If such a doubt arises, a piece of tissue must be excised for microscopical study.

**Treatment.**—Order the patient to suspend nursing. The physician endeavors to arrest the secretion of milk. Treat the nipple as advised on page 1424. Support the breast and apply ichthyol ointment.

Mastitis may undergo resolution; it may terminate in organization and induration; it may eventuate in suppuration.

**Acute abscess of the breast** follows acute mastitis. There may be but one area of suppuration, or multiple foci may exist, which eventually fuse. The symptoms of mastitis, local and constitutional, are greatly aggravated. After a time the skin becomes dusky and edematous. The axillary and superficial cervical glands enlarge. The abscess will eventually open spontaneously at one or more points, leaving branching fistulæ. A superficial abscess is situated just beneath the nipple, and pus may flow from the nipple.

An intramammary abscess is in the depths of the gland. There are often nultiple foci of suppuration. Nodules are felt in the gland, pus may run from the nipple, but cutaneous redness is late in appearing.

Retromammary abscess is a rather rare condition. It may occur alone or be associated and connected with an area of intramammary suppuration. It may result from metastatis or from caries of a rib. The breast is lifted up by the fluid beneath it.

**Treatment.**—Open a superficial abscess by an incision radiating from the nipple. Treat as any other acute abscess. An intramammary abscess should be opened by a radiating incision, and pockets of pus should be broken into with the finger. An examination is made to determine if a retromammary abscess also exists If this is found to be the case, an incision is made at the point of junction of the thorax and mammary gland, and at the lower border of the gland. The gland is raised from the chest wall, the pus evacuated, a drainage-tube is inserted, and a few sutures are introduced. If retromammary abscess exists alone, make the last-named incision in the first place.

**Chronic Mastitis.**—This condition may be present in only a portion of the breast, or may attack many lobules (lobar mastitis). The ordinary form may arise after weaning a child, or may be due to a blow, to the pressure of corsets, or to numerous slight traumatisms. It may occur in the young, the middle aged, or the old. The patient has slight pain at times in the gland. Examination detects a firm, elastic area, which is somewhat tender and does not possess distinct margins. The skin is not adherent to the mass

unless suppuration occurs. If the mass is pressed against the chest by the surgeon's fingers, it becomes evident that no real tumor exists.

**Treatment.**—Remove any cause of irritation. Support the breast by a spica bandage. Apply ichthyol ointment During the night employ a hot-water bag. If pus forms, treat as before directed.

**Chronic lobular mastitis** is a condition in which numerous lobules become indurated. The real cause of this condition is unknown. It may occur at any age after puberty, and often attacks both breasts. Such a breast is apt to be painful, especially at the menstrual periods; it feels unnatural, solid, and careful examination detects numerous indurated areas, each of which is of small size. At the menstrual period the breast enlarges and new nodules may be detected. In some of these cases violent neuralgic pains are present in the gland (*mastodynia*). Chronic lobular mastitis is apt to lead to cyst formation. When cysts form fluid may occasionally discharge from the nipple.

**Treatment.**—Support the breast and apply ichthyol ointment or belladonna ointment. Examine the generative organs and correct any existing abnormality. Improve the general health by good food, tonics, and open-air life. In cases in which multiple cysts are known to exist the question of treatment is uncertain. There seems to be little doubt that such cases tend in some instances to eventuate in cancer. I believe that the proper treatment when multiple cysts exist is extirpation of the breast.

**Tuberculosis of the Mammary Gland.**—Sir Astley Cooper in 1829 wrote on "scrofulous swellings" of the breast, and Velpeau also referred to them. Nevertheless, Virchow, in his treatise on tumors, stated that the mammary gland was not subject to tuberculosis. Durbar first proved the existence of the condition by histological and bacteriological observations. Primary tuberculosis of the breast is a rare condition. If we are to judge from English and American literature, it is a very rare condition. In 1902 Bindo de Vecchi was able to report 1 case and collect 77 from literature ("Extratis della Clinica Chirurgica," No. 8, 1902). Braendle in 1906 reported 11 cases from the Tübingen clinic ("Beit. zur klin. Chir.," vol. i, 1906). Powers, of Denver, reported 4 cases ("Annals of Surgery," Feb., 1913). About 80 cases confirmed by bacteriological findings and histological study have been reported. It is seldom that both glands are involved. Tuberculosis of the breast may be secondary to tuberculosis of the skin, of related glands, of the rib, etc. It may result from some distant tuberculous lesion of bone, of joint, of lung, etc. It may be a part of general miliary tuberculosis. We consider here primary or, as Geissler named it, solitary tuberculosis, tuberculosis apparently limited to the breast in an individual free from evidences of antecedent tuberculosis, and of tuberculosis elsewhere. It occurs usually in those of excellent general health. The route of infection may be by the blood, by the lymph-ducts, and perhaps by the lymphatics from the skin or nipple. The lesion begins in the periacinous and periductal connective tissue. The ducts and acini become involved later. E. M. Von Eberts ("Amer. Jour. Med. Sci.," July, 1909) states that there is no reported case before the age of puberty, that the most advanced case reported was fifty-three years of age, that maturity of the gland and lactation predispose, and that the reported cases show the proportion of the married to the unmarried as 4 to 1. It is vastly more common in women than in men. In many cases there is a history of antecedent inflammation or abscess during lactation. In some cases there is a history of traumatism. There are two forms of the condition, and in each form, sooner or later, degeneration occurs, and fistulæ from a cold abscess arises (see page 242); these forms are *nodular* and *confluent* (Von Eberts, Ibid.). In the *nodular* form a nodule, several nodules, or many nodules arise in the glandular tissue. There is little

or no pain. If nodules are under the nipple retraction may occur. The condition is very slow in progress and a year or several years may elapse before degeneration occurs. Degeneration results in cold abscess (see page 236) and often in fistula formation. Schley has pointed out that cold abscess is a termination more common in the confluent than in the nodular type ("Annals of Surgery," 1903). In the confluent form the condition develops much more rapidly, is associated with pain, is most apt to arise during lactation, is particularly prone to abscess and fistula formation, and is liable to acute exacerbation from secondary pyogenic infection. The axillary glands are found enlarged in three-fourths of all cases of primary tuberculosis. Cases have been reported of carcinoma and of adenoma associated with tuberculosis.

**Treatment.**—In a very slowly developing nodular case, in which it is certain lactation will not arise, it may be considered proper to treat the condition with tuberculin, etc. (see section on Tuberculosis). In the confluent form and in cases of the nodular form in which tuberculin treatment has failed, or in which we cannot exclude the possibility of pregnancy, the breast should be removed and the glands and fat should be removed from the axilla. Most of the cases recover permanently after radical operation (Braendle, of Tübingen, in "Beit. zur klin. Chir.," 1906, Bd. 1). Powers ("Annals of Surgery," Feb., 1913) advises the thorough removal of the breast, pectoral fascia, and axillary contents, and with this recommendation I am in accord.

**Cysts and Tumors of the Nipple.**—Tumors are rare in the nipple, but do sometimes occur. The following growths are occasionally seen: fibroma, angeioma, papilloma, myxoma, myoma, and epithelioma. Sebaceous cysts of the nipple and areola are not very unusual. A cancer of the nipple may be a primary growth, or may be secondary to gland cancer. Primary epithelioma of the nipple presents the same general characters as epithelioma in any other part. It begins as an indurated area in the areola, or an excoriation of the nipple. Ulceration soon occurs. The ulcer is irregular in outline, has hard edges, and furnishes a foul, red, sanious, and fetid discharge. The mammary gland becomes infiltrated at an early period. The subclavian glands enlarge, and later the axillary glands. Such a growth must not be confounded with a chancre of the nipple.

**Treatment of Tumors of the Nipple.**—Innocent tumors are to be excised and the breast need not be removed.

Epithelioma of the nipple requires the complete extirpation of the breast, and also the clearing out of the lymphatic contents of the axilla, and possibly of the subclavian triangle.

**Paget's Disease of the Nipple** (*Malignant Dermatitis*).—This condition is held to be a chronic inflammation of the epithelial layer of the nipple and areola occurring in women beyond middle life, and is regarded as a not unusual precursor of epithelioma of the nipple and of duct cancer. Paget's disease is not a simple eczema, it is not associated with the usual causes and attendants of eczema, either local or constitutional, and is not cured by remedies which control the ordinary disease.

The diseased area is raw and red, and from it exudes copiously a thick, yellow discharge. In some cases Paget's disease is secondary to duct cancer, auto-infection of the nipple having been effected by the fluid flowing from the ducts. Investigations have shown the presence of psorosperms in areas of Paget's disease. I am of the opinion that Paget's disease is usually, from the very beginning, cancer.

**Treatment.**—Removal of the entire breast and clearing out of the axilla.

**Tumors and Cysts of the Mammary Gland.**—These tumors may be innocent or malignant. Tumors may occur in childhood (angeioma, sarcoma, fibro-adenoma). Malignant tumors are very rare before the age of twenty-five.

**Innocent Tumors of the Mammary Gland.**—The innocent tumors are: *Periductal fibroma, fibrocystadenoma, papillary cystadenoma, simple adenoma, periductal fibromyxoma, myxoma, angioma, lipoma,* and *enchondroma.* It is maintained by most authorities that any innocent tumor of the gland may and is apt to become malignant.

**Periductal Fibroma, Fibro-adenoma.**—The nomenclature of fibro-adenomata is in a state of great confusion. The name fibro-adenoma was given by Cornil and Ranvier to the same sort of growth which the younger Gross called a fibroma, Billroth an adenofibroma, and Sir Astley Cooper a chronic mammary tumor. It is doubtful if a pure fibroma ever occurs in the mammary gland. A fibro-adenoma consists of acini surrounded by fibrous tissue. Each of these structures proliferates, but the fibrous tissue does so much more rapidly than the glandular. Bloodgood ("Amer. Jour. Med. Sci.," Feb., 1908) says, "the fibro-adenoma microscopically is nothing more than an encapsulated area of normal breast tissue, in which the parenchyma, in the early stage of the tumor, is greater in amount than the normal breast. Later the parenchyma undergoes pressure atrophy and the tumor may become calcified." A growth of this character is surrounded by a capsule and is movable. It is firm, elastic, lobulated, superficially situated, and of slow growth. It is unassociated with retracted nipple, glandular enlargement, adhesion to the skin, or cachexia, and may occur at any age up to fifty, but is most common between twenty and thirty (Sir J. Bland-Sutton). Such a tumor is rarely very painful, but it may be tender on rough handling and may be painful at the menstrual period. As a rule, there is but one of these tumors in a mammary gland, but the tumors may be multiple in one gland, or one or more may exist in each gland. It is not very common for sarcoma or carcinoma to arise.

**Periductal Fibromyxoma.**—It is most common in young women. It may be multiple in one breast or both. It is an encapsulated, lobuled, and elastic growth, which is seldom painful, is usually small, and often remains quiescent indefinitely or even disappears. It may enlarge and even attain a large size. When it enlarges it is apt to become cystic and sarcomatous.

*Treatment of Periductal Fibroma and Fibromyxoma.*—Extirpation of the tumor. If a supposedly innocent growth, removed by a limited operation, was really the seat of beginning malignancy, the wound will soon thicken after healing and recurrence will be rapid. Halsted has shown that in such a case re-operation, however extensive, will not obtain a cure. Hence, it behooves us in doubtful cases, if we err at all, to err on the side of radicalism. In a case recognized as doubtful, when the growth is removed a frozen section should be made at once and be studied then and there, and the surgeon should suspend operation while he waits for the report. If the report shows that the growth is malignant, at once remove the breast radically and clean out the axilla. A tumor known to be innocent may be removed through an incision made along the junction of the mammary gland and breast, at the lower margin of the gland, as Thomas proposed, or, better, at the edge of the outer hemisphere, as advocated by Warren ("Annals of Surgery," June, 1907). The incision exposes the fibers of the great pectoral muscle, the gland is raised from the muscle, and its posterior surface exposed. Any growth is exposed by an incision from center to periphery and this incision is exploratory. Warren removes an innocent tumor, a cyst, or cysts by a V-shaped incision, the apex of the V being at the center of the gland, and he wisely insists that the tumor is not to be dissected out. Several radiating incisions may be necessary to explore a cystic breast. The V-shaped space from which the tumor or cyst was removed is closed by a double row of catgut sutures. Incisions for exploration seldom need to be closed by suture. The gland is sutured to the outer edge of the pectoral fascia, and a row of sutures is inserted through the deep layer of the superficial fascia.

Warren calls this operation *plastic resection of the breast.* It leaves the patient free from deformity.

**Fibrocystadenoma** or **cystic adenoma** (**adenocele**) is a rare form of slowly growing tumor, which is apt to attain a large size, which is nodular in outline, hard to the touch, and firmly attached to the mammary gland, but mobile upon the chest. A cystic adenoma has a distinct capsule. This form of tumor is painless, and is most apt to occur in women between thirty and forty who have borne children. The growth is adherent to the skin, but the cutaneous surface is not discolored, the cutaneous veins are not distended, the axillary glands are not enlarged, and the nipple is not retracted. From the walls of the dilated acini papillomatous growths are apt to arise (intracystic vegetations). The growth may be a precursor of cancer.

*Treatment.*—Radical removal of breast and clearing of axilla.

**Papillary Cystadenoma.**—This condition is often called villous papilloma or duct papilloma. There is much more epithelial proliferation than in the fibrocystadenoma, and the warty masses project into the cyst cavities. These growths are firm, grow slowly and painlessly, and seldom fluctuate. They do not adhere to the skin, attain a large size, or cause glandular enlargement. They are situated near or under the nipple, and occur particularly in middle life. Discharge of serous fluid from the nipple is a common symptom. In many cases there is a bloody discharge. The condition tends to become cancerous.

*Treatment.*—The danger that duct cancer will arise is so great that the old operation of excision of the tumor should give way to radical removal of the breast. In any duct growth with a serous discharge from the nipple it is wise to remove the breast and clear the axilla. If the discharge is bloody that radical procedure is imperative.

**Simple Adenoma.**—This is a very rare tumor. It occurs in young and middle-aged women. It is soft, nodular, and freely movable. It does not adhere to the skin and does not cause lymphatic involvement. It consists of glandular acini and a very delicate stroma of connective tissue. It tends to become cancerous.

*Treatment.*—Extirpation of the tumor. Touch the wound with chlorid of zinc solution (10 gr. to the ounce) in order to destroy cells which might lodge and grow.

**Myxoma** is a rare tumor, and only occurs in a person of middle age or beyond. The growth is solitary, is soft, may be round or lobulated, and occasionally fungates. The nipple is not retracted, the superficial veins are not distended, and the axillary glands are not enlarged.

*Treatment.*—Removal of the mammary gland.

**Angioma.**—This form of tumor is very rare. It may arise secondarily to a nevus of the skin (Sir J. Bland-Sutton). The diagnosis of angioma of the skin is readily made. In a cavernous angioma of the breast it will be found that the tumor can be lessened in size by pressure, and will be increased in size by coughing, laughing, and holding the breath. Pulsation may be detected and a bruit may be audible.

*Treatment.*—For treatment of nevus see page 366. If a cavernous angioma exists in the mammary gland, it will be necessary to extirpate the gland.

*Lipoma* and *enchondroma* occasionally occur in the breast.

**Cysts of the Mammary Gland.**—**Involution cysts** (*cystic degeneration of the mamma*) occur in women who are approaching the menopause. Such cysts occur earlier in those who are sterile than in those who have borne children, and may arise after chronic mastitis. The parenchyma of the gland undergoes atrophic change, but the ducts remain, become blocked and dilated. Numerous small cysts form, and both glands, as a rule, suffer. Villous growths

may arise in the walls of the ducts. In some cases there is much white fibrous tissue between the cysts (cystic fibroma).

The subjects of this disease are often nervous, hysterical, and despondent. One or more ill-defined indurations are detected. Frequently there is a history of discharge from the nipple and of attacks of lancinating pain in the breast. Cystic breasts are dangerous, because the intracystic vegetations are liable to eventuate in duct cancer. One-fourth of these cases are cancerous when first seen (Speese, quoted by Primrose in "Amer. Jour. Med. Sci.," Jan., 1913).

*Treatment.*—In such cases, after confirming the diagnosis by an exploratory incision, remove the entire breast.

**Lacteal cyst (galactocele)** is an accumulation of milk brought about by blocking of some of the milk-ducts. It usually arises soon after the delivery of the child, but may not be noted for months or even several years after child-bed. It grows rapidly from the time it is first detected. A large quantity of milk may collect, and rupture of the cyst walls may occur, the fluid passing into the glandular connective tissue.

A galactocele is rounded, fluctuates distinctly, and increases in size during nursing. There is little or no pain. In some cases the contents of the cyst coagulate and a solid mass is formed.

*Treatment.*—Incision and drainage.

**Hydatid cysts** are rare, but do occasionally occur. There are 33 positive cases on record (Le Conte, in "Amer. Jour. Med. Sci.," Sept., 1901). A small, hard, movable, and painless mass appears in the mammary gland. Usually it gradually increases in size, but it may grow rapidly for a time and then remain apparently almost stationary for a period. If rapid growth takes place there is always pain, and pain is usual in any case when the cyst attains considerable size. Fluctuation is often absent and crepitation is never obtained (Le Conte). Suppuration is apt to occur and sinuses may form.

*Treatment.*—A small and recent cyst may be extirpated. If the cyst is not recent, but is fairly large and adherent, incise, evacuate, and pack with gauze. If the cyst is large and adherent, but is surrounded by considerable breast tissue, partially amputate the breast. If the cyst is large and the breast practically destroyed, or if the nipple adheres to the cyst, remove the mammary gland (Le Conte, Ibid.).

**Malignant tumors of the mammary gland** are ten times more common than innocent tumors. We should regard every palpable tumor in the gland as malignant until it is proved to be innocent. In other words, we reverse the rule of jurisprudence. We regard every tumor as guilty of malignancy until its innocence is proved. If the mistake is made of regarding an innocent tumor as malignant, the woman loses her breast needlessly. If a malignant tumor is regarded as innocent the woman loses her life needlessly. "The fact that malignant degeneration of benign growths in the breast is of frequent occurrence is obviously another convincing argument for the removal of all breast tumors. A recent study by Speese showed that no less than 26 per cent. of the cases of chronic cystic mastitis (that is, that form of 'abnormal involution' occurring at the menopause) examined by him showed malignancy. So, too, cyst adenomata are found associated with carcinoma, the frequency of such association being placed by some authors as high as 15 per cent. Inflammatory conditions of the breast resulting in mastitis are often found in the early history of cancer cases. All these facts point conclusively to the frequent occurrence of malignant degeneration in benign growths of the breast" (A. Primrose, Loc. cit.).

**Sarcoma of the mammary gland** is a very rare growth (not over 3 per cent. of breast tumors). It may occur at any age from puberty to old age. It was long thought to be most common from twenty to thirty-five years

of age, but Rodman's investigations show that one-half the cases occur in the fifth decade of life (Rodman on "Diseases of the Breast"). The growth may be composed of round cells or spindle cells; both varieties may be present, and myeloid cells may be found. Circumscribed sarcoma arises usually between the ages of twenty and thirty; it is firm to the touch, as it contains much fibrous tissue, is painless, does not grow very rapidly, glands are seldom involved, and there is no cachexia. The nipple is not retracted. The growth may adhere to the skin. If it is composed of giant cells or spindle cells it will rarely return after extirpation of the breast.

Diffused sarcoma is composed of small round cells, arises in the center of the breast, and grows with great rapidity. It is most commonly met with

Fig. 1000.—Scirrhous carcinoma (J. Collins Warren).

about the age of thirty-five, and a history of injury can often be elicited. The tumor is soft, some parts being softer than others because of cyst formation. It is usually mobile upon the thorax, though it soon becomes adherent to the skin. The tumor reaches a very great size, and soon fungates through the skin. There is little or no pain. The cutaneous veins over the tumor are distended, the nipple is not retracted, and the axillary glands are not often enlarged. Diffuse sarcoma is apt to recur after removal.

*Treatment.*—Remove the breast, and if the muscles of the chest wall are infiltrated, remove them. The axillary glands should be removed whether they are enlarged or not. Operation will not cure when metastases exist. If the case is inoperable, we can try the use of Coley's fluid. If the toxins

of erysipelas fail to arrest the progress of the disease, keep the patient as comfortable as possible by the administration of cocain and morphin.

**Endothelioma.**—This is a very rare tumor. I have had 1 case of it. The diagnosis cannot be made from carcinoma.

*Treatment.*—As for cancer.

**Carcinoma or Cancer of the Mammary Gland of the Female** (Fig. 1000).—The great majority of mammary tumors are cancerous. Cancer is due to proliferation of the epithelium of the acini (acinous cancer) or of the ducts (duct cancer).

**Acinous cancer** is vastly more common than duct cancer. Usually there is much connective tissue and but little parenchyma in the growth (scirrhous cancer). In some cases there is little connective tissue and much parenchyma (encephaloid or medullary cancer). If colloid degeneration of the parenchyma or stroma occurs, the growth is spoken of as colloid cancer.

*Scirrhus* (Figs. 1000 and 1001), the common form of acinous cancer, is almost as hard as stone. On section it is concave, and Sutton says "resembles an unripe pear." The tumor is without a capsule, and the epithelial cells are surrounded by masses of fibrous tissue. Portions of tissue even some distance away from the tumor proper contain foci of proliferating embryonic epithelial cells. In atrophic or withering scirrhus the fibrous stroma contracts and epithelial cells undergo fatty degeneration.

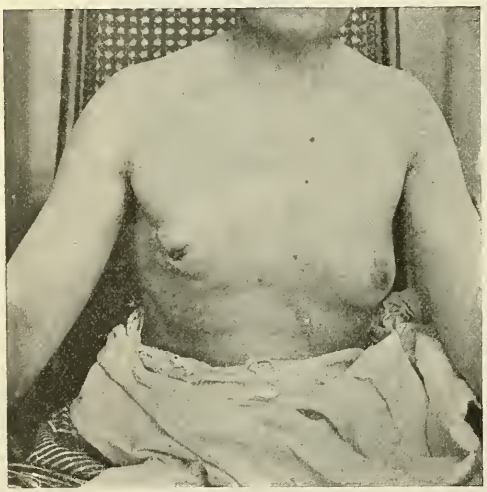


Fig. 1001.—Scirrhous carcinoma of right breast showing retraction of nipple (Dr. Blanck's patient).

Halsted in 1898 described *adenocarcinoma*. It is the initial movement in the direction of unrestrained epithelial proliferation, and sections of the tumor show the formation of tubular acini. The most characteristic sections resemble adenoma. Adenocarcinoma is not the common form of breast cancer. In the common form the proliferating epithelium attains no resemblance to glandular structure, but multiplies irregularly in connective-tissue spaces or lymph-spaces.

*Causes and Symptoms.*—Scirrhus is more common among women who have borne children than among those who have not. Heredity is manifest in only about 10 per cent. of cases. The younger Gross found it in 1 case out of 9. Trauma has no apparent influence in producing cancer. The disease is rare before the age of thirty-five, and is most common between forty-five and fifty. The author operated for scirrhus of the breast on a woman only twenty-seven years of age. Henry saw a woman of twenty-one with cancer. It is frequently met with in the aged. These tumors are rare in the negro race. A hard nodule is found in the breast, usually under the nipple, but possibly far away from it. The growth is nodular, and is immobile from the beginning. In a large, fat breast there is often a deceptive sense of mobility, because some of the breast tissue moves with the tumor. The cancer may have been present for a considerable time before being discovered. Sometimes widespread lesions develop from a small or an undiscovered breast cancer (pleural effusion, enlarged glands of the neck, disease of the spinal cord, bones of the

skull or brain). In obscure lesions of bones and viscera examine the mammary glands, because the trouble might be due to metastasis from an undiscovered carcinoma of the breast. The glands of the armpit always and soon become diseased, the glands above the clavicle often enlarge, and the arm may swell. Growth may arise within the chest, either by lymph regurgitation from the axillary and subclavian glands, or directly through the chest walls to pleura and lung or to mediastinal glands. Oelsner and Poirier showed that there is a lymph tract running from the breast to glands within the thorax, passing through the great pectoral muscle and "the fourth interspace at the level of the costochondral articulation" (Primrose, in "Amer. Jour. Med. Sci.," Jan., 1913). "Fortunately, these glands are not frequently involved, a circumstance which may be accounted for by the atrophy of this channel in senile mammæ, in which cancer usually develops (Poirier)" (Primrose, Ibid.). Retraction of the nipple is present in over one-half of the cases. It occurs when the growth is near the nipple, and is due to the contracting fibrous tissues of the tumor pulling on the milk-ducts. If the growth is far away from the nipple,

Fig. 1002.—Carcinoma of right breast. Lesion first noticed six months before photograph was made.

a dimple is apt to form on the skin of the breast because of the pulling upon the suspensory fibers. Neither retraction of the nipple nor a cutaneous dimple proves the existence of cancer. One or both may be noted in a breast containing a scar (from a wound or a healed abscess), in tuberculosis of the breast, and in mammary syphilis. The dimple is not due to adhesion between the tumor and the skin. It is noted even when the tumor is far away from the skin. It may not be obvious unless the gland is moved to and fro or unless the skin over the breast is pushed in various directions. When this is done it becomes evident that the skin, at a certain point, is held inward. The dimple is a very valuable early symptom.

Glandular enlargement in the axilla soon follows the appearance of a scirrhus; the glands become very hard and adherent. In over 60 per cent. of persons the glands of the axilla are felt to be enlarged when the patient first comes for treatment. Because the surgeon cannot feel enlarged glands is no proof that there are none. As a matter of fact, the glands are usually involved within two months of the beginning of the disease, but the involvement can rarely be detected externally until months later. Enlargement of

the axillary glands is followed by enlargement of the glands in the posterior cervical triangle and in the mediastinum. Herbert Snow has shown that the blocking of the axillary glands often leads to regurgitation of lymph containing cancer-cells, the cells being thus deposited in the head of the humerus and the thymus gland. Cancer in the thymus and in the mediastinal lymph-glands after a time causes a projection of the sternum (the *sternal symptom*). When the axillary lymphatics are extensively involved the arm swells from obstruction to the lymph-flow (lymphedema) or pressure upon the vein. If there is lymphatic obstruction the skin of the breast becomes pitted and resembles *pig skin*. The skin is actually cancerous or soon becomes so by infiltration. Each pit is the opening of a sweat-gland. The sweat-duct is held down by contracting fibrous tissue. This condition is termed *peau d'orange*, or pigskin saddle appearance. The tumor usually grows rather slowly unless lactation is established; then it grows with

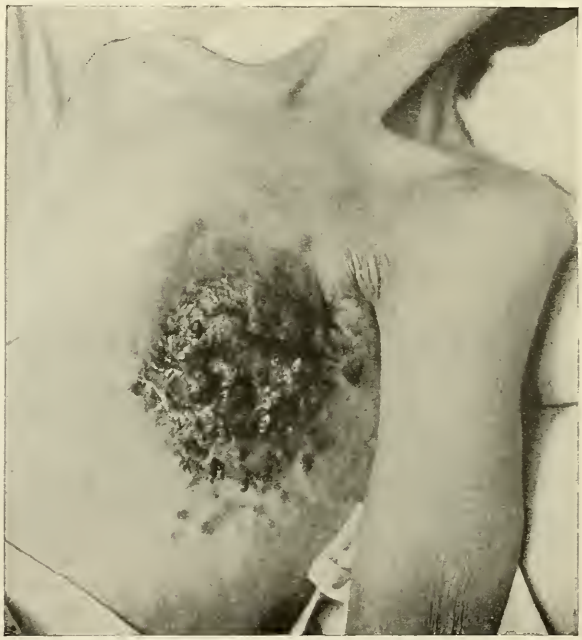


Fig. 1003.—Recurrent carcinoma. Cancer en cuirasse.

frightful rapidity. As it grows it infiltrates adjacent structures (the pectoral fascia, pectoral muscles, subcutaneous cellular tissue, and skin). When a tumor becomes adherent to the skin the skin becomes congested and of a dark purple hue. When the skin is destroyed, an ulcer forms, and around this ulcer the skin becomes red and filled with cancerous nodules, which feel like shot in the skin. Metastases are apt to occur into the bones, liver, brain, pleura, lung, spine, thymus gland, and rarely the eye. The pleura and lung may be attacked by direct spread of the growth through the chest wall, from infected mediastinal glands, or by lymph regurgitation from the axillary and subclavian glands.

Pain, absent at the start, is usually present later in scirrhous carcinoma. It is lancinating and neuralgic in character, and not brought on or increased by handling. It ceases if colloid degeneration begins. The general health is usually unimpaired until ulceration takes place, when cachexia arises. In 1792 Howard described the condition which Velpeau called

particular attention to in 1838 as a deep cancer of the integument due to a cancerous state of the deep cutaneous lymphatics. Velpeau named it *ligneous cancer* and also *cancer en cuirasse* (Figs. 1003 and 1004). The cancer en cuirasse of Velpeau is a condition in which the lymphatic vessels

Fig. 1004.—Cancer en cuirasse.

of the skin are distended because of obstruction. The skin thickens as in elephantiasis. The blocked-up lymph contains cancer cells and the skin early becomes nodular and cancerous. In most cases the condition is secondary, but primary cases have been reported. The condition may arise from cancer in the breast or may follow an operation for cancer of the breast. The skin of the chest becomes thick and rigid like a leather cuirass. The growth adheres and the soft parts come to adhere to the chest wall. The skin is very extensively invaded. In this condition the chest wall is fixed, respiration is difficult, temperature is commonly somewhat elevated, and there is probably a pleural effusion. The corresponding upper extremity is usually the seat of great swelling from hard edema.

Fig. 1005.—Recurrent carcinoma of the breast.

In *atrophic* or *withering scirrhus* the breast becomes very small. In some cases the contraction is so great that it seems as though the mammary gland had been removed. The duration of scirrhus, when left to run its course, varies, but the disease generally produces death within two and a half years.

Occasionally it causes death within a year. In atrophic scirrhus the patient may live for many years.

*Duct cancer* is not a common growth. It arises from the duct walls in conditions of cystic degeneration of the mammary gland. The tumor is softer than the acinous growth and is not nodular. There is no pain, no retraction of the nipple, no skin dimple. Serous or bloody fluid may often be squeezed from the nipple. A duct cancer grows and infiltrates less rapidly, and involves adjacent glands later than does an acinous growth.

*Carcinoma mastitoides*, *acute cancer*, *brawny cancer*, or *inflamed cancer* is a condition originally described by Volkmann in 1875 under the name of *mastitis carcinoma*. It comes on almost suddenly, grows very rapidly, causes violent irritation, and hence widespread infiltration by small cells. The condition resembles inflammation (Edward A. Schumann, in "Annals of Surgery," July, 1911). In this condition the surgeon cannot make out the outlines of a distinct tumor, but the entire breast is hardened and enlarged, and the skin of the breast is reddened, infiltrated and edematous, and adheres to the gland. There is often pain and heat.

Fig. 1006.—Ulcerating scirrhous carcinoma.

Anatomically related glands enlarge. The disease is most apt to arise late in pregnancy or during lactation and is most common in rather young women. It may be limited to one breast, but both breasts may be involved, successively or simultaneously. The nipple may or may not be retracted. Slight elevation of temperature is usual.

There is rapid metastasis, profound toxemia, and early death. Death occurs in less than a year, perhaps in a few weeks. Billroth's case died in six weeks after the discovery of the cancer.

Any persisting case of supposed acute mastitis should make us suspicious. In such a case excise a piece of tissue for examination. In cutting out the piece small abscess cavities may be discovered even when the condition is cancerous. Schumann (Ibid.) says there are only 13 reported cases of carcinoma mastitoides. Since Schumann's paper Morris Booth Miller reported a case ("Annals of Surgery," May, 1913). I showed a case to the class at Jefferson Hospital in the winter of 1912–13. The woman was pregnant and both breasts were involved. She perished miserably a few months after the onset of the disease and a few weeks after her confinement.

**Cancer of the Male Breast.**—This condition is seldom met with, though I believe it to be more common than is generally supposed. I have seen 4

cases within the last ten years. Each patient was in the early forties; neither complained of pain. In one the breast had been extremely large from early years. In each case the growth was indurated, but in neither was there any retraction of the nipple. The condition in each patient was scirrhous carcinoma. Warfield has collected 32 cases from literature and has added 5 others ("Bull. of Johns Hopkins Hosp.," Oct., 1901). The patients were between forty and seventy years of age; 8 gave a history of injury; in 9 cases there was pain, and in 12 the nipple was retracted. Palermo has collected 750 cases of tumor of the male breast ("Semaine Médicale," May 20, 1908) and 649 of them were cancerous.

**Treatment of Carcinoma of the Mammary Gland.**—The treatment is early and thorough operation; the earlier and the more thorough, the better. The older surgeons operated simply to prolong life a few months; the modern surgeon operates with the hope of curing the patient. The mortality of the operation is surprisingly small. It is certainly under 2 per cent. Rodman's statistics (2133 operations performed since 1893 by twenty-one American surgeons) show a mortality of less than 1 per cent. I have personally lost 5 patients in over 250 operations. In 1878 Billroth's statistics showed only 8 cures in 143 cases. In 1896 W. Watson Cheyne reported 12 cures out of 21 cases (57 per cent.). His cases now show 54.8 per cent. alive and well from six to thirteen years after operation. Depage's statistics show that 48 per cent. of cases passed the three-year limit ("Presse Médicale," Oct. 21, 1908). Greenough, Simmons, and Burney consider 320 cases operated upon radically ("Annals of Surgery," July, 1907); 88 cases passed the three-year limit.

E. S. Judd ("Jour. Amer. Med. Assoc.," April 27, 1912) publishes the following statistics from the Mayo Clinic. He says: "The prognosis as to the probability of a cure in a case of carcinoma of the breast will depend—1, on the length of time the neoplasm has been developing; 2, on the degree of outlying involvement; 3, on the activity of the gland, which will be determined by the age of the patient and the relation to a period of lactation; and, 4, on the thoroughness of the removal of the gland-bearing fascia.

"Data Collected from the Mayo Clinic on 518 Cases of Carcinoma of the Breast, From January 1, 1890, to January 1, 1900:

| | |
|---|---|
| Average age | 55 years, 6 months. |
| Oldest | 75 years. |
| Youngest | 21 years. |
| **Number of cases operated on over ten years before:** | |
| Alive and well | 21 (23.5 per cent.). |
| Dead | 23 |
| Not heard from | 45 |
| Total | 89 |
| **Number of cases operated on over five years before:** | |
| Alive and well | 74 (30 per cent.). |
| Dead | 76 |
| Not heard from | 89 |
| Total | 239 |
| **Number of cases operated on over two years before:** | |
| Alive and well | 233 (44 per cent.). |
| Dead | 134 |
| Not heard from | 151 |
| Total | 518" |

Bloodgood ("Amer. Jour. Med. Sci.," Feb., 1908) sets forth Halsted's statistics: "The statistics in Halsted's Clinic up to the present time show that among 210 cases, in which three years or more have passed since the operation, 42 per cent. are apparently well. If we consider the cases in which the axillary

glands, studied microscopically, showed no evidence of metastasis, 61 cases, or 85 per cent., are well. In cases in which the axillary glands showed metastasis (110), 30 per cent. remained free from recurrence for three years. When the glands in the neck showed metastasis (40 cases), only 10 per cent. remained well for three years. In all of these groups metastasis has been observed after an interval of three years of apparent cure. Such late metastasis may take place up to eight years after operation. Excluding these cases of late recurrence, the definitely cured in these three groups is reduced to 75, 24, and 7 per cent. respectively, or, for all cases together, 35 per cent. I have not time to give the facts, but there is evidence to indicate that when the microscope fails to demonstrate metastatic cancer cells in the lymphatic glands in the axilla, this is not a positive proof that metastasis has not taken place, and for this reason and others, which space prevents me from stating, there should be no restriction in the complete operation for carcinoma of the breast." It will be observed that of these 40 cases, with involvement of neck and axilla, only 4 passed the three-year limit. If they had not been operated upon, statistics would have been bettered. The surgeon who would not operate on such cases would have a higher percentage of cures for his statistics, but he would have sacrificed to statistical glory these 4 cases. If there is the *slightest* doubt of the diagnosis, make an exploratory incision before making the incisions for the removal of the breast (see Dawbarn's remarks on the deceptive signs given by thick-walled abscesses in the "Annals of Surgery," March, 1908). A frozen section can be made and examined in a few minutes, and this procedure is demanded in a doubtful case. If the mass proves to be cancer, I always pack in a piece of gauze just wrung out of boiling water and go on at once with the removal of the breast. As Dawbarn shows, this method seals up the open mouths of lymphatics. A radical operation should remove the breast and much of the skin above it, the pectoral fascia, the pectoral muscles, the fat and glands of the axilla, and the fascia over the serratus magnus. As Cheyne says, remove all the glands along the axillary vein and lift up the vein at the apex of the axilla and remove the glands and fat behind it. The sheath of the vein should always be removed. Cheyne points out that the line of spread must be traced upward along the vessels and nerves and downward along the external respiratory nerve of Bell ("Lancet," March 12, 1904). If three years after an operation there has been no return, we regard the case as cured (Volkmann's limit). As a matter of fact, recurrences are noted after five years, and this limit should be used instead of three years. It is true that 80 per cent. of those passing the three-year limit remain free from recurrence. Over 90 per cent. of those passing the five-year limit remain free. Coley reported 65 cases of recurrence—15 per cent. recurred after three years and 6 per cent. after fourteen years. Ransohoff collected 10 cases of recurrence during the seventh and eighth years, 2 each after the ninth, tenth, eleventh, twelfth, and fifteenth years, and 1 each after various intervals, from fifteen to twenty-five years. Martin suggests that these later so-called recurrences are really new growths in persons predisposed to cancer ("Annals of Surgery," Oct., 1908). Certain cases are unsuitable for a radical operation: cases in which metastases exist; cases of *cancer en cuirasse;* cases with mediastinal involvement; cases where axillary involvement is very great. Cheyne would also rule out cases in which large glands may be felt above the clavicle, believing that in such cases the mediastinal glands must be cancerous.[1] Operation is well-nigh useless for carcinoma mastitoides.

*Halsted's Operation.*—Halsted performs a very radical operation. He removes suspected tissue *in one piece*, and thus prevents carcinoma cells falling into the wound, for it is well known that if such cells should fall

[1] See "Objects and Limits of Operation for Cancer," by W. Watson Cheyne.

into the wound they may grow just as may a graft of healthy epithelium. The neck, shoulder, arm to the elbow, the entire surface of the chest down to the waist, both breasts, the axilla, the side and the back of the diseased side must be sterilized. It is necessary to have, besides scalpels and the ordinary instruments for an operation, a great number of hemostatic forceps (80 to 100). Place the patient recumbent, with a sand-pillow under the shoulder of the affected side. The shoulder is right at the edge of the bed, and a nurse holds the arm from the side, keeping it at a right angle with the body. Halsted's operation is performed as follows:[1] The skin-incision is made as shown in Fig. 1007, and is carried at once through the fat. The triangular skin-flap (*a*, *b*, *c*) is turned down. The costal insertions of the great pectoral muscle and the muscle are split between the clavicle and costal portions and up to a point opposite to the scalene tubercle, and at this point the clavicular portion of the muscle and the tissue overlying it are cut through close to the clavicle, and the apex of the axilla is at once exposed. The cellular tissue under the clavicular portion of the muscle is dissected

Fig. 1007.—Halsted's operation for carcinoma of the breast: the first incision.

from the muscle, and the splitting of the muscle is continued on to the humerus. The part of the muscle to be removed is cut through close to its humeral insertion. The whole mass circumscribed by the first incision (skin, breast, areolar tissue, and fat) is raised with considerable force in order to put the submuscular fascia on the stretch as it is stripped from the thorax close to the ribs. It is well to include the delicate sheath of the pectoralis minor muscle. The lower and outer boundary of the lesser pectoral having been passed and exposed, the muscle is cut at a right angle to its fibers and a little below the middle. The tissue over the pectoralis minor muscle near its coracoid insertion is divided as far out as possible, and is then reflected inward to prepare for the reflection upward of this part of the minor muscle. The upper portion of the minor muscle is retracted upward. Some surgeons do not remove the lesser pectoral muscle. I believe it should be removed, because the axilla can then be more easily and rapidly cleared. The removal of the muscle does not impair arm movements, and its retention leads to the formation, when healing is complete, of a cord-like band in front of the axilla. (See Douglas Drew, in "Brit. Med. Jour.," May 17, 1902.) The small blood-vessels under the minor muscle are carefully separated from it, are dissected out very clear, and are ligated close to the axillary vessels. Having exposed the subclavian

[1] "Johns Hopkins Hosp. Reports," vol. iv; "Annals of Surgery," Nov., 1894.

vein at the highest possible point below the clavicle, the contents of the axilla are dissected away with a sharp knife and the vein and its branches are stripped absolutely clean. The loose tissue about the artery and the nerves should also be removed. When the vessels are cleared, the axillary contents are rapidly stripped from the inner walls of the axilla and the lateral wall of the

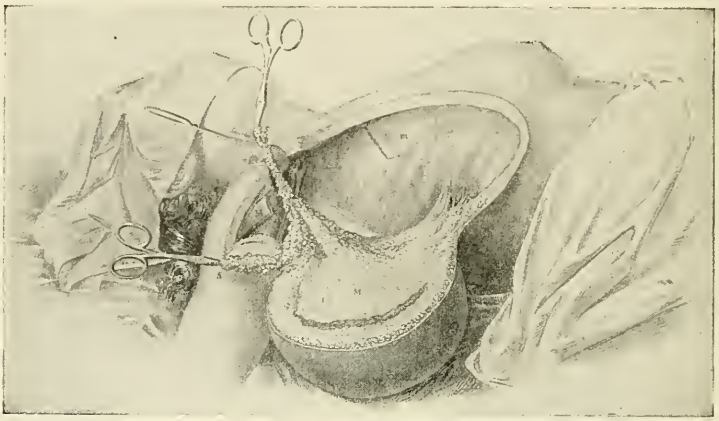


Fig. 1008.—Halsted's operation for carcinoma of the breast: The mass turned down.

thorax (Fig. 1008). The fascia which binds the mass to the chest is cut loose to the ribs and the serratus magnus muscle. Just before reaching the junction of the posterior and lateral walls of the axilla an assistant draws the triangular flap of skin outward in order to spread out the tissue which lies upon the subscapularis, teres major, and latissimus dorsi muscles. The operator cleans the posterior wall of the axilla from within outward. The subscapular vessels are clearly exposed, and are caught before they are cut. In some cases the subscapular nerves are removed, in others they are permitted to remain. Having passed these nerves, the mass is turned back into its normal position and severed from the body of the patient by a stroke of the knife from *b* to *c*, repeating the first cut through the skin. Every bleeding point, however small, is tied with fine silk. From 60 to 100 ligatures or even more may be required.

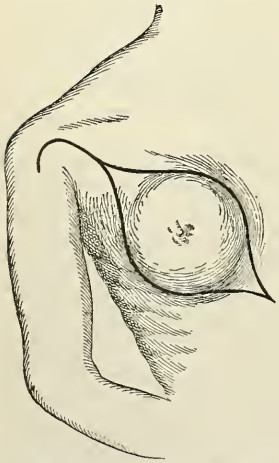


Fig. 1009.—The younger Senn's incision for amputation of the breast.

After the completion of the operation the wound into the axilla is closed with a subcuticular stitch of silver wire; if a cut has been carried above the clavicle, it is closed in the same manner, and the edges of the elliptical opening are brought nearer together by a purse-string subcuticular stitch. Thiersch grafts cut from the patient's thigh are used to cover the gap. Silver-foil is placed over the wound, this is covered with gauze, bandages are applied, and the dressing is overlaid by a plaster-of-Paris bandage, which includes the head, neck, chest, and arm. The area from which grafts were taken is dressed with sterile gauze or an ointment containing boric acid.

Formerly I did not open the subclavian triangle. I believed that these glands were involved only from the axillary lymphatics, that when they were involved the mediastinal glands were sure to be affected (the route to them

being more direct) and operation was certain to be useless. When the subclavian glands are involved from the axillary lymphatics this is true, but in some cases they are involved by way of the direct lymph paths from the mammary gland. In such a case the mediastinal glands may be free, and cleaning out the subclavian triangle may save the patient. I always open the subclavian triangle and clear out fat and glands if no glands or only a few small glands were palpable before operation. If there is a large glandular mass in the

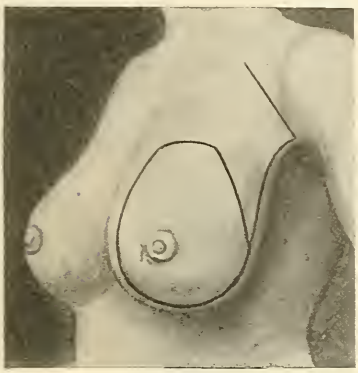


Fig. 1010.—Jabez N. Jackson's incision for removal of the mammary gland.

Fig. 1011.—Method of approximating flaps after breast amputation.

triangle, operation is useless. I always open the triangle if the tumor of the mammary gland is in the upper hemisphere, or if I discover enlarged glands at the apex of the axilla, whether there are or are not small palpable glands above the clavicle.

*The Younger Senn's Incision.*—A very useful incision is that described by the younger Senn, and shown in Fig. 1009. The breast is circumscribed by two curvilinear incisions which meet above, at the border of the great pectoral muscle. The incision is continued a little internal to the outer border of the muscle to about 1 inch above the apex of the axilla, when it is curved outward in the deltoid region, and terminates at the level of the apex of the axilla. The breast is removed from the wall of the chest, and is then suspended by axillary glands and fat, which are removed *en masse*.[1] This incision gives a free exposure, opens the axilla from in front, enables the surgeon quickly to locate and freely expose the axillary vein, and the resulting scar does not materially limit the motions of the arm.

Fig. 1012.—Warren's incision for removal of the mammary gland.

*Jackson's incision* (Jabez N. Jackson, "Jour. Amer. Med. Assoc.," March 5, 1906) is shown in Fig. 1010. The axilla is entered from above, a quadrilateral flap of skin is raised, and is subsequently pulled down to and inclosing the wound (Fig. 1011).

*Warren's incision* is shown in Fig. 1012. It enables the surgeon to close the wound.

[1] See the younger Senn in "Jour. Amer. Med. Assoc.," May 27, 1899.

*Willy Meyer's Operation* ("Jour. Amer. Med. Assoc.," July 29, 1905).—For the last few years I have been performing the operation devised by Willy Meyer. I consider it a most excellent procedure, with distinct points of superiority over other plans. We owe to Gerster the principal of opening the axilla in the beginning of the operation in order to prevent the diffusion of cancer cells and so diminish the chance of rapid recurrence. Gerster's paper was published in the "Amer. Jour. Med. Sci." in 1888. The younger Gross, in his later years, used to open the axilla first when there was an axillary mass, but he did it in order to determine in the beginning of the operation if the axillary mass was really removable. Willy Meyer emphasizes the value of his procedure in lessening hemorrhage. In Meyer's operation two flaps are formed by the skin-incision (Fig. 1013)—a lower and an upper flap. The incision for the formation of the lower flap begins at the point of insertion of the great pectoral muscle on the humerus, and is carried downward and inward $\frac{1}{2}$ inch

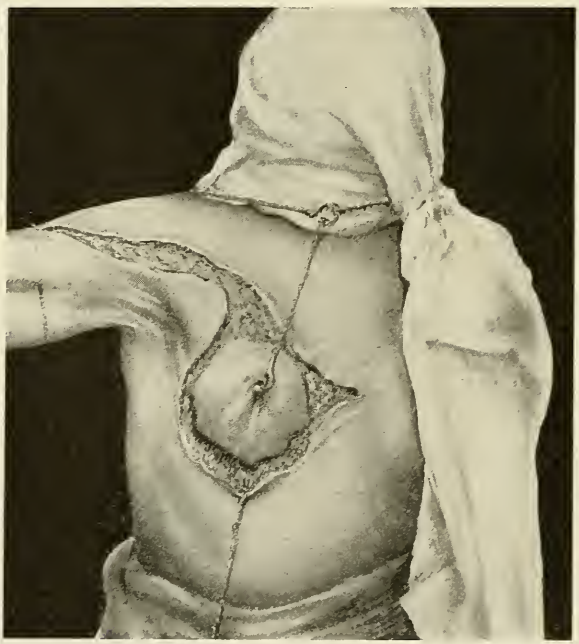


Fig. 1013.—Willy Meyer's operation for carcinoma of the breast. Skin incision as practised since 1898.

above the border of the muscle and parallel to it. When the incision reaches the base of the mammary gland, it is carried along the lower margin of the gland, and it ends over the sternum, a little beyond the midline (Fig. 1013). The lower flap is separated and turned down, a quantity of subcutaneous fat being allowed to remain attached to the breast. This turning down is carried to the border of the latissimus dorsi muscle, to the axillary cavity, and to the chest wall. Meyer then directs that the border of the latissimus dorsi be followed down to the serratus anticus major, and upward to the mass of fat that enters the bicipital sulcus of the arm. The fat is removed from the anterior border of the muscle by blunt dissection. This anterior lower wound is then packed with gauze.

The surgeon next forms the upper flap by uniting the inner and outer ends of the first incision with another incision carried along the upper margin of the breast (Fig. 1013). In this flap, as in the other, the surgeon leaves as

much subcutaneous fat adhering to the breast as he can spare without inducing the danger of skin necrosis. This upper flap is raised progressively until the cephalic vein is reached and there is exposure of the lower surface of the clavicle with the sternoclavicular articulation. Meyer directs that the tissues covering this articulation shall not be disturbed.

After the formation of these two flaps the next step in the operation is the division of the tendons of the two pectoral muscles and the exposure of the axillary and subclavian veins. Meyer advises that the cephalic vein be followed up until the insertion of the great pectoral muscle into the humerus is found. The tendon is fully exposed, care being taken to bare it of axillary fat. The arm is then carried a little nearer to the side to relax the great pectoral muscle. This tendon is cut off close to the humerus (Fig. 1014). The muscle is pulled downward and inward and is loosened from the cephalic

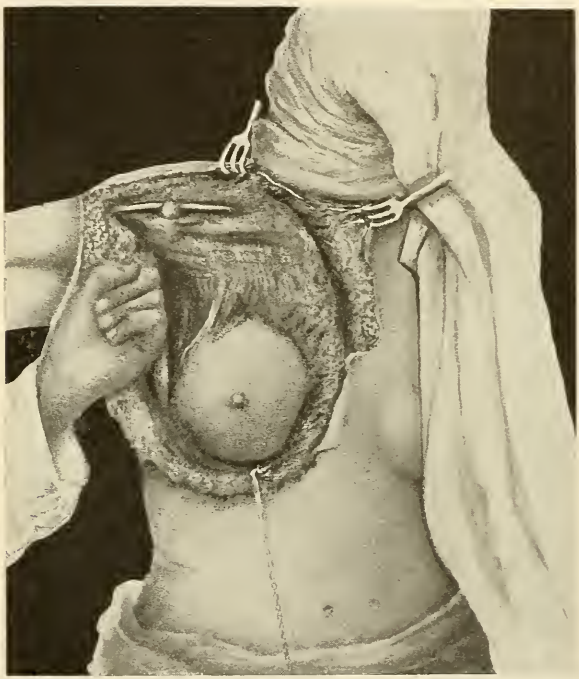


Fig. 1014.—Willy Meyer's operation for carcinoma of the breast. Insertion of pectoralis major muscle exposed. Operator's left index-finger encircling its tendon.

vein. It is then cut off near the lower border of the clavicle and the sternoclavicular articulation. It is necessary to divide the nerves that enter the pectoral muscle, and all the vessels that come into view are divided between two clamps and tied.

The next step is to divide the tendon of the lesser pectoral muscle near the coracoid process (Fig. 1015). Just beneath this tendon lies the subclavian vein. The surgeon now makes a transverse division of the fascia over the axilla, and thus exposes the axillary and subclavian veins (Fig. 1016).

Meyer's third step is to split the axillary fat over the upper portion of the latissimus dorsi up to the axillary vein, "thus dividing it from the mass of fat that enters the sulcus bicipitalis brachii."

Next, the axillary and the subclavian veins are followed up to where the subclavian passes below the clavicle, and every vessel that evidently must be

cut is divided between two ligatures and tied. This procedure saves a great amount of hemorrhage. Meyer directs us to be careful to preserve the two superior subscapular nerves, although the third subscapular must be sacrificed.

The next step in the operation is to have the assistant hold up the mass of partly loosened tissues without pulling upon them; for if he does pull upon them, Meyer truly says, he is apt to tear off pieces of periosteum or perichondrium; and such bare spots are liable to become necrotic. The surgeon now cuts to the wall of the chest, being careful not to damage the great serratus muscle. Meyer cautions us at this step to hold the blade of the knife horizontal; that is, "perpendicularly toward the thorax." "If he (the surgeon) should not thus turn the blade of his knife, but cut perpendicularly downward toward the subscapular muscle, he would enter the fat covering and enveloping the nerves and blood-vessels of this region, thus running the risk of unneces-

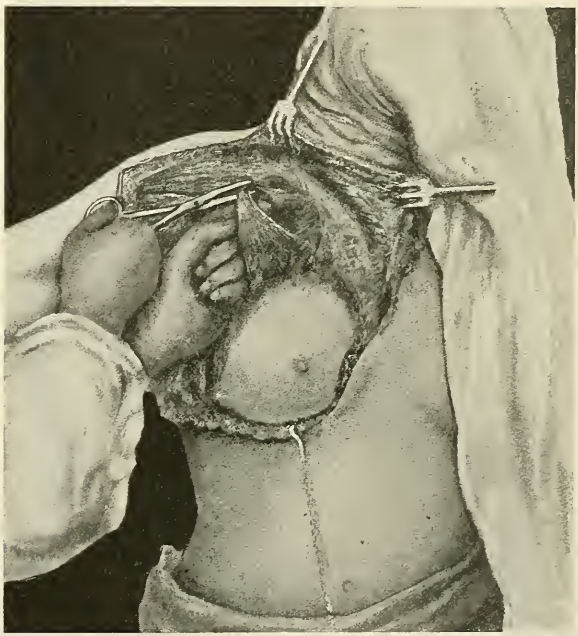


Fig. 1015.—Willy Meyer's operation for carcinoma of the breast. Finger under tendon of pectoralis minor muscle. Above, cut surface of clavicular portion of pectoralis major parallel to clavicle is visible (in the living the belly of the pectoralis major is not so thoroughly detached from that of the pectoralis minor. It is done here to show the latter's tendon).

sarily causing considerable hemorrhage and of injuring the subscapular nerves. In the general run of cases this region need not be explored; only in very advanced cases did I find a few injected glands in this area."

The pectoralis major muscle is now divided close to the wall of the chest, the cuts being parallel to the ribs, and almost level with them; and the mass being gently drawn toward the sternum. By watching carefully, one may see the perforating arteries and veins drawn out by traction before cutting them, and may usually catch each of them with two clamps and divide between the clamps. If this is impossible, they are divided and quickly picked up. The last tissue that holds the mass to the chest wall is composed of the muscle-fibers from over the sternum. These are divided close to the sternum (Fig. 1017). The final steps consist in tying all blood-vessels, draining, and suturing the wound. The draining is done through a perforation in the

posterior flap. It may be tubal or by gauze. Gauze has the advantage of restraining oozing of blood.

This operation has noteworthy merits. It can be performed far more rapidly than any other method that I have ever employed. The loss of blood is comparatively trivial, because in this operation the chief blood-vessels are divided early, are cut close to the axillary artery, and are tied. In removing the mass from the chest wall there is little bleeding, except what comes from the perforating vessels, hemorrhage from the branches of the axillary being entirely absent; and even many of these perforating vessels are tied before being *divided*. We are far less apt by this method than by the usual plan to milk lymph which contains cancer cells into the wound, or in aberrant directions through the lymphatics. As Dawbarn says, the squeezing "of the breast by the

Fig. 1016.—Willy Meyer's operation for carcinoma of the breast. Subclavian and axillary veins fully exposed. So far, glands and fat tissue not removed; smaller vessels still in connection with main trunks. Finger under fat toward sulcus bicipitalis, its nail resting on axillary vein.

retractors during its oblation, as also its handling when separated, save for its attachments to the armpit," are real dangers and may be responsible for rapid recurrence of the growth ("Annals of Surgery," March, 1908). The drain is removed in from thirty-six to forty-eight hours. The patient is placed in a sitting posture on emerging from ether and is allowed out of bed on the fourth or fifth day.

*Dressing and After-treatment.*—The dressing must be wide and ample. Fluffed up gauze is pushed into the axilla to obliterate the dead space and the arm is bound to the side for forty-eight hours. When the binding is removed, the extremity is placed on a pillow in a position of moderate abduction, and is abducted a little more each day. If the incision was placed well above the axillary border the mobility of the arm will be such that in two weeks the

patient can place the hand on the back of the head. Of late I have been placing the arm at once after the operation in abduction (upon a pillow or a triangular splint which rests upon the side). This is unnecessary if the incision does not run on to the arm and in front and above the anterior axillary margin. If the old incision in the axilla is used, abduction and all other plans will fail to prevent decided limitation of movement.

**Inoperable Malignant Diseases of the Breast.**—This term implies that a radical operation looking to cure is impossible. The conditions in which it is impossible have already been specified (see page 1438). Even if the case is judged inoperable from the radical standpoint, it may be wise to remove the mammary gland, in order to free the patient from a hideous, ulcerating area, violent pain, or harassing hemorrhage.

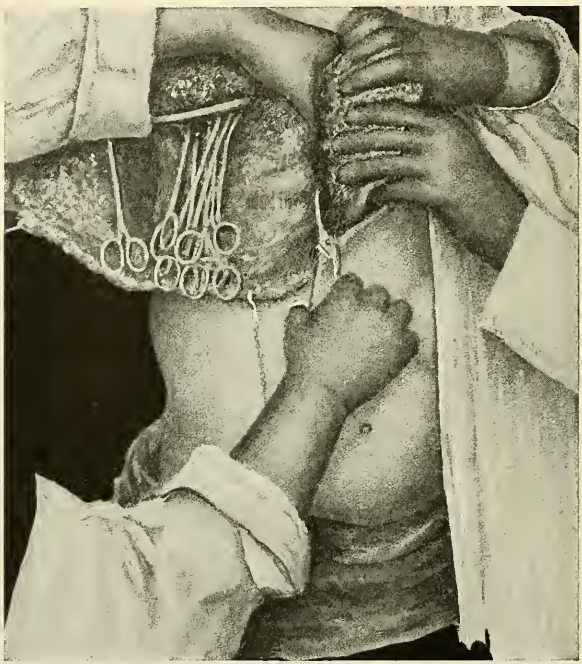


Fig. 1017.—Willy Meyer's operation for carcinoma of the breast. Pedicle of mass over sternum ready to be cut off.

It has been suggested that some cases inoperable by ordinary methods may be subjected to removal of the entire upper extremity or to disarticulation at the shoulder-joint with some prospect of cure. My own view is that when a case has advanced so far that it is not amenable to ordinary operative treatment, neither of the above-mentioned procedures offers any reasonable chance of success. If the pain is extremely violent in an inoperable case, the surgeon may relieve it by dividing the brachial plexus, or perhaps by disarticulating at the shoulder-joint.

Some inoperable cases may be greatly improved—for a time, at least—by the use of the *x*-rays; and even when the condition is not benefited in other ways, this force sometimes mitigates or greatly relieves the pain. It is said that in some cases radium is more efficient than the *x*-rays, but it is probable that most improvements by radium could have been obtained by the *x*-rays. In cases of lymphatic recurrence it is advisable to administer thyroid extract during the *x*-ray course (Woods, in "Brit. Med. Jour.," July 1, 1911).

**Beatson's Operation, or Double Oöphorectomy.**—It has been pointed out by Sir George Thomas Beatson that there is a certain similarity between the formation of cancer in the mammary gland and the process of lactation. In each there is an enormous production of embryonal epithelial cells; but in lactation the epithelial cells undergo fatty degeneration, and in cancer formation they do not do so, but penetrate into the tubules and the acini and infiltrate the gland structure. Beatson further points out that when a lactating cow is spayed, it continues to give milk indefinitely. This seems to indicate that removing the ovaries favors the fatty degeneration of the epithelial cells. This operation has been performed in cases of inoperable carcinoma of the breast in the hope of bringing about degeneration in the tumor mass. In the great majority of cases it fails utterly; but now and then it secures a notable improvement, and in a very few cases outward evidence of the disease disappeared, the general health improved, and there was gain in weight. The cure is apparent, but not real. The improvement is only temporary. Abbe obtained an apparent cure in 2 patients. It was at first thought that the operation would be applicable only to persons that have not passed the menopause, but one of Abbe's patients was over seventy years of age. Butlin, however, says that there is no genuine cure secured by this operation on record, and Beatson makes the same statement. The operation is not to be considered if visceral deposits exist. My own view is that the procedure offers but little prospect of success, but that, as it does offer some, the exact facts should be placed before the patient, and she should be permitted to choose whether or not she wishes the operation performed.

## XL. SKIAGRAPHY OR RÖNTGENOGRAPHY (THE EMPLOYMENT OF THE X-RAYS). THE FINSEN LIGHT; BECQUEREL'S RAYS; RADIUM RAYS

**The Röntgen or x-Rays.**—The cathode rays were discovered by Hittorf in 1869, while passing an induction current through a vacuum tube. Crookes, of London, greatly improved the vacuum tube, and obtained a rarefaction which left in the tube but one-millionth of an atmosphere. This last-named observer found that when an interrupted current of high potential is passed through a vacuum which is nearly perfect, fluorescence takes place. In Crookes's tube the positive electrode is placed at some indifferent point, and the current from the negative electrode flows not to the positive, but directly to the wall of the tube opposite the cathode, and at this point the phosphorescent glow is detected.

In 1895 Röntgen, of Wurzburg, while making a study of cathode rays as developed in Crookes's tubes, discovered the energy which he named the *x*-rays. Röntgen showed that at the wall of the Crookes tube opposite the negative electrode a new and hitherto unknown energy is generated. Because of the uncertain character of this energy he gave to its manifestation the name of the *x*-rays or unknown rays.

The *x*-rays are invisible; cannot be deflected, refracted, or concentrated; are not influenced by the magnet, and produce none of the ordinarily recognized effects of heat. The rays cannot be polarized, travel with the velocity of light, and cause fluorescence in certain substances, notably in the tungstate of calcium (Edison), platinocyanid of barium (Röntgen), and the platinocyanid of potassium. They have a marvellous power of penetration. Freund ("Radium Therapy") says, "Speaking broadly, one may say that the lighter the specific gravity of a body, the more transparent is it to *x*-rays. On the other hand, a body's opacity for the rays increases with its density, though not in the same

proportion." "V. Novak and O. Sule, also Voller and Walter, proved that the transparency of a body to *x*-rays depends less upon its density than upon its atomic weight."

The *x*-rays in their action on photographic plates or films exhibit actinic effects. If, therefore, an object whose component parts are of unequal density—*e. g.*, the hand—is placed on a photographic plate protected from ordinary light, and exposed to the action of *x*-rays, the plate when developed by photographic methods will exhibit a picture of the shadows cast by the several parts of the object. Such a picture is known as a skiagraph, radiograph, or a Röntgenograph. Similar shadows will be seen on a fluorescent screen if the object is between an excited *x*-ray tube and the screen. The portion of the screen free from shadow glows with fluorescence. Such a screen is known as a fluoroscope or Röntgenoscope.

The real nature of the rays remains unknown. They resemble the ultraviolet and Becquerel rays in their action on a charged electroscope and in producing fluorescence in certain substances, but neither the Becquerel nor the ultraviolet rays have the penetrating power of the Röntgen rays. Many theories have been advanced. The most acceptable has been on the electromagnetic theory of light. The difference between the Röntgen rays and a beam of sodium light is said to be that the thickness of the Röntgen ray pulse is very small compared with the wave length of sodium light, and that in the Röntgen rays there is not that regular periodic character which occurs in a train of waves of constant wave length (J. J. Thompson).

Solid bodies which are struck by the *x*-rays emit new rays having similar properties (Saganic), known as secondary rays, and less powerful than the primary rays.

For practical purposes we may consider that the *x*-rays have penetrating and actinic properties.

For the production of the Röntgen rays the essential is to have an electric current pass through a Crookes tube of rather high vacuum. The state or degree of vacuum controls the nature of the rays. A tube of low vacuum will emit soft rays of low penetrating power, while the rays from a tube of high vacuum will penetrate even pure sheet lead. The vacuum of a given tube does not remain constant with use, the tendency being for the vacuum to become higher. Some get so high that they cannot be used. All modern tubes have some device by means of which the vacuum may be regulated and controlled.

The soft rays are rich in actinic value, but because of their feeble penetrating qualities are of use only in treating superficial conditions, or for the Röntgenography of thin parts, and in conditions in which much contrast of shadows is desired. On the other hand, in deep and dense parts satisfactory results can only be obtained with hard rays. In the hands of the experienced operator the quality of the rays is adapted to the condition.

To excite the Crookes tube or, as it is more commonly called, the *x*-ray tube it is necessary to have an electric apparatus capable of delivering a current of high tension and as far as possible unidirectional. Röntgen used the induction-coil of Ruhmkorff. The fundamental parts of an induction-coil are a primary winding, which receives and stores up the electricity from its origin or from its source of supply, an interrupter, to make and break the current, and a secondary winding, which receives the current and delivers it to a negative and a positive pole. Wires carried from these poles to their respective terminals on the *x*-ray will complete the circuit.

A static machine is frequently used to excite Crookes's tubes, but only the most powerful are of practical value in taking skiagraphs. The static machine is ideal for Röntgen ray therapy, for the reasons that the current is without inverse and is of high voltage, hence will operate tubes of high degrees of vac-

uum; it is a current of constant voltage, that is, it is not a wave form of current, and, therefore, all of the current is efficient; whereas all other types of apparatus give off a wave form of current, so that the voltage is comparatively low at the bottom of the wave and very high at the peak of the wave, fully half of the wave being inefficient in the operation of high vacuum tubes; and the rays from a tube of a given degree of vacuum will be more nearly homogeneous, and can be delivered to more definite purpose, if produced by a static machine. The disadvantage of the static machine is that it will not deliver a sufficient quantity of current for rapid Röntgenographic work.

The induction coil has three disadvantages: 1, its current is not always unidirectional; 2, some form of interrupter is necessary—and all forms are troublesome; 3, the output is not sufficient for rapid exposures. It is largely used for therapeutic exposures and for exciting the *x*-ray tube on the fluoroscopic apparatus. The current is of high voltage, though low in amperage; it is easily controlled by a rheostat and switches, and operates equally well high or low vacuum tubes. The most popular machine for diagnostic work is the transformer type, the first of which was installed in the Jefferson Hospital in 1907. This apparatus is built either for direct or alternating current. When the current supply is direct, a rotary converting motor delivers an alternating current to a high tension transformer, and at the same time drives a rotating rectifying switch, so that the waves of opposite direction are collected and delivered as a unidirectional current. When the supply is alternating current the transformer receives it direct from the line, and the rectifying switch is driven by a synchronous motor. The output of the direct current machine is limited to the capacity of the rotary converting motor, while that of the alternating current type is limited only by the amperage that may be drawn from the source of supply. With the transformer the time of Röntgenographic exposure has been reduced from minutes and fractions of a minute to seconds and fractions of a second. It is almost free from inverse current in the tube circuit, but its voltage output depends upon its amperage intake to such an extent that a tube of high penetration will only operate well when a comparatively large amount of current is delivered to it. The tube heats rapidly, and thus becomes less desirable for fluoroscopic work or for therapeutic Röntgenization. Recently devices have been made to eliminate from the tube circuit a portion of the high tension waves, so that the transformer bids fair to become of universal applicability.[1]

**Physiological Effects of the Röntgen Rays.**—Clinical observations have determined that in small or moderate intensity the Röntgen rays act as a

[1] A new type of *x*-ray tube has recently been invented by Coolidge, of Tungsten lamp fame. A detailed description of this can be found in the "Physical Review" for December, 1913. The advantage of this tube over our present standard type is that the negative electrons are furnished by a heated cathode. The temperature of this cathode determines the amount of electrons to be given off, hence the amount of *x*-ray energy that may be emitted from the tube.

The cathode is heated by a storage-battery having a rheostat and meter in circuit, so that the temperature of the cathode can be definitely regulated. The penetrating qualities of the Röntgen rays given off by this tube are dependent entirely upon the voltage of current furnished by the high tension transformer. A definite quantity of *x*-rays of a definite degree of penetration can be given for any desired length of time. Any set of conditions as to hardness of rays or amount of rays can be duplicated accurately at will.

The vacuum of this tube is at least a thousand times greater than the vacuum of our present standard tube and requires no regulation, so that there will be none of the disadvantages due to regulation of the vacuum.

Dr. Coolidge believes that he will be able to produce an *x*-ray tube which with adequate exciting apparatus will produce *x*-rays that will be fully as penetrating, if not more so, than the most penetrating gamma-rays of radium.

A great deal is to be expected from the proper use of this new type of *x*-ray tube in the field of therapeutics. The tube is not yet obtainable commercially.

stimulant to cell-growth and metabolism, while prolonged radiation produces cell destruction.

Histological study shows that the cellular elements of the integument are first affected, and that only after repeated or prolonged exposures are the normal connective-tissue elements acted upon noticeably.

Cell degeneration occurs and is followed by inflammatory changes in the surrounding tissues, notably in the blood-vessels.

After sufficient irradiation this process will go on to endarteritis and necrosis.

The cause of these tissue changes under *x*-ray exposure has been the subject of varied conjectures, viz.: liberation of ozone in the tissues (Tesla); interference with cellular nutrition caused by static electric currents "induced by the introduction of the patient's tissues into the high potential induction-field surrounding the tube" (Leonard); the destruction of the nerve supply of the tissue (Hopkins); irritation of the peripheral extremities of the sensory nerves, causing vasomotor paralysis (Rudis-Jicinsky); "no doubt there is some chemical action which causes metabolic disturbances" (Beck); "the effects of *x*-rays upon tissues and upon substances sensitive to *x*-rays are due to actinic properties of the same character as those of light" (Pusey).

It is generally admitted that the rays themselves are responsible for the tissue changes, and not some external agent, such as electric discharges.

The earliest noticeable effect of exposure to *x*-rays is a tanning of the skin in dark people or reddening of the skin in blondes. Ormsby has noted this analogy between the action of *x*-rays and the sun rays.

Glandular structures undergo atrophic changes after repeated exposures. Its action on the nervous mechanism is more difficult to understand, but that it has anodyne effects in certain painful conditions is beyond question. There is also testimony, but not certain evidence, that the *x*-rays have an inhibitory influence on the sympathetic nervous system. It has occurred that a small ureteral calculus, lodged in the lower end of one ureter for a long period, has passed into the bladder within a few days after *x*-ray examination.

The influence of Röntgen rays is in many cases distinctly inhibitory to the growth of bacteria.

The untoward effects from overexposure to the *x*-rays may range from falling out of the hair or slight irritation of the skin to the production of sterility, extensive sloughing, chronic non-healing and painful ulcers, cancer, and death. These effects may be acute or chronic. The patient is more apt to develop acute conditions, while the operator or investigator is constantly in danger of the remote or chronic lesions.

The so-called *x-ray burn* is not a burn, but a dermatitis or a gangrenous process. A burn results from heat, begins on the surface and at once, is accompanied by pain from the moment of its origin, and is followed by inflammation starting from the surface burnt. An *x*-ray burn does not come in evidence for several days or for a longer time after the application of the cause, and the inflammation begins in the skin rather than upon its surface. So called *x*-ray burns may be classified by stages or degrees. In a burn of the first degree (*x-ray dermatitis*) the skin is hyperemic, more or less tender, and may itch or burn, there is increased pigment formation, and the outer layer of the skin will "pull off" as in sunburn. It may develop any time after a few days, but usually does so in from ten to twenty. Such a condition is popularly known as a "reaction." No treatment is required for dermatitis of this degree, unless there is severe itching or burning, when we may use the following preparation advised by Dr. Martin F. Engman ("Interstate Med. Jour.," July, 1903): It consists of 12 drams of boric acid, 1 oz. of zinc oxid, 1 oz. of starch, 1 oz. of subnitrate of bismuth, 1 oz. of olive oil, 3 oz. of lime-water, 3 oz. of lanolin, and 12 drams of rose-water. The powder is rubbed in a mortar, the lanolin is

added. The olive oil and lime-water, mixed, are slowly added to the powder and lanolin. The mixture is stirred, the rose-water is added, and the preparation is beaten into a creamy paste. If itching is severe, 1 to 2 per cent. of carbolic acid is added. The paste is spread on several thicknesses of gauze and the gauze is covered with a rubber-dam.

In an "*x*-ray burn" of the second degree the condition becomes more painful, vesicles or even bullæ form, there is swelling, and if the blisters are opened the denuded cutis will weep. The raw area is sensitive to touch and to direct contact with air.

This condition requires little treatment beyond aseptic attention, the use of the above-mentioned ointment, and protection from further exposure to *x*-ray or air.

The "*x*-ray burn" of the third degree involves all the layers of the skin and more or less of the underlying tissue in a process of sloughing. The slough is white, adherent, tough, and stringy. G. G. Hopkins calls the process *white gangrene* ("Phila. Med. Jour.," Jan. 6, 1900). The pain is excruciating and constant, seeming as if "red-hot coals were held against the body." Sloughing may continue for weeks or months, and the exudate is profuse and irritating. The process may eventuate in gangrene of a large area, even of a limb. Such ulcers require months to heal, if they heal at all, and are not improved by the treatment which relieves ordinary burns. Excision with subsequent skin-grafting or amputation may be necessary. In the early stages alkaline astringents give the best results, *e. g.*, a mixture composed of 2 drams each of zinc oxid and bismuth subnitrate, ½ fluidram each of liquor potassa and liquor plumbi subacetatis, 2 drams of glycerin, and sufficient lime-water to make 6 fluidounces.

This mixture may be applied twice daily and will relieve much of the itching and burning as well as control the exudate. It should be applied over a considerable area surrounding the seat of inflammation. It is generally taught that ointments should not be used to relieve the pain of ulceration. Leonard considers them conducive to malignant changes. Scarlet red ointment has been used by Manges in a case of non-healing ulcer of six months' duration. The results were distinctly encouraging at first, but the ulcer, being in the region of the umbilicus, was subjected to great tension by a coexisting ascites, and extended more rapidly in one direction than it healed in another. Then, too, the patient was cachectic from extensive carcinoma of the abdominal viscera.

In extensive ulceration the wound must be kept as clean as possible and free from irritants. When sloughing ceases and granulation tissue forms, mild stimulants may be applied.

The patient's general health should be improved in every possible manner.

Skin-grafting is not usually successful without excision of the floor of the ulcer, but should be tried. In spite of all treatment the condition may remain a chronic ulcer and be subject to malignant change.

Sometimes the results of an *x*-ray burn may be most serious. In a case reported by J. P. Tuttle it became necessary to amputate the thigh ("Med. Record," May 5, 1898).

The chronic "*x*-ray burn" or chronic *x*-ray dermatitis is seen among operators or men working more or less constantly with the rays. The onset is very insidious. Ulcers may not form for a year or more. The earliest signs of trouble are brittleness of the finger-nails and itching about the matrices. Scratches on the fingers heal sluggishly. The skin atrophies, telangiectases form, vesicles appear and disappear, keratoses develop, fall off, and return. Small ulcers start and gradually heal, reappear, become more extensive, painful, and sluggish. The finger-nails drop off, leave sensitive surfaces, and grow again indifferently or in a misshapen condition.

If exposure is continued the ulceration advances, and the tendons and even phalanges may slough away. Usually when ulceration of this kind has started, the damage is irreparable except through amputation or excision, with, of course, absolute protection from further exposure.

These lesions are most common on the hands, but may occur elsewhere.

That malignant changes take place in chronic *x*-ray lesions has been definitely shown clinically and by the microscope. Porter, of Boston, has made an extensive study of these lesions, and emphasizes the importance of both excision and skin-grafting and amputation as methods of treatment.

His results were excellent. Palliative treatment should be limited to cleanliness and care.

Sterilization of either sex by use of the *x*-ray is possible and should be guarded against.

*Can the x-rays cause death?* The *x*-rays do not cause death directly, but may inaugurate a lesion which eventually causes death. Death may follow a burn without being directly due to it. Carcinomata have developed in chronic *x*-ray lesions and produced death; at least 7 fatal cases from multiple carcinomata following chronic *x*-ray dermatitis are known to have occurred in the United States. Two of these cases occurred in Philadelphia, and one of them was in my charge. One of the fatal cases was from Hartford, Conn., and I was consulted by him.

Unnecessary exposure to *x*-rays is to be avoided. The operator should remain as far away from the excited tube as possible, and have between it and him a lead screen no less than $\frac{1}{4}$ inch in thickness and of dimensions sufficient to protect his entire person. The patient is to be protected from undue irradiation by means of lead-glass or lead-covered tube-holders and lead diaphragms. In the treatment of lesions beneath the skin some form of filter should be used over the exposed area. Sheet aluminum is commonly used. Pfahler has suggested the use of sole leather.

The function of the filter is to absorb the soft rays, which would be otherwise taken up by the skin and would irritate that structure. With proper care patients may be treated frequently and with perfect safety for a long time.

**The Uses of the Röntgen Rays.**—In the hands of trained and experienced workers this agent is a most valuable aid in nearly all branches of medicine and surgery; whereas, in the hands of the untaught and inexperienced, it may lead to grave error in diagnosis, and is, even at the present day, fraught with all the dangers of its early use. The general practitioner should not attempt its use without first having had practical training.

For diagnostic purposes studies may be made by means of the *fluoroscope* or by making Röntgenographs (skiagraphs, radiographs).

Edison's fluoroscope consists of four sides of a box, one end being open and made to fit tightly over the observer's eyes, the other end being closed with cardboard made fluorescent by smearing it with mucilage, and before the mucilage is quite dry sprinkling it with crystals of tungstate of calcium. If it is desired to examine the hand with a fluoroscope, the extremity is held opposite an excited Crookes tube and from 6 to 10 inches away from it; the end of the fluoroscope, which is covered with fluorescent paper, is placed near the surface of the hand which is away from the tube, and the observer looks through the other end of the instrument. The flesh seems but a dim haze, and the shadows of the bones are distinctly outlined. The fluoroscope is of advantage in the examination of the movable organs of the chest and abdomen. This method, except with special forms of apparatus, endangers both the patient and operator. It was used extensively in the pioneer days and caused the vast majority of injuries to the early workers. The fluoroscopic method has gained many American adherents in the last few years, owing to the development of appara-

tus which enables the Röntgenologist to work more nearly in safety. The American fluoroscopes are, as a rule, far more protective than the foreign types. Quite a few operators have apparatus built into their laboratories to suit the local conditions.

All modern fluoroscopes enable the operators to study their patients by the combined method of fluoroscopy and Röntgenography without changing the position of the patient or *x*-ray tube.

Röntgenographic technic has been wonderfully developed in the last few years. The method now used has the advantage of safety to patient and operator, as well as permanency of record. With the most improved apparatus a Röntgenograph of any portion of an average sized person can easily be made with an exposure of five seconds or less, so that the motion of breathing need not interfere as it did in former years. Children are skiagraphed by instantaneous exposure and seldom require an anesthetic. It is the most valuable agent we have for the study of bone conditions. In fractures the rays enable us to determine the nature of the injury, the amount of splintering, the existence of impaction, the question whether or not the fragments are in contact or can be brought in contact, the direction of the line of fracture, the variety of deformity, the existence of more than one fracture, the presence of epiphyseal separation or dislocation alone or with a fracture, the existence of an ununited fracture, the presence of callus, and if the splints are holding the fragments in apposition. By means of stereoscopic Röntgenographs fractures of any part of the skull can be detected; and the actual relations of the parts in fracture or dislocation of the hip, pelvis, or shoulder can be determined.

Fractures of the spine in the lower dorsal region are difficult to demonstrate in stout persons. In bone disease the experienced Röntgenologist can greatly aid the surgeon in making a differential diagnosis. The dissolved appearance of the bone in myeloid sarcoma without evidence of demarcation differs from the tuberculous bone, which presents a picture of impoverishment of mineral matter, but with more or less distinctly outlined foci of destruction. Bone atrophy due to pressure or disuse presents still another picture. Chronic periostitis, osteoperiostitis, necrosis, chronic osteomyelitis, and osteosclerosis cast shadows more or less characteristic. The bone changes in osteitis deformans (*Paget's disease*) are accurately differentiated. Bone-cysts, osteosarcoma, osteoma, and osteophytes show clearly. Conditions about the joints, such as arthritis deformans, ossifying bursitis, tabetic osteo-arthritis, and foreign substances within the joints, the character of deformity and whether due to disease or congenital defect can be determined. The bone changes of rickets and scurvy appear in distinguishable manner. Achondroplasia presents a picture unlike anything else.

Pathological processes in the accessory sinuses of the skull and in the mastoid cells are capable of detection, and the condition as well as position of the teeth can be shown with great clearness.

Stereoscopic Röntgenography of the head is often of very positive value in obscure intracranial lesions. Variations in the size of the pituitary body are determined by the measurement of the sella turcica. Likewise, disease of this gland or tumors in this region are shown by destruction of the floor of the sella turcica and the clinoid processes. Brain tumors which are superficial produce localized atrophy of bone by pressure on the inner table of the skull. In such cases the diagnosis is almost positive by means of Röntgenographs. Normally, the inner table of the skull is more or less indented by the convolutions of the brain. These indentations become striking in congenital syphilis, in which condition the skull sutures unite early and the bones expand less rapidly than the brain, and the ventricles dilate. This same condition develops with internal hydrocephalus from other causes, but in the latter instance the sutures

are ununited and even separated. On the other hand, in acute external hydrocephalus the sutures are separated, but the convolution markings on the internal table of the skull are absent.

In many cases of epilepsy there are hypertrophies or marked variations in size of the sella turcica, and, though the significance is not apparent, we frequently find evidence of calcareous deposit in the pineal gland from the size of a pin-head to a match-head or larger. They are usually associated with hypertrophies of the clinoids, are found occasionally in acromegalics and in cases of exophthalmic goiter, and headache or other nervous manifestations are sometimes discovered.

Intrathoracic conditions from very early tuberculous deposits in the lungs to generalized involvement; thickened pleura; effusion; cavity formation; consolidation; the excursions and relations of the diaphragm (*Williams's sign*) to fixation of this organ; from the normal heart to all sorts of variations in size, shape, and position; dilated aorta; aneurysm; the presence of enlarged mediastinal or peribronchial glands; and tumors of this region can be studied to great advantage in connection with physical and clinical findings.

Volumes have been written on the *x*-ray study of gastro-intestinal diseases. With adequate equipment as well as skill in technic and interpretation the Röntgenologist renders most efficient aid in the diagnosis.

It has become almost a matter of routine hospital practice to have an *x*-ray study of the progress of a bismuth meal in patients with gastro-intestinal symptoms. The bismuth meal consists of from 2 to 4 oz. of bismuth subcarbonate or oxychlorid suspended in a pint of buttermilk, one of the bacillated preparations of milk, or some especially prepared liquid food. "Kefir" and "fermilac" are popular in this country, for the reason that they keep the bismuth in suspension for a long time, are palatable, and are articles of diet suitable for most patients. Chemically pure barium sulphate has replaced bismuth in the good opinion of some workers. The stomach will empty a barium meal more rapidly than a bismuth meal. Bismuth subnitrate is no longer used because of the danger of nitrite poisoning. The study should be made fluoroscopically as well as by means of Röntgenographs.

The normal esophagus varies very little in different individuals, so that variations from a normal condition are due to functional disorder or disease. The stomach, however, does vary greatly in healthy individuals, and the study of this organ is complicated. We seldom find a stomach occupying the oblique position high in the abdomen, as described in medical text-books. In a very muscular, healthy person the stomach is usually more or less oblique, with the pylorus extending about 1 or 2 inches to the right of the median line and on a level, slightly above the umbilicus. The shape of this stomach is always controlled by its own power of contraction, or its resistance to expansion. The other extreme is the low, sagging, atonic stomach of the muscularly weak individual. The shape of the stomach depends upon the amount and weight of its contents. When filled, the greater curvature is several inches below the umbilicus, while the pylorus remains on a level with the umbilicus and is usually near the median line. This stomach is normal or, at least, true to the type of the individual. Generally, however, we speak of a stomach as being normally situated when the cardiac and median portions lie to the left of the median line, while the pyloric portion extends 1 inch or more to the right of the vertebral column, and when comfortably filled the pylorus is not more than 2 inches above the lowermost point of the greater curvature. Beyond this we find different degrees of ptosis or dilatation from congenital defect in support, disease, or atony. Schlessinger classifies the variations into hypertonic, orthonic, hypotonic, and atonic.

Röntgen ray examination should be preceded by chemical analysis of the

stomach contents and careful physical examination for points of tenderness, with markings on the skin to guide the Röntgenologist. The examination should be made with the patient in the erect posture, since the recumbent position is essentially a natural therapeutic measure for the relief of displacement producing symptoms.

The process of deglutition is observed on the fluoroscopic screen. Under normal conditions the bismuth mixture makes a sinuous, slowly moving shadow, and enters the pars cardiaca without hesitation, showing throughout the caliber of the esophagus. Diverticulæ will retain a portion of the bismuth meal and cast a locally enlarged shadow, smooth in outline. Pressure from without will cause a deviation of the normal line in one or more planes, but not necessarily narrow the normal width of the bismuth shadow. Functional constriction is differentiated by its regularity of outline, comparatively slight dilatation above the stricture, and a more or less sudden relaxation, permitting the meal to pass in volume. An organic stricture retains its shape, regular or irregular; the bismuth always passes in a thin stream. On the proximal side of the stricture, where the esophagus is dilated, the bismuth is retained and casts a broad shadow.

*Filling of Stomach.*—The normal hypertonic stomach fills slowly from cardia to pylorus. The atonic stomach receives the bismuth meal and lets it drop quickly to the most dependent portion, assuming shape only when filled. The orthotonic and hypotonic types present intermediary appearances. A Röntgenograph taken at this stage of the examination will serve as a permanent record of size, shape, and position of the stomach. The picture should be made in the erect posture with the plate on the abdominal aspect of the patient, but without pressure against the abdomen.

Fig. 1018.—Marked gastroptosis. (Taken by Dr. W. F. Manges.)

Defects in filling of the stomach are due to external pressure, adhesions to other organs, or to functional spasms, contracted scar-tissue in the stomach wall, or invasion of the stomach wall by new growth. Röntgenographs will reveal an indentation produced by the spleen, kidney, or colon; and changing the position of the patient or making pressure over the abdomen often determines the presence or absence of external adhesions, since the stomach is a more or less movable organ. Irritation of a florid ulcer by food will cause a more or less deep contraction of the transverse muscular fibers at the level of the ulcer, which contraction or indentation will last for from a few seconds to several minutes or longer.

The indentation is deeper than a normal peristaltic contraction, persists for an appreciable time, its edges are smooth, and it is opposite the area of ulcer. It can usually be reproduced by pressure over the stomach. Sometimes such a contraction persists so long as to be mistaken for an organic hour-glass contraction. It is usually associated with hyperacidity, and occurs most frequently in the middle portion of the stomach along the greater curvature; the stomach empties slowly. Contracted scar-tissue from healed ulcer will produce permanent defect in the stomach outline in extent dependent upon the amount of cicatrization. New growth invading the stomach wall will produce permanent filling defect so irregular in outline as not to be mistaken when extensive; is associated with absence of hydrochloric acid, and rapid empty-

ing of the stomach when the pylorus is not obstructed. The character of peristalsis is in itself not an important factor in diagnosis, since the type of stomach usually determines the depth of the contraction. Jonas has discovered reverse peristalsis. Haudek considers the phenomenon diagnostic of stenosis, ulcer, erosion, or carcinoma.

No less important than the filling is the emptying of the stomach. A normal stomach, that is, one that is free from disease within or obstructive influence without, except when it is of the extreme atonic type, should pass the bismuth meal above described in from three to six hours, according to the type or tone of the stomach. Conditions causing rapid emptying of the stomach are those associated with decrease in hydrochloric acid or increase in alkalinity of the duodenal secretion, and occasionally with non-closure of the pylorus. Those causing delay are, except in the extreme atonic type, associated with increased acidity or organic changes about the pylorus.

Holzknecht has arranged a number of symptoms-complex which, though condensed, are very instructive:

"Symptom-complex I: Bismuth residue after six hours. Normal stomach shadow. Achylia. Diagnosis: Small carcinoma of pylorus."

The retention is due to pyloric obstruction. If unobstructed, the presence of achylia alone would insure rapid emptying.

In symptom-complex IV he states: "Small residue after six hours. Sensitive pressure point over the stomach. Normal stomach shadow. Diagnosis: Simple gastric ulcer."

He continues: "This diagnosis is fairly certain and simple. In all cases of gastric ulcer there is always a certain amount of loss of motility. Handek has never found an ulcus ventriculi without this delay in the evacuation of the organ, and no case of spasm of the pylorus without some lesion of the stomach wall.

"As regards pressure point, it is not enough merely to find a sensitive point somewhere in the epigastrium, but we ought to demonstrate radiographically that the pressure point falls on the lesser curvature where an ulcer is most frequently situated, and that it moves with the stomach by pressure or indrawing of the stomach walls (Jonas). This complete diagnosis requires much experience both in radiology and in palpation; happily, there are a number of other symptoms which confirm the diagnosis. These are:

"1. Antiperistalsis. 2. Displacement of the pylorus upward and to the left. 3. Snail form of the lesser curvature. 4. Stabile transverse contractions. 5. Changing transverse contractions." (We would add hyperacidity.)

"Symptom-complex VII: 1. Large residue after six hours. 2. Dilatation. 3. Loss of tone. Diagnosis: Old ulcer stenosis.

"Symptom-complex VIII: 1. Large sickle-shaped residue. 2. Marked defect in filling of pars pylorica. Diagnosis: Carcinoma on base of an old ulcer, with stenosis. This picture is much more common than might be suspected. It was not known until recently that advanced stenosis of the pylorus, with dilatation and paralysis, might exist without vomiting or other severe symptoms. Vomiting may set in later, not from stenosis, but from the commencing carcinoma. The signs of dilatation and vomiting and the previous history of ulcer all point to stenosis of the pylorus, and do not in any way interfere with the indications for operation."

In addition to the above groups of symptoms depending on delay in emptying the stomach we must consider mechanical stenosis due to external adhesions. Manges has observed 2 cases, in each of which a pendulous gall-bladder filled with gall-stones was in close relation with and adherent to the pylorus. One stomach was of the orthotonic type, the other of the atonic type and more or less dilated. Both retained a portion of the bismuth meal long after six hours.

At operation, after freeing adhesions in each case, the pylorus was patulous and no evidence of organic change in the stomach wall was found by palpation. Both patients have made good recoveries, further substantiating the belief that disease of the stomach did not exist. One of the two was operated on by DaCosta, the other by Stellwagen, and the diagnosis of gall-stones in each instance was only made after incision. Pericholecystitis and perihepatitis are common conditions, and frequently involve the pylorus and first portion of the duodenum, nearly always producing more or less mechanical obstruction. Except in the case of pendulous gall-bladder, the pyloric end of the stomach, the duodenum, or both are drawn upward and to the right. The relations between these organs cannot be altered by change of posture or pressure, and there is usually tenderness. Again, delay in emptying the stomach may be caused by stricture of the duodenum from old ulcer.

The symptom of a sensitive pressure point is only of localizing value, *per se*, when a gastric ulcer has sufficient depth to produce inflammation of the peritoneum. It is always present in perforating ulcer, and is suggestive of deep ulcer when accurately traced to some definite point with relation to the stomach.

Organic hour-glass stomach is nearly always due to contracted scar-tissue the result of an old ulcer, and is usually situated in the median portion of the stomach. Carcinoma may develop secondarily, and when present usually produces more or less rapid cachexia. Marked hour-glass contraction, if non-malignant, may exist a long time without loss of weight.

Pfähler ("American Quarterly of Röntgenology," Feb., 1913) groups Röntgenological evidences of gastric ulcer under three headings, as follows:

"1. The evidence of perforation: (*a*) A projecting shadow outside the gastric shadow. (*b*) A gas bubble lying above this collection of bismuth. (*c*) Perigastric adhesions or involvement of other organs. (*d*) A palpable tumor connected with the stomach, but not affecting the lumen. (*e*) The above may be associated with either an organic or spasmodic hour-glass contraction of the stomach. (*f*) Retention of bismuth in the ulcer after the remainder of the stomach has been emptied. (*g*) Resistance corresponding to the projecting shadow.

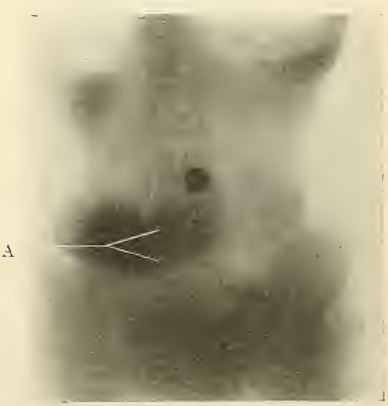



Fig. 1019.—*A*, Functional hour-glass contraction of stomach. (Taken by Dr. W. F. Manges.)

"2. The evidence of irritation, due either to a florid ulcer or to an irritable scar of an ulcer: (*a*) Spasmodic contraction. (*b*) Retention of food in the stomach after six hours. (*c*) Painful pressure point corresponding to the location of the ulcer. (*d*) Normal outline of stomach.

"3. Secondary effects usually associated with a callous ulcer: (*a*) Pyloric stenosis and gastrectasis. (*b*) Fixation. (*c*) Organic contraction—hour-glass. (*d*) Interference with peristalsis. (*e*) Reversed peristalsis. (*f*) A contracted lesser curvature with retraction of the pylorus to the left."

The diagnosis of duodenal conditions requires the Röntgenologist's best skill in technic and interpretation of both the fluorescent screen and plates. The principal findings are:

1. Rapid emptying of the stomach. This symptom is only of practical value when achylia or non-obstructive carcinoma of the stomach is absent.

2. Irregularity in outline at all times of the first portion of the duodenum,

familiarly known as "the cap." The value of this symptom depends upon the fact that the first portion of the duodenum, when not diseased, is a very constant anatomical structure. It may, however, be confused with defective outline due to result of disease in adjacent organs. In either case surgical interference is indicated.

3. The majority of duodenal ulcers occur in the first portion.

4. Dilatation of the duodenal "cap" indicates obstruction in some other portion of the duodenum.

5. Penetrating duodenal ulcer permits small projections of bismuth beyond the rest of the mass. These small portions remain for some time and occasionally contain a bubble of gas.

6. A sensitive pressure point is found in many cases to be directly over some portion of the duodenum, and when there are no external bindings the tender spot moves with the organ.

Cole's method of serial radiography of the bismuth meal is of great value in duodenal conditions. He makes twelve or more exposures in rapid succession, after the proper position of the patient has been determined fluoroscopically ("American Quarterly of Röntgenology," March, 1912). George and Gerber report only three partial errors in the Röntgen diagnosis of 59 operated cases of duodenal lesions (Ibid., June, 1913).

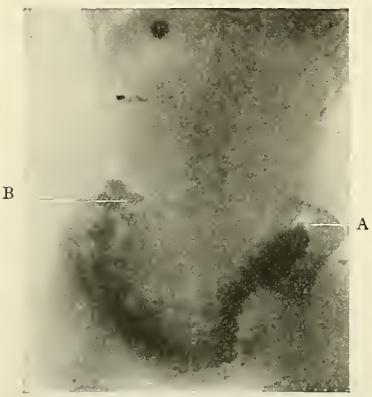



Fig. 1020.—Extreme ptosis of colon: *A*, Hepatic flexure; *B*, splenic flexure. (Taken by Dr. W. F. Manges.)

Adhesions and constrictions of the rest of the small intestine cause stasis of the bismuth meal and gas above points of involvement.

The colon is studied Röntgenologically after ingestion of the bismuth meal, and during and after injection of bismuth suspension per rectum. Also both in the erect and recumbent postures. The ingested meal under normal conditions will have passed through the ileocecal valve in from eight to twelve hours, and in from twenty-four to thirty-six hours will have been expelled. The injected bismuth suspension should be studied fluoroscopically. The findings are usually of a more positive nature than are those of the stomach and small intestine.

They are, briefly: The position and course of the colon. Evidence of adhesions, constrictions, and dilatation. Incompetency of the ileocecal valve (Case, "American Quarterly of Röntgenology," Nov., 1912). The obstructive effect of gas in elongated sigmoid loops (Pfähler, "Jour. Amer. Med. Assoc.," Nov. 16, 1912). The presence of impacted feces. The extent of motility of the different portions of the colon; the presence and extent of congenital dilatation. And often the location and extent of malignancy.

Frequently more than one examination is necessary before attempting a diagnosis. It is not essential to purge prior to examination of the ingested meal, but the colon should be thoroughly emptied by irrigation before injection. Sedative suppositories may be used before injection if the rectum is very irritable, and the temperature of the bismuth mixture should be a little above that of the body.

In a study of the urinary organs the Röntgenologist should be able to determine the size and position of the kidneys in a normal subject weighing 160 pounds or less. In larger persons only the best skiagraphs will show the outline of the kidneys. In patients under 160 pounds renal calculi from the size

of a No. 2 shot to the largest found, whether single or multiple, in one kidney or both, and relatively their situation in the kidneys (except in stones composed of pure uric acid) can be determined in Röntgenographs of the best quality.

Ureteral calculi are subject to the same display as renal calculi. The course of the ureters is by no means constant, so that one cannot say that a shadow lies in the line of the ureter unless this line is determined by additional means. However, in the great majority of instances the history of renal colic; a point of tenderness on deep pressure; the presence of microscopical quantities of blood in the urine; the fact that ureteral calculi are almost never round, but oval or irregular, in contrast to the small round shadows cast by phleboliths, or calcareous deposits in the mucous membrane of the female genitalia; and, the mulberry appearance of calcified lymphatic glands, are sufficient to confirm a Röntgenographic diagnosis of ureteral calculus. Vesical calculi are more apt to escape notice since they are not infrequently composed entirely of uric acid. When they do contain more dense material the Röntgen diagnosis is usually positive as to size and number.

In the past few years great progress has been made in the Röntgenographic study of surgical conditions of the urinary organs with the aid of ureteral catheterization and injection of solution of colloidal silver. The study demands the assistance of one skilled in the use of the ureteral catheter, and this part of the subject is treated of in another section (see page 1301).

The part of the Röntgenologist is, to be prepared to make Röntgenographs at intervals during the injection and with the patient in both the recumbent and erect postures when desired.

The points of information gained by the Röntgen study of the kidney pelvis injected with collargol (*pyelography*) are: the position, shape, size, and the extent of mobility; also the relation of the kidney pelvis to calculi in the kidney, ureter, or gall-bladder.

The normal kidney pelvis will hold from 10 to 30 c.c. of the solution, and the calices are sharp in outline, branching at the apices, and well separated. The hydronephrotic kidney may hold any amount above this to 500 c.c. or more. The calices are less separated, the apices are rounded and smooth, and all the calices have much the same appearance. The pyonephrotic kidney may hold small or large quantities; one or more of the calices may be involved, and the outline is irregular, never smooth, as in hydronephrosis. Tumors involving the kidney produce distortion, but usually not marked enlargement of the pelvis and calices. One or more of the calices may be long, narrow, and irregular in outline. Abscess of the kidney when communicating with the renal pelvis permits of injection with collargol solution, and shows as a more or less large shadow external to the shadow of the renal pelvis.

The mobility of the kidney may be shown in some cases by skiagraphing the injected kidney before and after manual displacement of the organ, but when possible the exposures should be made with the patient recumbent one instant and erect the next. The change must be made quickly, as the collargol promptly leaves the unobstructed pelvis. A stone in any portion of the kidney pelvis will be included in the shadow of the injected pelvis; a point which not only serves to differentiate stone in the pelvis from stone external to the pelvis, but shows the necessity of preliminary Röntgen examination.

The study of the ureters by this method requires that the ureters be filled to their maximum capacity throughout their entire length at the time of exposure. The patient may be placed in the Trendelenburg position with the ureteral catheter inserted only a few centimeters, so that gravity will retain the collargol solution in the ureter and kidney. In this way the true line of the ureter and diameter of the lumen will be shown. In a case of hydronephrosis, due to ureteral kink associated with a movable kidney, to pressure

from without, constriction by adhesions, or obstruction within the ureter, there will be shown a dilated ureter above the impediment and a narrow lumen below. If there is more than one point of interference the ureter will show a corresponding number of areas of dilatation. An almost completely obstructed ureter will be evident by the presence of only small areas of collargol shadow above the point of obstruction. Here again, as pointed out by Fowler and Stover ("American Quarterly of Röntgenology," Aug., 1912), skiagraphing the patient very promptly after putting him in the erect posture will often show the nature of the kink. Stone in the ureter may be positively diagnosticated by finding that collargol will enter the diverticulum occupied by the stone; more or less distortion of the line of the ureter at this point and dilatation above, if the stone has been there for some time and caused obstruction. A stone acting as a "ball valve" in the ureter will be associated with dilatation including the stone shadow.

Kelly has used a water suspension of small quantities of bismuth subnitrate injected into the bladder and stereo-Röntgenographs to show the presence of and extent of tumors involving the bladder.

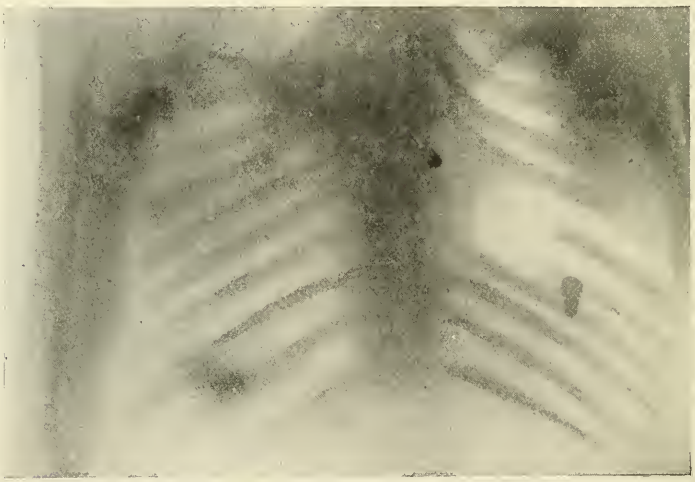


Fig. 1021.—Gunshot-wound of the lung. Rib resection for secondary hemorrhage into the pleural sac ten days after the injury; bullet not removed. Hemorrhage arrested by packing with gauze. Skiagraph taken three months afterward shows the bullet (author's case).

In addition to the above pathological findings, all sorts of anomalies and anatomical peculiarities of the urinary organs are plainly shown. Kelly and Lewis prefer an emulstion of silver iodid to collargol for pyelographic study, but the latter is more generally used.

The ureteral catheter containing lead shows the course of the ureter less accurately than proper injection of collargol, but does have the advantage of always being there at the time of exposure.

The proper preparation of the patient is an essential requirement in any *x*-ray study of the urinary organs. This consists of thorough purgation and abstinence from food or drink for at least six hours prior to the examination.

The liver and spleen offer but a small field for useful study. Enlargement may be seen. Biliary calculi are not often seen in the *x*-ray negative, because they are lacking in density and are surrounded by thick structures. One cannot make a negative diagnosis of biliary calculus by means of the *x*-ray.

Chronic discharging sinuses may be outlined and studied after they have been injected with bismuth paste. This study is best made by means of stereo-

scopic negatives. In advanced atheroma the outlines of the arteries of the extremities are seen in the Röntgenograph.

**Localization of Foreign Bodies.**—Metallic bodies, such as bullets, pieces of steel or iron, coins, pins, needles, tin, zinc, brass, etc., can be detected in any portion of the body and accurately localized. Fragments of stone, granite, marble, and lead-glass can be skiagraphed except when very small and deeply situated. Drainage-tubes and iodoform gauze can be found if lost in a sinus or cavity. Anthracite coal, glass other than lead-glass, or splinters of wood are difficult to detect unless they are of considerable thickness, are not superimposed by bone, and are embedded in thin structures so that the foreign body can be brought close to the sensitive plate. When the fingers are the seat of injury, anteroposterior and lateral views are sufficient. When in the esophagus, trachea, or bronchi the fluoroscope should be used for differentiation, after which a Röntgenograph will assist.

Foreign bodies elsewhere should be localized. If near a joint or in close relation to some surgical guide, localization is accomplished by stereoscopic

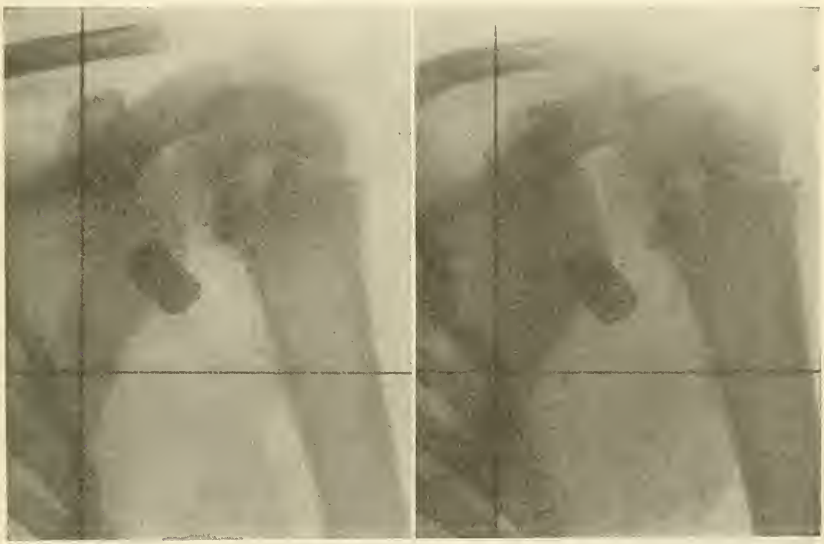


Fig. 1022.—Bullet localized by means of stereoscope. Removed by author.

Röntgenographs. (See stereoscopic skiagraph, Fig. 1022, of bullet in a shoulder case of mine. The bullet lay in close relation to the anterior surface of the glenoid margin, and on a line perpendicularly below the coracoid process. This bullet was removed with ease.)

Pieces of needle in the palm of the hand or soft parts near the surface are best localized stereoscopically when a definite relation between some known surface mark and the foreign body can be determined. A thin coating of bismuth on the palm of the hand will cause every line in the skin to show on the Röntgenograph. When the foreign body is deeply situated and not in close relation to a surgical guide, some method of localization must be used to indicate the depth and direction from a known point on the surface in which the body lies. All localizing methods are based upon displacement of the foreign body shadow by making two or more exposures with the *x*-ray tube in different and known positions, while the part containing the foreign body and sensitive plate remain in one position. This has been called the triangulation method.

Mackenzie-Davidson first used the cross-threads, from the known positions

of the focus point of the *x*-ray tube to their respective shadows on the negative, the crossing of the threads representing the position of the foreign body in relation to the sensitive plate and focus tube. Measurements were then made from some point on the skin of the patient that had registered with a known position on the negative. The Röntgenologist could then say that the foreign body lay perpendicularly beneath a certain point to a definite depth. Nearly all the modern localizing schemes are modifications of this general plan, and differ mainly in the manner of determining the position of the focus point of the *x*-ray tube.

A foreign body, unless it be large, may easily lie to one side of an imaginary line as drawn by the scalpel to the depth of 2 or more inches; again, a change in position of the patient will alter the relation of the foreign body to a perpendicular line from the indicated surface point. A modification of the Mackenzie-Davidson scheme was devised by Sweet. Manges improved the apparatus by

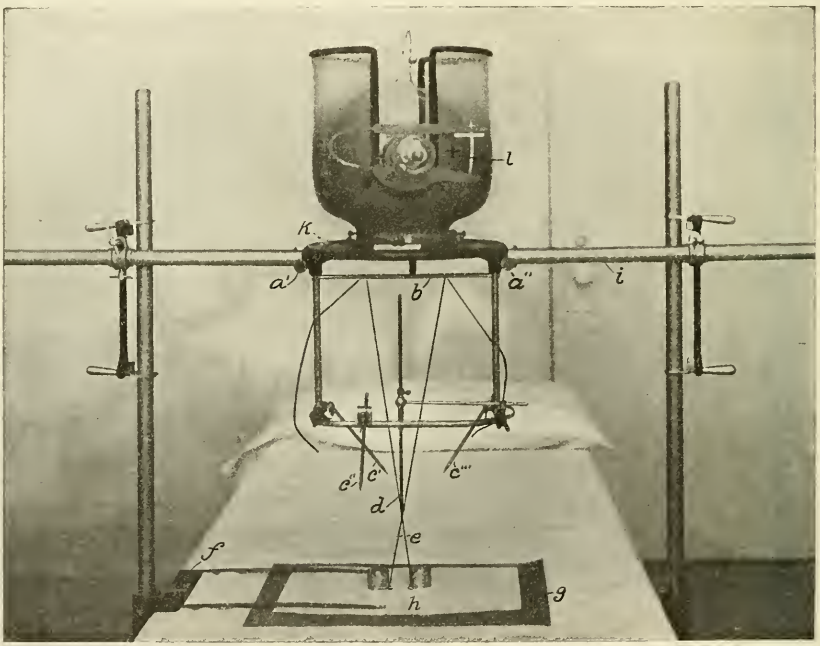



Fig. 1023.—Manges's modified Mackenzie-Davidson localizer.

limiting the possibility of error. He uses the apparatus as a guide during the surgical operation. With this apparatus all relations between the patient, foreign body, source of *x*-rays, and sensitive plate are obtained, maintained, and regained with ease and mechanical accuracy.

The localizer is a detachable part of the tube carriage of the Röntgenographic table, and consists of a metal frame having two upright bars ($a'$, $a''$), connected near their top by a horizontal bar ($b$) and at the bottom by a curved bar. The upper bar is deeply notched to a scale of inches on each side of its center. To the curved bar are attached four adjustable clamps and rods, three of which ($c'$, $c''$, $c'''$) are used to establish a relation between the localizer and the patient, the fourth ($d$) to indicate the depth and direction of the foreign body. The fourth rod can be moved through a sleeve in the direction of its long axis, and can be held at the desired depth by a collar and set-screw attachment. The horizontal bar of the tube carriage is graduated

on one side to correspond with the notches on the cross-bar of the localizer. The focus point on the target of the tube (the source of the rays) is accurately determined by sighting from cross-lines on one side of the lead-glass tube shield to cross-lines on the opposite of the shield (*l*), and is exactly 6 inches above the level of the notched bar of the localizer. The relation between the focus point and the sensitive plate is obtained by means of a clamp on the edge of the table (*f*). This clamp has two flat metallic arms projecting over the sensitive plate and a fixed rod externally, against which the upright bar of the carriage rests.

In making the localization the patient is placed on the table as if for surgical operation. A sensitive plate, 10 by 12 or 11 by 14 inches (*g*), is put under the part containing the foreign body, the tube clamp applied, and the tube carriage, localizer, and tube are adjusted and leveled. Three of the adjustable rods of

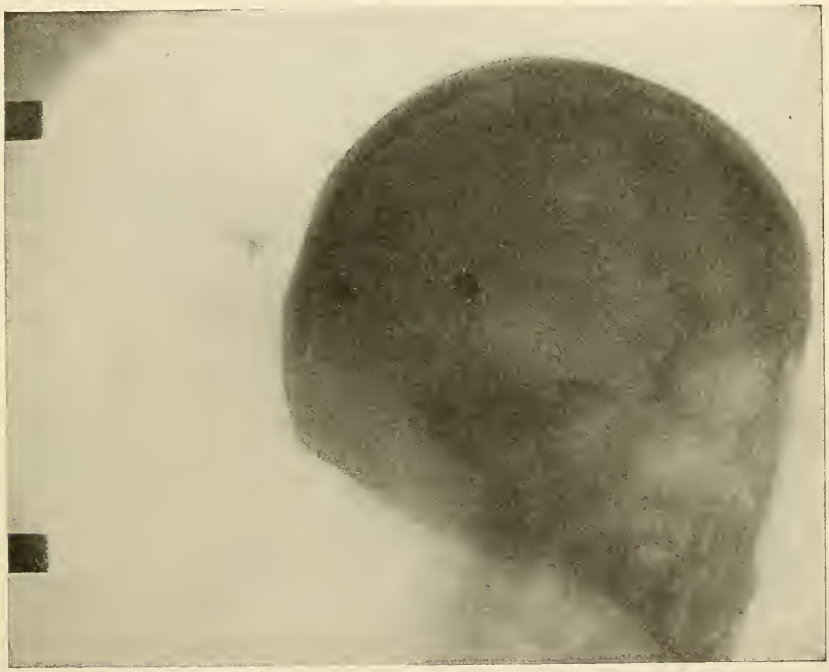


Fig. 1024.—Bullet localized by method described in the text. Removed by author.

the localizer are made to touch the surface of the patient at as widely separated points as possible, in which positions they are tightly clamped. These three spots are touched with silver nitrate.

After all adjustments are examined, and the height of the tube from the plate noted, the carriage cross-bar is elevated and the localizer removed. By lowering the tube-carrier to 6 inches below its original position, the focus point of the tube will be at the level formerly occupied by the cross-bar of the localizer, and if the carriage cross-bar (*i*) is shifted to a definite distance to one side of zero, the focus-point will be in the corresponding position held before by a notch on the cross-bar of the localizer. After carefully adjusting the tube-holder an exposure is made. The carriage is then shifted to the opposite side of zero in the same extent and manner and a second exposure made. The patient is removed, the plate developed, and tracings of the shadows of the foreign body and arms of the table clamp made on paper. This tracing is

taken to the table and put in relation with the table clamp (*h*). The localizer is then made to occupy its original position, and threads are carried from the notches on its cross-bar to the tracings of the foreign-body shadows (*e*). At the crossing of these threads is the position of the foreign body in relation to the localizer. The point of the fourth adjustable rod can be made to touch the cross-threads from any direction (*d*). If the Röntgenologist cannot determine the best direction for incision, he may consult the surgeon, and when this is known, the point of the rod is made to touch the crossing of the threads, and its collar and set-screw fixed.

At the operation for removal of the foreign body the localizer is sterilized and applied to the patient, preferably by the Röntgenologist, if he has had sufficient surgical training to render his assistance of value from the standpoint of surgical technic; or he may thoroughly instruct the surgeon in the manipulation of the apparatus. The following precautions are to be observed: First, the localizer must be handled carefully to prevent displacement of the rods; and second, the patient should be placed on the operating table in about the position he occupied on the Röntgenographic table, so that the three marked spots on the skin receive the three fixed points of the localizer. With the apparatus in position, the indicating rod is placed in its carrier or sleeve, and when its point touches the skin the long axis of the rod indicates the direction of *incision*, while the distance between the collar on the rod and the sleeve shows the depth at which the foreign body lies. During the course of the operation the localizer should be repeatedly applied, so that excessive manipulation of the tissues may be avoided.

Manges combined the localizing process above described with stereo-Röntgenography, and devised an accurate method of measuring the diameters of the female pelvis.

The determination of the localization of pieces of metal, glass, or other substances in the eye or immediately adjacent structures by means of the Röntgen rays requires that the shadow of the foreign body, as shown on the radiograph, be studied in relation to the shadow of one or more opaque objects of known position. Dr. William M. Sweet has designed a localizing apparatus which consists of two ball-pointed indicating rods, one opposite the center of the cornea and the other situated at a known distance from the first to the temporal side. The patient is placed recumbent, with the photographic plate to the side of the head corresponding with the injured eye. Two negatives are made, one with the tube horizontal or nearly so with the plane of the two indicating rods, and the other at any distance below this plane. A special chart is employed, containing a section of the normal adult eyeball, and lines are drawn indicating the planes of shadow at the two exposures. Where these planes cross is the position of the foreign body in relation to the structures of the eyeball and orbit.

The accuracy of this method of localization has been fully demonstrated by its author and other *x*-ray workers, but the learner finds so much difficulty in understanding the lines to be drawn upon the chart to represent the planes of shadow at the two exposures, that Dr. Sweet has recently designed an entirely new apparatus, in which a single indicator is employed. As shown in the illustration (Fig. 1025) the tube-holder, indicating-ball, and plate-holder are upon a movable stage, and the tube is in a fixed carrier, so that the angle of the rays with the eyeball and the distance of the tube from the plate are always the same. A telescope and reflecting mirror permit the observer to adjust the instrument until the image of a cross-wire in the tube is in direct contact with the lateral image of the summit of the cornea. After this adjustment is made, the indicating-ball is exactly 10 milimeters from the center of the cornea.

The patient fixes with the good eye upon a circular mirror placed 12 inches above the injured eye, so that there is no movement of the eye during exposure, and the visual line of the injured eye is parallel with the plate. The two exposures are made upon one plate, one with the tube in the zero position of the apparatus, so that the rays pass in a direction corresponding with the horizontal plane of the eyeball; and the second exposure with the tube at its farthest point on the graduated rod to the right or left of the first position, depending upon which eye is to be examined. Two metal shutters are used to uncover any desired portion of the photographic plate.

Since the relative position of the tube in reference to the indicating-ball and the photographic plate remains fixed and known, the direction of the rays in passing through the eyeball follows a definite course, which is the same for the two separate exposures. Lines have, therefore, been drawn upon the localizing chart to indicate the direction of the rays at the two exposures, each line

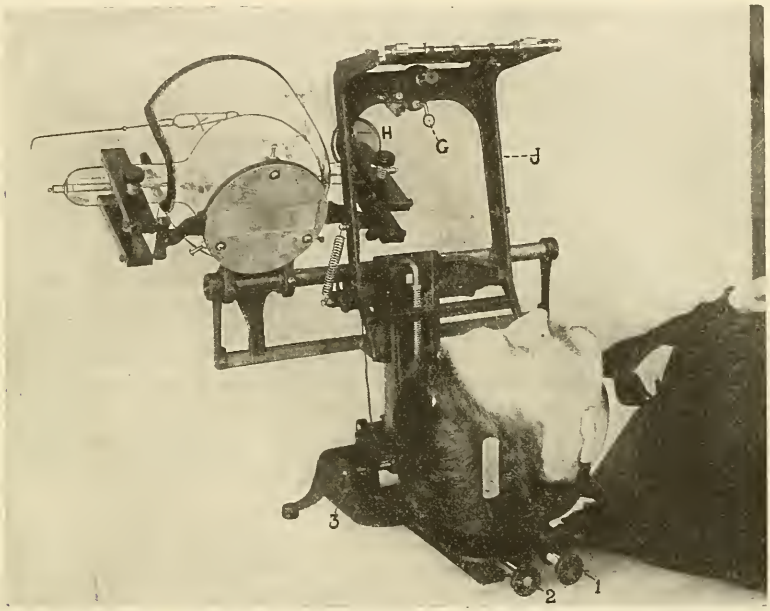



Fig. 1025.—Sweet's apparatus for locating foreign bodies in the eyeball and orbit.[1]

having the required amount of divergence to represent rays coming from a point the distance of the tube from the photographic plate. After development the photographic plate is placed in a frame containing cross-lines indicating the focal coördinates of the rays. The radiograph is moved until the shadow of the indicating-ball is in apposition with a spot representing the indicating-ball on the key-plate. A reading is then made of both the vertical and horizontal coördinate lines which pass through the shadow of the body on the radiograph, and this is transferred to the corresponding lines on the localization chart. Three readings are taken, and after having been transferred to the chart, the point of crossing of the several lines indicates the location of the foreign body in the eyeball or orbit.

The accuracy of the localization depends entirely upon the care with which the operator adjusts the indicating-ball opposite the center of the cornea and at a definite and fixed distance from it. After the exposure is made and the plate developed, the determination of the situation of the foreign body is

[1] "Transactions of the American Ophthalmological Society," 1909.

simply a question of reading from a key-plate and transferring these readings to a localization chart.

**The *x*-Rays in the Treatment of Diseases.**—It may be said that the beneficial results obtained by the use of the *x*-rays is in direct proportion to the skill of the Röntgenologist. No two Crookes's tubes are alike in their behavior; they vary widely from day to day, and even during one operation.

Unfortunately, there is no accurate method of computing dosage either in quality or quantity.[1] The quality of rays may be fairly well estimated by determining the electric resistance the tube manifests. Tubes of high vacuum offer great resistance to the passage of the electric current and emit hard or highly penetrating rays, while tubes of lower vacuum offer less resistance and emit softer rays. The quantity of a certain quality of rays may be roughly estimated by the amount of current passing through a tube of known vacuum in a definite period of time. The instrument commonly used in this country for this purpose is known as the milliamperemeter, the standard of measurement of electric units. In Europe the physiochemical properties of the rays are more in vogue as a means of measurement of dosage. In the United States most Röntgenologists give repeated short exposures, while abroad the tendency is to use infrequent but powerful applications of the rays.

In any case, ability gained by experience is essential for the proper selection and control of the Crookes tube in the treatment of a given condition.

The effects of *x*-rays which offer possibilities of therapeutic application are, according to Pusey, as follows ("The Röntgen Rays in Therapeutics and Diagnosis," Pusey-Caldwell): "(1) Their effect in causing atrophy of the appendages of the skin; (2) their destructive action upon organisms in living tissues; (3) their stimulative action upon the metabolism of tissues; (4) their power of destroying certain pathological tissues; (5) their anodyne effect." A qualitative generalization may be made, *i. e.*, superficial diseases should be treated in most instances with tubes of low vacuum, while deeper-seated lesions should be exposed to the rays from tubes of higher vacuum. Quantitatively, for causing atrophy of the skin appendages, stimulating action upon metabolism, and for the relief of pain in other than malignant conditions, mild applications or short exposures should be given; on the other hand, for destructive action maximum exposures are demanded.

In diseases of the skin the surrounding healthy parts are to be protected from the action of the rays by means of sheet-lead, while the diseased portion is directly exposed. In treating lesions beneath the surface it may be necessary to expose an area beyond the evident limits of the disease, but all overlying healthy skin must be protected from the non-penetrating rays by a filter of aluminum or leather.

Because of the possible untoward effects of exposure to *x*-rays, diseased conditions amenable to other forms of treatment should not be treated by the rays until after the simpler treatment has failed. In such conditions the rays are the "last resort." It is strikingly true, however, that the most signal results of *x*-ray therapy are obtained in conditions that are frequently incurable by other means. Many of the chronic diseases of the skin that yield but slowly to medication may be found to be safely and promptly amenable to *x*-ray treatment. Among these diseases are acne, eczema, lupus vulgaris, lupus erythematosis, psoriasis, and mycosis fungoides. The treatment is also beneficial in tinea tonsurans, favus, sycosis, alopecia areata, hypertrichosis, and prurigo.

In the treatment of malignant growths the Röntgen rays find their greatest field of therapeutic usefulness. With improved technic, permitting of massive doses of the rays with safety to overlying and adjacent healthy tissue, the results are far more favorable than in the earlier *x*-ray days.

[1] See foot-note on page 1449.

In superficial epithelioma, rodent ulcer, and Marjolin's ulcer, without glandular involvement, cure is the rule, recurrence is becoming less frequent and is amenable to cure, and the cosmetic effect is good.

When deeper structures are invaded by carcinoma cure becomes less frequent and recurrence more frequent in direct proportion to the depth of the lesion and the degree of glandular involvement. Even in advanced cases, however, for instance, when the entire contents of an orbit are involved, large portions of the nasal structure destroyed, or extensive areas of subcutaneous tissue the seat of foul and painful ulceration, and where operative interference is contra-indicated, the *x*-rays relieve pain and decrease discharge. Some reach such a stage of improvement that surgical procedure may be instituted with hope of success, and in a few actual cures result from *x*-ray treatment alone.

Epitheliomata involving mucous membranes, particularly of the lower lip, anus, or vagina, should not be treated by *x*-ray until after surgical methods have been exhausted. The rays are used to prevent recurrence after operation. The reason why the rays should be regarded as a last resource in these cases is that mucous membrane is very susceptible to the action of the rays and is as apt to be destroyed as is the cancer. Further, in the lower lip, glandular involvement is almost sure to exist. All forms of carcinoma with glandular involvement, if operable, should be subjected to radical surgical extirpation, and the rays should be used as a prophylactic measure against recurrence.

All cases of primary inoperable carcinoma, regardless of situation, extent, or the reason for being inoperable, should be exposed to vigorous irradiation. Likewise, all recurrences after operative procedure is prohibited ought to be afforded the possible benefits of *x*-ray treatment.

In sarcoma also the results are becoming more favorable, but this form of treatment is by no means on such a basis of hope that it should be allowed to interfere with or supersede surgical methods. It is a fact, however, that in some cases in which operation was not agreed to by the patient or parents, or in which the condition was deemed by the surgeon to be inoperable, surprising results have been obtained with the Röntgen rays alone or in connection with the use of Coley's toxins. I know of a case of rapidly growing periosteal sarcoma of the humerus in a young girl, the onset of which was so rapid that the mass was considered to be a subperiosteal abscess, but which was proved by *x*-ray examination and later by incision to be sarcoma. It was treated at first by *x*-rays and Coley's toxins, and later by *x*-rays alone. It has remained well for eighteen months. The arm is strong and useful, and the girl is robust. At the site of origin there remains a spur of bone which is about 1 inch in length, ½ inch in thickness, and has a density equal to that of the densest portion of the humerus. Isolated cases of inoperable retroperitoneal sarcoma, fibrosarcoma of the uterus, and other deep-seated tumors, clinically or after exploratory operation considered sarcomata, have been reported as greatly benefited, controlled, or cured.

Good results in *x*-ray treatment of inoperable malignancies can be obtained only by the best apparatus. In the deep-seated growths hard, penetrating rays in maximum quantity are demanded. The treatment must extend over a period of months, so that great care and skill are required. There is little reason to believe that *x*-rays cause metastatic involvement in other parts.

The rays have been extensively used in European countries in the treatment of selected cases of uterine myomata and menorrhagic conditions.

The indications for this plan of treatment usually mean contra-indications for surgical procedures. The results, as reported by Hainisch, Runge, Bordier, and others, are very satisfactory in cases during or near the menopause. The

hemorrhage is stopped and fibroid tumors diminish or disappear. Atrophic changes occur in the ovaries and the menopause is terminated promptly. In women under forty years of age good results are not constant.

Tuberculous sinuses that are not deep seated are frequently quickly benefited, and old tuberculous glands in which ulceration has recurred after operation should always receive the *x*-ray treatment, as some remarkable results have been obtained.

Of late years exophthalmic goiter has been treated more or less successfully by Röntgen rays. The rays are of distinct value in many cases that are not operable or amenable to other forms of treatment. The Mayos use the rays for a time befure operating. Many of the distressing nervous symptoms may be relieved, the pulse-rate is often materially lowered, and changes favorable to future operation take place in the gland and its capsule. Dr. Manges, the Röntgenologist at Jefferson College Hospital, in an extreme case of exophthalmic goiter, obtained a remarkably good result from *x*-ray treatment, so far as relief of the nervous symptoms, tachycardia, and improvement of the general health of the patient are concerned. There is not a marked diminution in the size of the goiter, but the exophthalmus is distinctly less. The patient has been following for nearly four years and without inconvenience her occupation as a weaver.

Here again more data is to be desired before other methods of treating this disease, especially surgery, are to be replaced by Röntgenization. These rays are perhaps the most valuable therapeutic agent we have in the treatment of myelogenic or splenomedullary leukemia and lymphatic leukemia. Since Senn's report some years ago this plan of treatment has been widely used in connection with other remedies. A spleen of enormous size will sometimes go back to normal, high leukocyte counts may disappear, red cells and hemoglobin may increase, and the general health of the patient is apt to improve. Stengel and Pancoast have improved the technic of *x*-ray treatment in their systematic exposures of the bony skeleton, particularly the long bones, with comparatively little irradiation of the spleen until late in the course of treatment. Even very advanced cases are apt to improve greatly for a time, but even in the early cases recurrence usually appears sooner or later, and becomes less responsive to treatment. Life may be prolonged for many years, but permanent cure is very rare, if it ever occurs.

Chronic suppurating sinuses sometimes heal under the influence of these rays, and varicose ulcers are benefited by moderate exposures. The pain in rheumatic affections and neuralgias is often greatly relieved.

### THE FINSEN LIGHT

It is known that below the spectrum of red light are heat rays, and above the spectrum of violet light are short violet, actinic, or chemical rays. The short violet, with the indigo rays and blue rays, constitute the Finsen light. Ultraviolet rays cause an electrified body to discharge, excite fluorescence in certain substances, affect a photographic plate, and are bactericidal, but have little power of penetrating tissues and, it is said, do not inflame tissues. Ultraviolet rays pass readily through rock salt or ice, which will not transmit heat-rays.

Finsen taught us to use these rays therapeutically. He first obtained the rays from sunlight, intercepting the heat-rays by ice or rock crystal. Later he obtained them from the arc light.

Blood in part prevents the passage of the Finsen light, hence in using the light we must make the area on which the rays are to act nearly bloodless. This is done by pressing firmly upon the part with a rock crystal through

which water passes. The rays pass through the crystal and the water absorbs the heat-rays. The rays are especially serviceable in lupus.

## BECQUEREL'S RAYS

Becquerel discovered in 1896 that uranium and some of its compounds give off a radiation similar to but much weaker than the *x*-rays. Among these radiant substances are pitchblende, radium, and uranium. These rays are luminous, actinic, and skiagraphic (McFarland), and may produce, by prolonged action, dermatitis similar to *x*-ray dermatitis.

## RADIUM RAYS

Monsieur and Madame Currie, after prolonged research, found that thorium and certain ores of thorium and uranium (pitchblende) are radio-active, pitchblende being more strongly so than uranium itself. The conclusion was that pitchblende contained a strongly radio-active element and that it was not uranium. In 1903 they discovered the sources of radio-activity to be two hitherto unknown elements, radium and polonium (Dawson Turner, in "Brit. Med. Jour.," Dec. 12, 1903).

Turner (Ibid.) tells us that radium gives off a radio-active emanation and three kinds of rays (alpha-rays, beta-rays, and gamma-rays). It also emits heat, and is itself at a higher temperature than the medium in which it rests. The emanation from radium is a luminous gas, which can be condensed by great cold, and which imparts radio-activity to certain bodies. It is to this gas that most of the curative effects of radium can be attributed.

Alpha-rays consist of a stream of positively charged gaseous particles. Turner points out that these particles are each about twice the size of a hydrogen atom, travel at a velocity of 20,000 miles a second, and have little power of penetration. In fact, the penetrating power of the alpha-rays is so slight that they do not pass through the glass of a tube (Robert Abbe, in "Med. Record," Oct. 12, 1907). The beta-rays consist of particles each being one one-thousandth the size of a hydrogen atom and being strongly actinic. These rays are said by Turner to resemble cathode rays and to be far more penetrating than alpha-rays. Gamma-rays resemble *x*-rays and have great penetrating power (Dawson Turner, Loc. cit.). According to Abbe ("N. Y. Med. Jour.," Feb. 10, 1912), the soft beta-rays and the alpha-rays irritate and stimulate; the hard beta-rays and gamma-rays destroy. It is probable that radium also generates or helps to generate a gas called helium, which has no action on tissues.

The actions of radium are extraordinary. A man entirely blind cannot perceive light when radium is brought near him, but one not quite but almost blind can, and one quite blind to form but with retention of some light perception can actually see the shapes of objects near a screen rendered luminous by radium. Turner tells us that a man retaining vision, who covers his eyes, can detect radium held in a box behind his head. If dry seeds before planting are exposed to radium rays sprouting will be retarded in proportion to the time of exposure. When meal worms are exposed to the rays "they go on living as meal worms, 'veritable Methuselahs,' as it has been said, while their sisters and brothers, unradiumized, progress for generations, completing several cycles of beetles, eggs, meal worms, etc." (Abbe, in "Med. Record," Oct. 12, 1907). Radium rays are germicidal, but act very slowly and feebly. Skiagraphs can be taken with the rays. Water and other materials may be rendered radio-active by exposure to radium rays. Probably certain natural waters have subtle powers due to radio-activity. On the tissues radium may

act to produce a retrogressive effect; may increase self-digestion; may cause irritation and inflammation, and so block blood-vessels. Severe reaction may result in ulceration. On a tumor radium produces inflammation, first of the fibrous stroma, then of the tumor-cells, or a primary necrosis (Sticker, in "Presse Médicale," May 18, 1912). It seems to have a selective action on cancerous tissue. Sometimes a spreading eruption, like that of scarlet fever, follows overaction. Abbe says that when an ulcerated surface is treated or when a tube of radium is inserted in a wound for twenty-four hours, a "specific toxemia" frequently arises. "The symptoms will be headache, chill, general aching, coated tongue, fever up to from 103° to 106° F., and an occasional rash-like scarlatina." Some hold that radium acts similarly to the *x*-rays and that the *x*-rays can do anything radium can do. Other observers believe that the radium rays have a specific action and can accomplish some few things impossible to the *x*-rays.

Radium therapy is commanding profound and widespread public interest. How real its claims are and how great its future is to be we can only guess. Sir Frederick Treves bids us be cautious in our estimates, although he thinks there may be a great future for radium therapy in surgery ("Brit. Med. Jour.," Jan. 30, 1909). It has cured many *surface* carcinomata and sarcomata, keloids, angeiomata, moles, pigmented moles, and warts. It is particularly valuable in lesions about the face when it is desirable to avoid scars; in lesions of the nostril, mouth, and other cavities. It has a very powerful action on angeiomata. So far radium has been used chiefly for inoperable cancer. It has been used most advantageously for epithelioma of the eyelid (see the striking cases reported by Abbe, in "Med. Record," Oct. 12, 1907). When treating a surface lesion the rays are obtained from radium bromid, which material is kept in a hermetically sealed platinum or lead tube. In a deep-seated tumor an incision may be made and a tube of radium bromid be inserted in the wound and allowed to remain for forty-eight hours. Attempts are being made to treat internal conditions by the inhalation of radium emanations or by the administration of materials which have been rendered radio-active and contain, so to speak, radium emanations in solution. Radio-active water has been tried for cancer of the stomach. Treatment by radium is called radium therapy; treatment by radio-active substances, radiotherapy. It is extraordinary how even very brief applications of radium may be followed by notable changes. In one of Abbe's cases an epithelioma of the forehead disappeared after one exposure of an hour's duration. Another on the side of the nose disappeared in four weeks after one exposure of an hour's duration. A cancer involving one-third of the upper eyelid entirely disappeared in two weeks after three five-minute exposures. After an exposure no changes are apparent for several days or a week. The skin at the site of application then begins to burn and itch and becomes reddened. The irritation endures for about two weeks.

Confusion has arisen because of the varying strengths and amounts used by different operators. Bromid of radium is the salt usually employed. Abbe takes as a standard 10 mg. of bromid of radium and tests other specimens by this. He calls the 10 mg. of strong German bromid of radium "the working unit." In malignant disease the best results have followed one hour's exhibition of the "working unit" to small growths and three or four hours to large growths, "with an interval of one month for study of the effect" (Abbe, Ibid.). Ischemia favors powerful action of radium rays.

In order to obtain the best results from radium heavy "doses" seem to be necessary, and heavy "doses" are very apt to burn the skin. The penetrating rays are the gamma-rays, and in order to make them reach a deep growth in sufficient intensity to do good the superficial parts are endangered.

So far radium has found its chief use in inoperable cases and in superficial lesions. Abbe ("New York Med. Jour.," Feb. 10, 1912) says, we may suppress the short rays which burn, and yet use the deeply penetrating rays by placing a considerable amount of radium in lead which is from $\frac{1}{10}$ to 2 mm. in thickness.

Macdonald ("Brit. Med. Jour.," Dec. 9, 1911) uses for a deep growth at least 250 mg. of bromid of radium in platinum $2\frac{1}{2}$ mm. thick. He keeps the tube applied from twenty-four to forty-eight hours. In some cases he inserts the tube in the tumor and leaves it in place for forty-eight hours. A long application is not to be repeated for five or six weeks. For a superficial growth he uses radium in a glass tube every other day for a week.

It may develop that radium has decided power in preventing recurrence after operation. In some few cases of cancer radium may convert an inoperable into an operable case.

A serious bar to the extensive use of radium is its immense cost. The little there is in the world is in the hands of a few men and a few institutions. Altruistic persons in many lands are striving to give greater numbers access to the supposedly beneficent influence of that wonderful element. No process ever was so thoroughly and dramatically advertised as the radium treatment of cancer. There is a hideous cruelty in the newspaper exploitation of radium therapy, because great numbers of persons who are victims of cancer have been caused to believe that they could certainly be cured if they could gain access to radium. Such hopes cannot be realized. Any statement that an extensive cancer can *usually* or even *often* be cured by radium is a stupid mistake or a heartless deception. At the present time the real truth of the matter is being slowly and carefully studied out by earnest men who are not seeking profitable notoriety. There is little evidence at present that radium, except in certain exceptional cases, has greater curative power than the *x*-rays, and the Coolidge *x*-ray tube may remove even these exceptional cases from the radium side of the scale.

## XLI. INJURIES BY ELECTRICITY

**Effects Produced by Lightning.**—Every year in the United States about 224 persons are killed by lightning (McAdie, as quoted in Draper's "Legal Medicine"). An individual may be struck directly or he may be shocked by the lightning having struck a nearby object. A person can be struck while in a room, but there is more danger when he is exposed, especially in the open country. To be under a single tree or under a tree at the margin of a forest during a thunder-storm is dangerous, but to be in a wood or under a hedge is reasonably safe. The oak is struck more often and the beech less often than other trees. During a thunder-storm it is not safe to stand by a chimney or fireplace, in an open doorway, or close to cattle (McAdie, Ibid.). One should not use a telephone or ring a bell during a storm. It is unwise to run, as the current of air thus created is a danger. Telegraph posts should be avoided. A pool of water is a dangerous neighbor. Horses and other animals should be given a wide berth. The victim of lightning may be killed instantly. The body may show no mark, but there are usually burns. Burns may be superficial, or a large area (as a limb) may be carbonized. Death is the fate of over one-third of those struck. Tidy ("Legal Medicine") states that out of 54 cases, 21 died and 33 recovered. Recovery may follow even when there has been severe burning. Postmortem examination may fail to reveal a lesion, but in many cases severe burns are discovered; in some there are laceration of tissue, crushing of bones, and fearful injury. Burns are especially apt to occur at the points where the current entered and emerged. The clothes are usually singed or torn, shoes are especially apt to be torn apart or

destroyed. Yet a man may be burned or killed and the clothing be undamaged, or the underclothing may be destroyed and the outer garments escape uninjured. A person's clothing may be destroyed, he may be left naked, and yet he may not be injured. Clothing may be cast far off, and in some cases is said to have utterly disappeared. The typical lightning-marks are arborescent tracings, representing the course of blood-vessels, produced by disorganization and effusion of blood as the fluid travels through it. Occasionally metal objects, such as buttons, knives, money, keys, etc., are fused, and spread as a metallic film over a considerable portion of the surface of the body. Bichat stated that in death from lightning rigor mortis does not occur. This statement is now known to be an error (see the 3 cases reported by M. Tourdes). As a rule, there are early but perhaps brief rigor mortis, retained fluidity of blood, and distention of the brain with venous blood. Putrefaction is early and rapid. A man killed by lightning may remain in the exact attitude in which he was struck dead. He may not be disfigured, his clothing may be intact, and yet, almost at a touch, he may crumble to ashes because the body was practically incinerated (see the apparently authentic cases quoted by Flammarion in "Thunder and Lightning"). In most cases killed the body shows burns. The cause of death by lightning was supposed by Hunter to be destruction of muscular contractility, and by Richardson the resolution of the blood into gases. It is asserted that some deaths are due to actual disorganization of vital structure and that others are due to shock or inhibition. Spitzka believes that death from electric shock is due to asphyxia, to paralysis of the heart with fibrillary contractions of that organ, or to a combination of these conditions. An individual struck by lightning may recover even when he is *apparently* dead. Sestier[1] collected reports of 77 persons struck by lightning; 7 of them were apparently dead for a number of hours, but finally reacted. Brouardel says in such cases the death-like state may be ascribed to inhibition, caused by a *maximum* degree of stimulus.[2] When death from lightning is not immediate, the condition may be as above outlined, the individual being apparently dead, without obvious respiration or pulse. He may be insensible, with slow and labored respiration, a weak and irregular pulse, and dilated pupils, and may remain in this condition for a few minutes or for several hours. The above condition is scarcely to be distinguished from severe concussion of the brain. Every individual suffering from the effect of lightning should have his entire body carefully examined to see if physical injures exist (fractures, wounds, burns, ecchymoses, arborescent tracings). The consequences of lightning-stroke are many and various. There may be rapid and complete recovery, gradual recovery, traumatic neurasthenia, sloughing burns, partial paralysis, which is usually recovered from (Nothnagel), but which may be permanent; hysteria, blindness, change of character, and actual insanity.

**Treatment.**—Do not pronounce a person dead until a thorough attempt at resuscitation has been made. Raise the head a little, draw the tongue forward. Make artificial respiration in the prone position (Schäfer's method). Occasionally tickle the epiglottis, apply external heat, massage over the heart, and, if the means are at hand and the man is apparently dead or all but dead, throw salt solution and adrenalin into a large artery and toward the heart, as advised by Crile and Dolley (see page 468). Do not give alcoholic stimulants. If the respiration is feeble, make tongue traction and employ artificial respiration. Apply the stream of a hot douche to the head, rub the limbs with mustard, put a mustard plaster over the heart and another to the back of the neck, wrap the individual in hot blankets, give

[1] Sestier, "De la Foudre," Paris, 1866. Quoted by Brouardel in his lectures upon "Death and Sudden Death."

[2] Benham's translation of Brouardel's lectures upon "Death and Sudden Death."

enemata of hot saline fluid, and strychnin hypodermatically. In some cases venesection has seemed to be of benefit. Lumbar puncture may be tried. When the individual reacts, treat any existing condition symptomatically, and treat particular physical injuries according to their character.

**Effects of Artificial Currents.**—Individuals may receive dangerous or fatal shocks by contact with wires carrying a powerful electric current, by contact with a dynamo, or with some metal object which has become accidentally charged by a powerful current. The shock may be from contact with both poles of a circuit (while standing on the ground and touching one pole of a "grounded" circuit); when actual contact has not occurred, but a person has been very close to a high-tension current and the current has junped an inch or several inches through the air; when a person insulated from the ground grasps the conductor with both hands (Spitzka, "Jour. Med. Soc. of New Jersey," 1909). Workmen for electric companies, pedestrians in the streets of a city which is lighted by electricity or in which trolley cars are employed, roofers, and firemen are liable to be injured by electricity. During many fires in cities live electric wires fall and charge the rails of a street-car track, the iron of a hook-and-ladder truck, water-tower, or a fire-escape. Firemen who come in contact with such charged materials are shocked. I have seen dozens of men thus shocked, but have as yet seen no fatal case. There are enormous differences in individual resistance and susceptibility. Spitzka points out that in 1 case death followed a shock of 65 volts, and others have survived shocks of many thousand volts. The amount of current the individual gets from the circuit is influenced by the tension of the current; the ground he stands on (metal floors and wet floors are most dangerous); the area of contact (limited area of contact means serious burning and less general shock, broad area means little or no burning and severe shock); the nature of the tissue (the thick palm offers more resistance than the lip, and the more callous the palm of the hand the greater the resistance), and the duration of the contact. An alternating current is decidedly more dangerous than a continuous current of equal strength. The constant current causes a shock only as the circuit is opened and closed. While the current is passing continuously there is no shock, although dreadful burns may be caused at this time. The alternating current causes rapidly repeated violent shocks. The arc light is either an alternating or a direct current. "Low tension currents with 30 to 150 alternations are more dangerous to the heart than if of more than 500 frequency. Greater frequencies, as in Tesla's currents, are practically harmless" (Spitzka, Ibid.). An artificial current acts like lightning. It may produce instant death, it may produce unconsciousness, delirium, stertorous respiration, Cheyne-Stokes breathing, or clonic spasms. Its effects can be often recovered from. Not unusually the victim is apparently dead, but subsequently recovers. D'Arsonval reports the case of a man who was apparently killed by the passage of 4500 volts. No attempt at resuscitation was made for one-half hour, and yet he recovered when artificial respiration was employed. Donnellan reports a case of recovery after the passage of 1000 volts. Slight shocks may cause temporary numbness and even motor paralysis. An electric shock frequently causes burns or ecchymoses and, occasionally, wounds. Wounds caused by electricity bleed profusely and are apt to slough. Spitzka (Ibid.) sets forth the effects of electric shock as follows:

"I. *Local Signs:* (*a*) Burns of the skin and hair. (*b*) Puncture and rupture of tissues. (*c*) Superficial necroses. (*d*) Metallic impregantion of the surface tissues. (*e*) Hemorrhages. (*f*) Edema, erythemas, 'lightning' figures.

"II. *General Effects:* (*a*) Loss of consciousness and of nerve-functions generally. (*b*) Paralyses or spasms of muscles. (*c*) Disturbances of respiration and cardiac action; high temperature.

"*Later:* Affections of the bowel activity: meteorism, constipation, albuminuria, icterus, incontinence or retention of urine, bloody urine, arterial rigor or spasms of arterioles, acute edema of various parts (joints); eye-symptoms of various kinds: blinding, conjunctivitis, keratitis, iritis, cataract, dislocation of lens, etc.; ear-symptoms: rupture of tympanic membranes, deafness, bleeding, epistaxis; thermal symptoms: usually a rise of temperature to 38° to 39° C., amnesia, neuritis, etc."

If death occurs it is due to asphyxia, to cardiac paralysis with fibrillary contractions of the heart, or to both conditions (Spitzka, "Jour. Med. Soc. of New Jersey," 1909). An *electric burn* looks like a blackened crust; it is surrounded by pale skin, and for twenty-four hours remains dry, when inflammatory oozing begins and the skin around it reddens. These burns are seldom as painful as ordinary burns, but sometimes cause severe pain, and recovery requires a long time. When inflammation begins and suppuration occurs, tissue is extensively destroyed; tendons, bones, and joints may suffer; some portions become deeply excavated, and other portions show dry adherent masses of dead and dying tissue, and a burn which was at first small may be followed by a large area of moist gangrene;[1] lack of tissue resistance, due to trophic disturbance, is largely responsible for the progress of the sloughing. Even an apparently trivial burn may be followed by extensive sloughing.

**Treatment.**—If a person is in contact with a live wire, the first thing to do is, if possible, to shut off the current. If it is not possible to shut off the current, catch a portion of the clothing of the victim and pull him away from the wire, but do not touch his body with the bare hand. If a pair of rubber gloves can be obtained, the subject can be moved with impunity and the wires can be safely cut. If it is not possible to drag a person away from electric wires, an individual can wrap his hands in dry woolen material and safely lift the portion of the body in contact with earth or wire, and thus break the circuit and permit removal of the body.[2] A dry cloth can be pushed between the body and the ground, and the body can then be removed from the wires. It may be possible to push the wires away by means of a dry piece of wood, or to cut them with shears which have wooden handles and which are perfectly dry, or to push or draw the body away from the wire by the employment of sticks of dry wood. Spitzka warns us to be careful in using shears "as the momentary arc formed between the separated ends may blind the rescuers." Treat the general condition in the manner set forth in the article on Lightning-stroke (see page 1472). Raise the head a little, draw the tongue forward, and tickle the epiglottis. If he does not breathe, place the patient prone and make artificial respiration by Schäfer's method. Artificial respiration may be carried out with the patient supine by Meltzer's method of tracheal insufflation or by the pulmotor (see "Report of the Commission on Resuscitation from Electric Shock," June, 1913). Always apply external heat and massage over the cardiac region. If facilities are at hand and the victim is apparently dead, inject at once adrenalin and salt solution into a large artery (see page 468). While any heart action remains there is a chance of resuscitation. When heart action and respiration are present the prognosis is good. Very severe burns may be caused. The author has dressed many electric burns with hot fomentations of salt solution during the first few days. This facilitates the separation of the sloughs and seems to aid the weakened tissues in resisting microbic invasion; after sloughs separate, the part is dressed with dry sterile gauze. Antiseptic dressings can be used from the beginning, but they often fail entirely to arrest the sloughing. Iodoform produces much irritation and

[1] See the article by N. W. Sharpe on "Peculiarities and Treatment of Electrical Injuries," in "Phila. Med. Jour.," Jan. 29, 1898.

[2] See the directions in "Med. Record," Dec. 28, 1895, from "Med. Press."

should not be employed. Ointments are very unsatisfactory. When the dressings are changed, the part should not be washed with corrosive sublimate, as this agent produces irritation; peroxid of hydrogen should be employed, followed by warm normal salt solution. Sharpe removes sloughs by applying the following mixture: 2 parts of scale pepsin, 1 part of hydrochloric acid, U. S. P., 120 parts of distilled water. This mixture is washed off after two hours with peroxid of hydrogen. The same surgeon treats necrosis of bone by injecting every few hours a 3 per cent. solution of hydrochloric acid, using every second day the pepsin solution, and when necrotic areas come away, packing with gauze. When repair begins, the raw surface should be covered with silver-foil. Skin-grafting by Reverdin's method or Thiersch's method is rarely successful. In some regions it is possible to slide a large flap in place to cover a raw area which will not heal. In a very severe case amputation or resection may be necessary.

In New York, Pennsylvania, New Jersey, and several other states electricity is employed to execute criminals convicted of capital offenses. The infliction of death by electricity is popularly spoken of as *electrocution*. It is beyond doubt, in the words of my colleague, Professor Spitzka, "the most humane method of executing criminals." The first electrocution in New York was in Auburn Prison in 1890, and since then over 100 criminals have been executed by electricity in New York state alone. Dr. Spitzka has witnessed 36 electrocutions and made autopsies on 27 of the victims ("Jour. Med. Soc. of New Jersey," 1909). The apparatus used is "an alternating dynamo capable of generating 2000 volts, a 'death-chair' with adjustable head-rest, binding straps, and adjustable electrodes. (At Trenton a 2400-volt current is taken from the public service wire and lowered to the desired tension by a rheostat.) The notch to control the current is in the death-chamber and the dynamo is in another apartment, communication being had by electric signals. The prisoner (usually without fetters) walks in when everything is ready and sits down in the chair, and his arms, legs, head, and chest are strapped to the chair. An electrode moist with salt solution is fastened to the head and another to the bare calf of the leg. The head need not be shaved.

"The application of the current is usually as follows: The contact is made with a high potential—1800 volts—for five to seven seconds, reduced to 250 volts until a half-minute has elapsed; raised to high voltage for three to five seconds; again reduced to low voltage until one minute has elapsed, when it is again raised to the high voltage for a few seconds, and the contact is broken. The ammeter usually shows that from 7 to 10 amperes have passed through the criminal's body.

"A second or even a third brief contact is sometimes made, partly as a precautionary measure, but more to completely abolish reflexes in the dead body.

"The time consumed by the strapping-in process is usually about forty-five seconds and the first contact is made a few seconds later. In all, about sixty to seventy seconds elapse from the moment the convict leaves his cell until he is shocked to death" (Spitzka, Ibid.).

After electrocution the temperature of the body rises and may reach 129.5° F. Dr. Spitzka finds that after removing the brain the temperature in the vertebral canal is often over 120° F. The brain shows capillary hemorrhages, arterial anemia, and venous congestion. In some of Spitzka's cases sections of the pons, oblongata, and spinal cord showed areas resembling gaseous emphysema, which were perhaps caused by "electrolytic liberation of gas."

In electrocution there is no pain, consciousness is at once abolished, death is certain, and resuscitation is impossible. The lurid stories about criminals being killed by the necropsy and not by the electric current are nonsense.

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